

<p style="text-align: right;">1</p> <p style="text-align: center;"><b>Thursday, 27 February 2020</b></p> <p>1 2 <b>(10.30 am)</b> 3 SIR BRIAN LANGSTAFF: Can I before they are sworn, 4 once again welcome a panel of distinguished 5 experts, two of whom you have already met during 6 this week, those of you who were here yesterday, 7 welcome back. But welcome to the other four. 8 I know that you are all busy practitioners and 9 so we are especially pleased that you have made 10 time in busy timetables, with other 11 responsibilities, to be here to help us. Thank 12 you. 13 May they be sworn. 14 <b>PROFESSOR GARETH TUDOR-WILLIAMS (affirmed)</b> 15 <b>PROFESSOR GRAHAM COOKE (sworn)</b> 16 <b>SIAN EDWARDS (affirmed)</b> 17 <b>DR DAVID JOHNSTON (sworn)</b> 18 <b>DR IAN WILLIAMS (affirmed)</b> 19 <b>PROFESSOR JANE ANDERSON (affirmed)</b> 20 SIR BRIAN LANGSTAFF: Ms Scott. 21 <b>Examined by MS SCOTT</b> 22 MS SCOTT: I am going to start by asking you to 23 introduce yourselves and say a bit about the 24 experience and expertise that you bring to the 25 Inquiry, but can I start, first of all, with</p>	<p style="text-align: right;">2</p> <p>1 you, Professor Tudor-Williams, with an apology 2 that you have been demoted on your name tag up 3 there to Dr, but to reassure you that your 4 correct title has been put in the report. So 5 can I start with you. 6 PROFESSOR TUDOR-WILLIAMS: Well, let me say I am 7 a doctor, and I'm a paediatrician, and I first 8 looked after a child that we diagnosed with HIV 9 at Saint Mary's Hospital along the road in 10 Paddington in 1988, and since that time I have 11 been working with children and young people 12 infected and affected by HIV. I spent five 13 years in America specialising from 1989 to 1994, 14 and since then I returned to join an 15 extraordinarily accomplished group of colleagues 16 running a family clinic for HIV infected 17 children and young people, and now adolescents 18 and people in their 30s who are still from our 19 surviving cohort. Thank you. 20 MS SCOTT: Professor Cooke? 21 PROFESSOR COOKE: I am Graham Cooke. For those who 22 weren't here yesterday, I have been working with 23 patients living with HIV for over 25 years. 24 I am based at the same institution as 25 Professor Tudor-Williams and my focus really is</p>
<p style="text-align: right;">3</p> <p>1 on co-infection. I lead the co-infection 2 services in that hospital and the majority of my 3 time now is spent on research, particularly 4 around co-infection and the aspects of HIV, 5 including HIV diagnosis and cure work. 6 MS SCOTT: Ms Edwards? 7 MS EDWARDS: Hello again. So my name is Sian 8 Edwards. I started HIV work as a clinical nurse 9 specialist, a bit of a misnomer at the time, 10 which was in 1986 in Sydney. I came over and 11 spent lots of the subsequent years in Zambia in 12 Africa and in London and Australia doing 13 education and clinical work teaching nurses and 14 to care for patients with HIV and AIDS 15 infection. 16 I also spent five years as a clinical 17 nurse specialist in the haemophilia unit in 18 London at St Thomas' Hospital and one of the 19 reasons that I think I have been brought in by 20 the Inquiry is because I undertook two research 21 projects with people with haemophilia and HIV, 22 which many of you know of, looking at people's 23 life histories and the experiences that they had 24 been through and then a second project with 25 people who were the relatives and the family</p>	<p style="text-align: right;">4</p> <p>1 members of those who had died of AIDS who also 2 had haemophilia. I'm currently based in 3 Melbourne in Australia. 4 MS SCOTT: Dr Johnston. 5 DR JOHNSTON: Good morning, my name is David 6 Johnston. I'm a general practitioner and I have 7 been a general practitioner for 25 years. 8 During that time I have been involved in 9 a number of community initiatives and have also 10 been involved in researching rural and isolated 11 practice, so I believe I bring to the Inquiry 12 the perspective of a generalist, 13 a non-specialist, if I can use that term, so 14 thank you. 15 MS SCOTT: Dr Williams? 16 DR WILLIAMS: My name is Ian Williams and I have 17 been involved in the care of patients with HIV 18 since 1987. I have been -- I am a consultant in 19 (<i>unclear</i>) medicine and a clinical academic and 20 have been directly involved in out-patient care 21 and in-patient care first at the Middlesex 22 Hospital, then at University College London 23 Hospitals and I continue to do out-patient work. 24 I have been chair of the British HIV Association 25 and my clinical academic role has largely been</p>

<p style="text-align: right;">5</p> <p>1 in treatment trials of anti-retroviral therapy.</p> <p>2 MS SCOTT: Professor Anderson?</p> <p>3 PROFESSOR ANDERSON: Thank you, good morning.</p> <p>4 I first trained at Saint Mary's Hospital. I'm</p> <p>5 a doctor. I specialise in HIV infection and</p> <p>6 I trained at Saint Mary's Hospital and first met</p> <p>7 somebody living with HIV in 1984 when</p> <p>8 I qualified, and I continued doing my junior</p> <p>9 doctors years working in infectious diseases and</p> <p>10 was appointed a consultant in HIV medicine at St</p> <p>11 Bartholomew's Hospital in 1990. I helped set up</p> <p>12 the first HIV unit at Barts and also worked at</p> <p>13 Homerton during that time.</p> <p>14 In 2004 I moved across to Homerton</p> <p>15 Hospital where I still practice in Hackney.</p> <p>16 I have been particularly interested in the care</p> <p>17 of women and families and, because of the</p> <p>18 population who use our clinics in East London,</p> <p>19 I've been interested in black and ethnic</p> <p>20 minority communities and in migration. Other</p> <p>21 roles: I have also been chair of the British</p> <p>22 HIV association, like Ian, and I currently chair</p> <p>23 a national campaign on policy charity, the</p> <p>24 National AIDS Trust.</p> <p>25 MS SCOTT: Thank you. You have been provided with</p>	<p style="text-align: right;">6</p> <p>1 two letters of instruction from the Inquiry and</p> <p>2 a summary of some of the evidence given to the</p> <p>3 Inquiry. You have provided a report to the</p> <p>4 Inquiry and you are due to answer some of the</p> <p>5 questions that were raised in the supplementary</p> <p>6 letter of instruction in a supplemental report;</p> <p>7 is that right?</p> <p>8 PROFESSOR ANDERSON: That's right.</p> <p>9 MS SCOTT: The report that you have provided is that</p> <p>10 a report that all of you have read and all of</p> <p>11 you agree with the contents of?</p> <p>12 <i>All agree.</i></p> <p>13 MS SCOTT: There are no areas of disagreement</p> <p>14 between you.</p> <p>15 <i>All agree.</i></p> <p>16 MS SCOTT: Can I start then by asking questions</p> <p>17 about what HIV actually is, and I'm going to ask</p> <p>18 them generally so anyone that wants to answer</p> <p>19 them do feel free to answer.</p> <p>20 PROFESSOR ANDERSON: Where shall we start? HIV is</p> <p>21 a virus which is an infectious agent and it</p> <p>22 belongs to a particular family, the</p> <p>23 retroviruses, and it particularly affects white</p> <p>24 T cells, the T lymphocytes, and its effects mean</p> <p>25 that when it infects somebody and gets into the</p>
<p style="text-align: right;">7</p> <p>1 cells, the immune system becomes damaged and</p> <p>2 disordered. Essentially, it's an infectious</p> <p>3 agent that is particularly affecting the immune</p> <p>4 system of human beings. It comes in two main</p> <p>5 sorts, HIV-1 and HIV-2, which are quite</p> <p>6 significantly different and the treatment of the</p> <p>7 two is different. HIV-1 has got a variety of</p> <p>8 subtypes within it but we have some major</p> <p>9 subtypes which are the most common ones, and it</p> <p>10 appears that the natural history, what happens</p> <p>11 to somebody with the different subtypes of</p> <p>12 HIV-1, is pretty similar across all of them.</p> <p>13 MS SCOTT: What are the differences in terms of what</p> <p>14 are the clinically significant differences</p> <p>15 between HIV-1 and 2?</p> <p>16 DR WILLIAMS: HIV-1 is responsible for the main</p> <p>17 pandemic across the globe. HIV-2 is largely</p> <p>18 limited to West Africa but can be imported into</p> <p>19 other parts of the world. HIV-2 doesn't have</p> <p>20 the same natural history. It's a much more --</p> <p>21 it doesn't have the same impact in terms of</p> <p>22 clinical consequence as HIV-1.</p> <p>23 PROFESSOR ANDERSON: I think the other thing I would</p> <p>24 add to that is the structure and the way the</p> <p>25 virus is, the structure of the virus, means that</p>	<p style="text-align: right;">8</p> <p>1 some of the drugs that work for HIV-1 don't work</p> <p>2 properly for HIV-2 because the virus is of</p> <p>3 a different enough structure for the molecules</p> <p>4 not to sit in the right areas and work as well.</p> <p>5 MS SCOTT: Is that the same the other way round?</p> <p>6 Some of the drugs that worked for HIV-2 don't</p> <p>7 work for HIV-1?</p> <p>8 PROFESSOR ANDERSON: It hasn't worked like that</p> <p>9 because, as Ian I think has alluded to, there</p> <p>10 are far fewer people living with HIV-2 and so at</p> <p>11 the moment there are no specific drugs being</p> <p>12 designed for HIV-2. It's using the HIV-1 drugs</p> <p>13 in a way that is most appropriate for that</p> <p>14 virus.</p> <p>15 MS SCOTT: Again, in terms of the different subtypes</p> <p>16 of HIV-1, are they clinically significantly</p> <p>17 different?</p> <p>18 DR WILLIAMS: No, not really.</p> <p>19 MS SCOTT: How does a person become infected with</p> <p>20 HIV? What are the infection routes?</p> <p>21 PROFESSOR ANDERSON: Where shall we start?</p> <p>22 Worldwide, the most common route of infection is</p> <p>23 through sexual transmission and that can be from</p> <p>24 vaginal sex, from anal sex, from oral sex. So</p> <p>25 sexual transmission and sexual fluids are the</p>

<p style="text-align: right;">9</p> <p>1 most common route of transmission.</p> <p>2 It's also transmissible by blood, blood</p> <p>3 products and it can also be vertically</p> <p>4 transmitted from a mother to an unborn child,</p> <p>5 and it's also transmissible by breast milk. The</p> <p>6 other modes of transmission, so, for example,</p> <p>7 people who use intravenous drugs, that will be</p> <p>8 a microinfusion of blood, so that's blood to</p> <p>9 blood transmission usually. So sex, blood,</p> <p>10 vertical, and breast milk. Have I missed</p> <p>11 anything out?</p> <p>12 DR WILLIAMS: No.</p> <p>13 MS SCOTT: What is the extent of viral exposure</p> <p>14 a person requires to become infected? You</p> <p>15 mentioned a very small amount. Could one</p> <p>16 particle of HIV infect somebody?</p> <p>17 PROFESSOR ANDERSON: I don't know whether, Graham --</p> <p>18 we had been putting some of that --</p> <p>19 PROFESSOR COOKE: I think we will summarise some of</p> <p>20 this in the supplemental responses but, yes,</p> <p>21 that is broadly the case and, therefore, the</p> <p>22 risk of being infected depends very much on</p> <p>23 a number of factors, including the quantity of</p> <p>24 virus that an individual is exposed to and the</p> <p>25 proportion of that batch or product that may</p>	<p style="text-align: right;">10</p> <p>1 have been administered. The risk of infection</p> <p>2 is then balanced by also the individual's</p> <p>3 susceptibility to be infected. So not all</p> <p>4 individuals are similar in their ability to be</p> <p>5 infected and sometimes a similar exposure may or</p> <p>6 may not result in infection itself.</p> <p>7 DR WILLIAMS: Just giving a sense of that, if you</p> <p>8 look at sexual transmission, then maybe 1 in 300</p> <p>9 times when you might have a risk of sexual</p> <p>10 transmission, so about 0.3 per cent, while if</p> <p>11 you have a transfusion of infected blood product</p> <p>12 then that's clearly -- you know, the likelihood</p> <p>13 of infection is uniform. If you have a sexual</p> <p>14 exposure then not everyone who has sexual</p> <p>15 exposure will get infected.</p> <p>16 MS SCOTT: So could it also be the case that if you</p> <p>17 had a number of people exposed to a batch of</p> <p>18 infected blood or blood products, not all of</p> <p>19 them would become infected?</p> <p>20 DR WILLIAMS: Correct.</p> <p>21 PROFESSOR COOKE: Yes, I think that is possible.</p> <p>22 I think without going into too much of the small</p> <p>23 print, then, for example, there are a small</p> <p>24 proportion of individuals who carry particular</p> <p>25 genetic variation who may actually be refractory</p>
<p style="text-align: right;">11</p> <p>1 to infection. That's quite unusual. It varies</p> <p>2 across populations but in a Caucasian population</p> <p>3 maybe 1 per cent of individuals may carry that</p> <p>4 mutation. If they have two copies, it might be</p> <p>5 very hard to be infected and there is evidence</p> <p>6 that such individuals tend to be overrepresented</p> <p>7 in groups where there's been exposure without</p> <p>8 infection. But I don't want to suggest that's</p> <p>9 the most common reason. It's a relatively</p> <p>10 unusual issue.</p> <p>11 PROFESSOR TUDOR-WILLIAMS: I'd just like to add from</p> <p>12 the perspective of a paediatrician, the data</p> <p>13 suggests that drinking 1 litre of breast milk</p> <p>14 from an HIV infected person who is not on</p> <p>15 anti-retroviral treatment would carry the same</p> <p>16 risk of transmission as one unprotected vaginal</p> <p>17 intercourse. So about 1 in 300 risk from 1</p> <p>18 litre of breast milk, just to give you an idea</p> <p>19 that other body fluids are implicated.</p> <p>20 MS SCOTT: Would repeated exposure to infected</p> <p>21 products increase the risk of a person being</p> <p>22 infected, so somebody that hadn't been infected</p> <p>23 on exposure 1, would that mean they wouldn't</p> <p>24 become infected at all on exposure -- up to</p> <p>25 exposure 10, or would that increase the risk?</p>	<p style="text-align: right;">12</p> <p>1 PROFESSOR COOKE: So, in general, yes, but I think</p> <p>2 with the caveats that actually there may be</p> <p>3 a small minority of individuals who are</p> <p>4 naturally resistant and actually won't get an</p> <p>5 increased risk of infection with more exposure,</p> <p>6 but in general that increased risk of exposure</p> <p>7 is likely to increase the risk cumulatively over</p> <p>8 time in broad terms, and I think in our</p> <p>9 supplemental report we'll go into a bit more</p> <p>10 detail about that.</p> <p>11 MS SCOTT: Does anyone else want to add anything on</p> <p>12 that?</p> <p>13 DR EDWARDS: Just the other added exposure, of</p> <p>14 course is the vertical transmission. So</p> <p>15 although somebody may be HIV positive, it does</p> <p>16 not mean that their baby will naturally be</p> <p>17 infected, and obviously treatment changes that</p> <p>18 a lot, but in the early days prior to treatment</p> <p>19 we were talking 15 per cent.</p> <p>20 PROFESSOR TUDOR-WILLIAMS: The data would be -- if</p> <p>21 a person infected with HIV doesn't know their</p> <p>22 diagnosis has a vaginal delivery and breastfeeds</p> <p>23 that child, about one third of those babies</p> <p>24 would be infected. But it very much depends on</p> <p>25 the maternal viral load, and it could be as much</p>



<p style="text-align: right;">13</p> <p>1 two thirds if the mother has a very high viral</p> <p>2 load.</p> <p>3 MS SCOTT: Is it possible to have HIV 1 and HIV 2 in</p> <p>4 one batch of infected blood product?</p> <p>5 PROFESSOR ANDERSON: It's certainly not impossible.</p> <p>6 It would depend -- certainly there are people</p> <p>7 who are living with both viruses, and so it</p> <p>8 would be, I think, very unusual, but I wouldn't</p> <p>9 like to put a figure on it, but not impossible.</p> <p>10 MS SCOTT: So if there are people living with both</p> <p>11 viruses, are there people living with different</p> <p>12 types of -- subtypes of the virus as well?</p> <p>13 PROFESSOR ANDERSON: That's more complicated.</p> <p>14 DR WILLIAMS: I think the -- subtypes are difficult</p> <p>15 because the virus evolves quite quickly in the</p> <p>16 body. It replicates very rapidly. It doesn't</p> <p>17 proofread itself, so it makes -- errors are</p> <p>18 introduced and, therefore, you get a very broad</p> <p>19 sort of quasi-species within the -- within the</p> <p>20 person who is infected, and so you may get</p> <p>21 a subtype, but they could be known as</p> <p>22 recombinants, which are basically where viruses</p> <p>23 have come together. But it's largely -- you can</p> <p>24 generally determine whether someone is a subtype</p> <p>25 B or subtype A generally from -- from their DNA</p>	<p style="text-align: right;">14</p> <p>1 analysis.</p> <p>2 PROFESSOR TUDOR-WILLIAMS: I think it is worth</p> <p>3 saying that in the early days, I certainly</p> <p>4 remember back in the '90s, there was reports</p> <p>5 coming out of various corners of the world,</p> <p>6 including India, where they were claiming that</p> <p>7 there were all sorts of people infected with</p> <p>8 HIV-1 and 2, which was a complete misconception</p> <p>9 based on the fact that the test was picking up</p> <p>10 antibodies to either HIV-1 or HIV-2. It was</p> <p>11 reported as positive for HIV-1/2, and people</p> <p>12 were being -- there were physicians who believed</p> <p>13 that that meant you got both viruses, and that</p> <p>14 simply was not true. In fact I think in the UK</p> <p>15 I simply don't remember a single child that we</p> <p>16 have looked after over 25 years who had both</p> <p>17 viruses.</p> <p>18 PROFESSOR ANDERSON: Certainly people can -- I think</p> <p>19 Gareth is absolutely right, the testing and the</p> <p>20 technology and the laboratory differentiation of</p> <p>21 the different sorts of HIV-1 or 2 has become</p> <p>22 much more sophisticated, and those early tests</p> <p>23 may -- often came back as a report which didn't</p> <p>24 necessarily differentiate. So I think that's</p> <p>25 a very fair comment. But in today's world you</p>
<p style="text-align: right;">15</p> <p>1 can differentiate them and we know that for</p> <p>2 certain parts of the world there are people who</p> <p>3 are living, definitely, with both 1 and 2.</p> <p>4 PROFESSOR COOKE: Just to add a couple of points.</p> <p>5 I think in our routine practice it's very</p> <p>6 unusual to see patients with both HIV-1 and</p> <p>7 HIV-2, but I think -- and -- so from a single</p> <p>8 donor, for example, that would be an unusual</p> <p>9 thing. But where you've got products pooled, as</p> <p>10 we discussed yesterday a little bit around</p> <p>11 hepatitis C genotypes, then it theoretically</p> <p>12 could be possible for more than one type of HIV</p> <p>13 to be present.</p> <p>14 I suppose the other thing that might be</p> <p>15 helpful to talk about is super-infections. So</p> <p>16 it is possible, once you have an infection with</p> <p>17 HIV, to have a super-infection if you have</p> <p>18 a further exposure, and we see this particularly</p> <p>19 with sexual transmission, even now, and then in</p> <p>20 that situation you may have more than one type</p> <p>21 of the HIV-1 in circulation. It doesn't</p> <p>22 necessarily have big implications, we may come</p> <p>23 on to that, but it is definitely possible.</p> <p>24 MS SCOTT: So super-infection relates to infection</p> <p>25 with a different type of HIV?</p>	<p style="text-align: right;">16</p> <p>1 PROFESSOR COOKE: Where you can distinguish that</p> <p>2 virus from the one the patient originally had.</p> <p>3 MS SCOTT: Presumably -- well, does repeated</p> <p>4 exposure to the same batch of infected product</p> <p>5 then, would that increase your viral load? Does</p> <p>6 that change the nature of your infection?</p> <p>7 PROFESSOR COOKE: I think we've tried to find</p> <p>8 evidence of this. I don't know if -- so we</p> <p>9 found relatively little evidence to suggest that</p> <p>10 the viral load will change but I think if people</p> <p>11 are aware of other evidence that would be</p> <p>12 helpful.</p> <p>13 DR WILLIAMS: I don't think the viral load would</p> <p>14 change. That very much depends on how the</p> <p>15 immune system of the individual who is infected</p> <p>16 responds to HIV and how well that protective</p> <p>17 immunity develops, and so the exposure to HIV</p> <p>18 and the inoculum with HIV in it -- well, you</p> <p>19 know, so the route of infection does not</p> <p>20 determine the natural history of HIV or the</p> <p>21 individual's response, right. It will determine</p> <p>22 the risk of acquiring HIV in terms of the size</p> <p>23 of the inoculum, which would be much greater</p> <p>24 with the blood product than it would be for</p> <p>25 sexual exposure. So the risks would be --</p>



<p style="text-align: right;">17</p> <p>1 definitely be different but in terms of how the</p> <p>2 immune system then responds to that inoculum</p> <p>3 will be dependent on other factors, viral and</p> <p>4 host factors, which are -- we can go into.</p> <p>5 MS SCOTT: Perhaps we should go on now then to the</p> <p>6 stages of infection of untreated HIV as you deal</p> <p>7 with it in your report. You identify three</p> <p>8 stages: early infection, or acute or primary</p> <p>9 infection; chronic infection; and then advanced</p> <p>10 infection.</p> <p>11 So if you can just start with early</p> <p>12 infection. How is this characterised? What is</p> <p>13 happening when somebody is first infected, in</p> <p>14 terms of the virus and in terms of their immune</p> <p>15 response?</p> <p>16 DR WILLIAMS: When someone first acquires HIV, the</p> <p>17 immune system will respond like it will respond</p> <p>18 to any infection. It will recognise that</p> <p>19 infection and it will be activated, and it will</p> <p>20 try to produce an immune response to that, and</p> <p>21 it -- generally that immune response can be</p> <p>22 detected after about two to four weeks following</p> <p>23 infection. So those anti -- those responses can</p> <p>24 be divided into largely a response by producing</p> <p>25 antibodies, which tends to protect or fight</p>	<p style="text-align: right;">18</p> <p>1 infection within cell-free virus, or produces</p> <p>2 responses that are cell-based, and that's --</p> <p>3 produces -- fights against infection within</p> <p>4 cells.</p> <p>5 That -- so that's the reason we use an</p> <p>6 antibody test to determine whether someone is</p> <p>7 infected, because that tends to become positive</p> <p>8 after two to four weeks and then evolves.</p> <p>9 After that two to four weeks, people --</p> <p>10 the viral load in someone, the amount of virus,</p> <p>11 it replicates. It starts at the area where it</p> <p>12 was first inoculum then spreads throughout the</p> <p>13 body, and it will cause -- replicate in high</p> <p>14 levels and the viral load in someone with</p> <p>15 primary infection is likely to be very high, in</p> <p>16 the figure of millions, as opposed to someone</p> <p>17 with a -- who's got a chronic infection, where</p> <p>18 it will be in the thousands or tens of</p> <p>19 thousands.</p> <p>20 Then the immune system kicks in and tries</p> <p>21 to control the virus, as it would do with any</p> <p>22 infection, but because of the way -- how the</p> <p>23 virus affects the immune system, the immune</p> <p>24 system fails to do an adequate job, it fails to</p> <p>25 clear the virus. But the quality and the</p>
<p style="text-align: right;">19</p> <p>1 strength of how someone responds to the virus is</p> <p>2 dependent on genetic factors, and so someone can</p> <p>3 clear the virus down to very low levels and</p> <p>4 other people they don't develop a very good</p> <p>5 immune response and they have a very high viral</p> <p>6 load after three or four months. But during</p> <p>7 that first two to four weeks someone can be</p> <p>8 unwell, with symptoms of primary infection, and</p> <p>9 those symptoms are things like fever,</p> <p>10 headache -- like flu-like symptoms, often</p> <p>11 unrecognised or sometimes quite severe.</p> <p>12 MS SCOTT: So in your report at page 21 you say this</p> <p>13 about the symptoms at that initial stage:</p> <p>14 "Symptoms most frequently described are</p> <p>15 a raised temperature, a sore throat, mouth</p> <p>16 ulceration, enlarged lymph nodes, aching muscles</p> <p>17 and joints and tiredness. A short-lived pale</p> <p>18 pink rash is sometimes seen. Nausea diarrhoea</p> <p>19 and weight loss can occur. Neurological</p> <p>20 symptoms are common and may include headache and</p> <p>21 aversion to light, photophobia."</p> <p>22 Are those fairly common side effects -- I</p> <p>23 mean, symptoms at this stage?</p> <p>24 DR WILLIAMS: Yes, they are. Because they are very</p> <p>25 much like flu-like symptoms, which people might,</p>	<p style="text-align: right;">20</p> <p>1 you know, get for -- with flu or similar</p> <p>2 viruses, they often go unrecognised. So, for</p> <p>3 example, in clinical -- in clinics, people</p> <p>4 presenting with primary infection is uncommon.</p> <p>5 People often get diagnosed much later. So we</p> <p>6 would -- you know, in terms of the -- it's far</p> <p>7 more common to get diagnosed with established</p> <p>8 infection than to present with primary infection</p> <p>9 because those symptoms which we have described</p> <p>10 are very similar to many other flu-like symptoms</p> <p>11 or glandular fever or something like that which</p> <p>12 people may not seek medical help with and,</p> <p>13 therefore, the diagnosis goes undiagnosed.</p> <p>14 MS SCOTT: And some people will report no symptoms;</p> <p>15 is that right?</p> <p>16 DR WILLIAMS: Yes.</p> <p>17 PROFESSOR ANDERSON: Although, interestingly,</p> <p>18 when -- quite often if you -- when you do meet</p> <p>19 somebody who is newly diagnosed, asking if they</p> <p>20 have ever had a set of symptoms like this, more</p> <p>21 people than you might expect will say,</p> <p>22 "Actually, yes, it does jog a memory". So even</p> <p>23 if they haven't presented to clinical care, on</p> <p>24 closer questioning with a diagnosis people may</p> <p>25 recall having had something.</p>

<p style="text-align: right;">21</p> <p>1 MS SCOTT: You go on in the report to say this:</p> <p>2 "In rare cases there may be signs of</p> <p>3 meningitis or of direct brain infection. In</p> <p>4 most people the illnesses last up to three weeks</p> <p>5 and resolve on their own and recovery is usually</p> <p>6 complete."</p> <p>7 So in those rare cases where there are</p> <p>8 more significant symptoms, do those symptoms</p> <p>9 also resolve on their own or do those continue?</p> <p>10 DR WILLIAMS: No, I mean severe primary</p> <p>11 seroconversion illness or primary infection can</p> <p>12 last several weeks and it can result in hospital</p> <p>13 admission, particularly those with meningitis,</p> <p>14 so it can be a differential diagnosis of</p> <p>15 somebody presenting with meningitis. It's part</p> <p>16 of how the immune system is responding to the</p> <p>17 infection. Some people, it can affect the</p> <p>18 immune system so severely that they develop an</p> <p>19 AIDS defining illness primary infection, but</p> <p>20 that is really quite uncommon in terms of</p> <p>21 presentation to hospital. But, yes, it can be</p> <p>22 quite severe and it can last several weeks in</p> <p>23 terms of symptoms but it does resolve.</p> <p>24 PROFESSOR TUDOR-WILLIAMS: Can I just add the only</p> <p>25 difference in children is that if you acquire</p>	<p style="text-align: right;">22</p> <p>1 HIV in the perinatal period then unfortunately</p> <p>2 this is a neurotropic virus and if it hits your</p> <p>3 brain as it is developing that often had</p> <p>4 life-long consequences. That wasn't so true for</p> <p>5 children meeting the virus for the first time</p> <p>6 during childhood outside the perinatal period</p> <p>7 but it's a very well-recognised consequence in</p> <p>8 the natural history of HIV in perinatal</p> <p>9 transmission from mothers to children.</p> <p>10 MS SCOTT: We heard yesterday about a window period</p> <p>11 when we were discussing hepatitis. Is that</p> <p>12 relevant to HIV and, if so, what is it and why</p> <p>13 is it relevant?</p> <p>14 DR WILLIAMS: It's relevant as I said before you</p> <p>15 develop symptoms of primary infection when the</p> <p>16 viral load in someone is quite high but the</p> <p>17 immune system has yet to develop sufficient</p> <p>18 antibodies to be detected by the assays that we</p> <p>19 use to diagnose HIV. So there used to be</p> <p>20 a period where from the last possible exposure</p> <p>21 to HIV to then having a test used to be a window</p> <p>22 period of three months in terms of if someone</p> <p>23 had had a potential exposure to HIV in that</p> <p>24 period of time we would say we would want to</p> <p>25 wait until at least three months after that</p>
<p style="text-align: right;">23</p> <p>1 exposure to get a test.</p> <p>2 The more modern assays that are now</p> <p>3 employed the fourth and fifth generation assays,</p> <p>4 that is not so much the case. We can now</p> <p>5 identify someone with primary HIV infection two</p> <p>6 to four weeks after exposure because the more</p> <p>7 sensitive assays that we use do detect virus.</p> <p>8 PROFESSOR COOKE: Just to follow up on that, and I</p> <p>9 think Dr Jefferey has summarised a lot of</p> <p>10 diagnosis development, but in HIV we have seen</p> <p>11 very good progress in terms of tests as</p> <p>12 Dr Williams is saying which detect both the</p> <p>13 immune response and the virus at the same time</p> <p>14 which are now routinely used and allow that</p> <p>15 early detection and narrowing the window,</p> <p>16 whereas that contrasts slightly with the</p> <p>17 development with hepatitis that was slightly</p> <p>18 behind.</p> <p>19 MS SCOTT: You use the term in your report</p> <p>20 seroconversion. Can you explain what that</p> <p>21 means.</p> <p>22 DR WILLIAMS: That's just from going to antibody</p> <p>23 negative to antibody positive, ie the detection</p> <p>24 of antibodies that are specifically aimed</p> <p>25 against HIV being detected by the diagnostic</p>	<p style="text-align: right;">24</p> <p>1 assays that we use.</p> <p>2 MS SCOTT: So the acute phase you have described as</p> <p>3 lasting a number of months. What is it that</p> <p>4 marks the end of the acute phase and moving into</p> <p>5 the chronic phase?</p> <p>6 DR WILLIAMS: I suppose after the seroconversion</p> <p>7 illness what happens is there's a balance that</p> <p>8 is struck between the virus trying to replicate</p> <p>9 and the immune system trying to defend and</p> <p>10 develop an immune response and so, as I said, as</p> <p>11 Jane has also alluded to, the virus attacks the</p> <p>12 cells in the body that are specifically</p> <p>13 important for co-ordinating the immune response</p> <p>14 to any infection, which are the T helper cells</p> <p>15 and these are very key in the co-ordination of</p> <p>16 the immune response, particularly adaptive and</p> <p>17 cellular immune responses, so what happens is</p> <p>18 the immune system is activated and it remains</p> <p>19 activated and it tries to constantly generate</p> <p>20 immune responses to HIV. In some people it can</p> <p>21 be very successful. In other people it's not so</p> <p>22 successful.</p> <p>23 Those where it's more successful, then the</p> <p>24 virus is better controlled and the viral load in</p> <p>25 someone is lower. Where it's less successful</p>

<p style="text-align: right;">25</p> <p>1 the viral load is much higher and, to a certain</p> <p>2 extent, what happens during acute infection</p> <p>3 determines the natural history of HIV in an</p> <p>4 individual, so how your immune response responds</p> <p>5 to HIV during primary infection, during that</p> <p>6 acute phase, will determine your natural history</p> <p>7 because it determines how well your immune</p> <p>8 system is coping with HIV.</p> <p>9 PROFESSOR ANDERSON: The time-line there, in general</p> <p>10 that bit of the process takes about six months</p> <p>11 to get to a steady state and so the arbitrary</p> <p>12 cut-off is that early infection is the first six</p> <p>13 months while these processes are going on and</p> <p>14 the viral load stabilises out to the viral set</p> <p>15 point and that sort of marks the end of early</p> <p>16 infection, if you like.</p> <p>17 DR EDWARDS: It is worth noting as well with regard</p> <p>18 to this issue is that people are very highly</p> <p>19 infectious during that initial stage, so you</p> <p>20 have got highly replicating virus and so</p> <p>21 infectivity even though people may go through</p> <p>22 a test that is antibody negative the infectivity</p> <p>23 is particularly high.</p> <p>24 MS SCOTT: So moving on then to the sixth month</p> <p>25 arbitrary six month point where we're at the</p>	<p style="text-align: right;">26</p> <p>1 chronic stage, the viral set point you've</p> <p>2 mentioned can differ between people. What are</p> <p>3 the factors then that contribute to that</p> <p>4 difference? In your report you said there are</p> <p>5 pathogen-related reasons and host-related</p> <p>6 reasons.</p> <p>7 DR WILLIAMS: I suppose in terms of age it's by far</p> <p>8 the most important sort of host-related reasons</p> <p>9 because as you get older your immune system</p> <p>10 doesn't work as well as when you are younger</p> <p>11 and, therefore, you are likely to have a much</p> <p>12 better immune response when you are younger than</p> <p>13 when you are older, so age is definitely</p> <p>14 a factor in terms of that.</p> <p>15 As Graham has alluded to, there are</p> <p>16 certain people who have genetically more</p> <p>17 resistance to HIV in terms of they don't express</p> <p>18 the certain receptors on their CD4 cells that</p> <p>19 make it much more difficult for HIV and there</p> <p>20 are other genetic factors that determine</p> <p>21 a person's immune responses to HIV in terms of</p> <p>22 how well -- like any infection, so if you take</p> <p>23 other infections, people can respond</p> <p>24 differently, their immune systems can respond.</p> <p>25 We don't all respond the same. Genetically we</p>
<p style="text-align: right;">27</p> <p>1 differ.</p> <p>2 MS SCOTT: In your report you mention people with</p> <p>3 higher numbers of CD8 lymphocytes circulating</p> <p>4 and people who express HLAB57 markers having</p> <p>5 lower viral set points. Are those genetic</p> <p>6 factors?</p> <p>7 DR WILLIAMS: Yes.</p> <p>8 MS SCOTT: You have also in your report drawn</p> <p>9 attention to the presence of other infections</p> <p>10 having an impact on the host-related</p> <p>11 contribution to viral set point. Can you tell</p> <p>12 us a bit about that.</p> <p>13 DR WILLIAMS: I think in terms of other</p> <p>14 co-infections, things like hepatitis B or</p> <p>15 hepatitis C don't have an impact in terms of</p> <p>16 how -- you know, in terms of how the immune</p> <p>17 system responds to HIV. It has an impact the</p> <p>18 other way round. Generally there aren't any</p> <p>19 other co-infections that will impact on the set</p> <p>20 point or on the natural history. There was data</p> <p>21 from a cohort looking at the cytomegalovirus in</p> <p>22 terms of whether that has an impact but that is</p> <p>23 not universally accepted. Generally, it's the</p> <p>24 quality and the strength of the immune response,</p> <p>25 which are genetically determined or age</p>	<p style="text-align: right;">28</p> <p>1 determined, that will determine the immune</p> <p>2 response to HIV. The subtypes makes no</p> <p>3 difference or the route of infection does not</p> <p>4 make any difference.</p> <p>5 PROFESSOR ANDERSON: I would just add that some</p> <p>6 other infections will also be challenging the</p> <p>7 immune system at the same time, and so you may</p> <p>8 be seeing the effect of two separate processes,</p> <p>9 and so that may in itself be accounting for --</p> <p>10 it may be more rapid CD4 decline but it is not</p> <p>11 necessarily working through the viral load</p> <p>12 mechanism.</p> <p>13 PROFESSOR COOKE: Yes, a similar point. So I think</p> <p>14 you can see in the setting of another acute</p> <p>15 illness that someone's HIV viral load may rise</p> <p>16 transiently and usually come back again. But as</p> <p>17 we have heard, these sort of commonly recognised</p> <p>18 chronic co-infections tend not to have a big</p> <p>19 impact on that viral load.</p> <p>20 MS SCOTT: Turning then to pathogen-related reasons</p> <p>21 I think you said the subtype doesn't have an</p> <p>22 impact but the type of HIV, 1 or 2, does that</p> <p>23 impact on --</p> <p>24 DR WILLIAMS: Yes. I mean, everything we've written</p> <p>25 to should really be related -- this should have</p>



<p style="text-align: right;">29</p> <p>1 -- the title should be "HIV-1", not "HIV-1 and 2 2". Because 2 is really a very different 3 disease, we see -- you know, it's something that 4 only, you know, in the UK there's only 200 or 5 300 people who are infected with HIV-2 and it's 6 largely only seen in West Africa or people who 7 come from West Africa, so when we talk about HIV 8 we are largely talking about HIV -- we are 9 talking about HIV-1.</p> <p>10 MS SCOTT: The other factor you mention in your 11 report is the type of cellular receptor that the 12 virus uses to enter the cells. It may be 13 important as a factor in the progression of HIV. 14 Can you tell us how that occurs?</p> <p>15 DR WILLIAMS: HIV needs, as the report says, the 16 reason why it is able to infect T helper cells, 17 which are the specific type of white cells that 18 are important for the immune response, is that 19 those cells carry a molecule called CD4. It's 20 the CD4 which the virus is able to attach to. 21 But in order to gain entry into the cell it 22 needs another co-receptor, which are determined 23 chemokine receptors, and just for -- technically 24 there are ones called CCR4, another one CCR5, 25 and the virus can use one of those co-receptors.</p>	<p style="text-align: right;">30</p> <p>1 Generally a virus that is transmitted uses 2 CCR5-bearing -- is tropic to CCR5 but later on 3 in the natural history of HIV can use CCR4, it 4 switches. It's usually CCR5. There are people 5 who do not express CCR5 and they have a genetic 6 deletion which means they do not express CCR5 7 and, therefore, they are people who are -- who 8 are less likely to become infected with HIV but 9 also people who are -- their natural history is 10 much longer. They are, really, as Graham said, 11 this is a small proportion in the UK.</p> <p>12 MS SCOTT: So over this chronic stage then, what's 13 happening to the virus and to the immune system?</p> <p>14 DR WILLIAMS: If I could -- usually when the immune 15 system responds to an infection it is activated 16 and once the infection is gone the immune system 17 goes back into resting state. But in HIV that 18 doesn't occur because the immune system is 19 constantly responding to ongoing viral 20 replication and to other factors. So the immune 21 system is in a state of hyperactivation and 22 inflammation.</p> <p>23 If I can give you a sense of -- if you 24 were going to the gym and you were running on 25 a treadmill you could walk quite comfortably for</p>
<p style="text-align: right;">31</p> <p>1 a long way but if you are having to run at 2 a very high level you get exhausted much more 3 quickly. That's what happens with the immune 4 system. It basically is in a state of immune 5 activation at a high level because there's this 6 constant level of replication of the virus. So 7 the patient remains clinically well but there's 8 ongoing viral replication. So you are in 9 a state of high inflammation and high immune 10 activation.</p> <p>11 MS SCOTT: Can I, Henry, ask you to pull up on the 12 report page 6, table 1, please. Can someone 13 tell us what this table is, what we're actually 14 looking at here?</p> <p>15 PROFESSOR ANDERSON: So this is one of the 16 classification systems used internationally for 17 HIV infection. This is one from CDC, the 18 Centers for Disease Control in America, and is 19 probably the most commonly used classification 20 to try and describe where along the spectrum the 21 infection has got to, going through some of the 22 stages we have already discussed.</p> <p>23 There are a number of classifications. 24 The WHO have got one but this is the one that is 25 the most commonly used when we're referring to</p>	<p style="text-align: right;">32</p> <p>1 where in the stage of infection people are.</p> <p>2 So we have been talking so far about acute 3 infection, early infection. Here we're talking 4 about category A, which is the first column. 5 Then we're talking about this latent period, if 6 you like, chronic infection, where we talk about 7 B. Then we get to a point which is 8 classification C, which is where the HIV 9 infection has now caused enough damage for 10 somebody to get sick with a variety of 11 particular clinical conditions which allows 12 a diagnosis of AIDS to be made.</p> <p>13 Now, in addition to that, there is the 14 ability to put in the numbers of CD4 cells to 15 modify that classification about whether 16 somebody's got a very robust immune system, 17 which is with the CD4 count above 500, or 18 whether they are already in a place where the 19 CD4 count is very low, i.e. below 200.</p> <p>20 So this is really I suppose the 21 international shorthand that is used in the 22 world of HIV classification. It's probably, 23 I would say, it's used much more as perhaps 24 a public health and epidemiological tool as 25 a way of charting what's happening, allowing you</p>

<p style="text-align: right;">33</p> <p>1 to compare populations. It's also important for</p> <p>2 an individual patient, to give them a sense of</p> <p>3 where on the spectrum they sit. So this is the</p> <p>4 recognised international classification.</p> <p>5 MS SCOTT: In this chronic phase, this middle phase</p> <p>6 before AIDS is diagnosed, as I understand your</p> <p>7 report somebody could be showing no symptoms or</p> <p>8 somebody could be showing one or a number of the</p> <p>9 symptoms that are listed there in clinical</p> <p>10 category B.</p> <p>11 PROFESSOR ANDERSON: Absolutely, yes.</p> <p>12 MS SCOTT: Presumably people could move from no</p> <p>13 symptoms to symptoms back to no symptoms.</p> <p>14 PROFESSOR ANDERSON: Yes, although on the whole if</p> <p>15 somebody has moved into early symptomatic</p> <p>16 infection often there's something grumbling</p> <p>17 around. It's --</p> <p>18 DR WILLIAMS: Can I say, once they have gone into</p> <p>19 category B, that's an indication that their</p> <p>20 immune system has been damaged quite</p> <p>21 significantly so they can't move back to being</p> <p>22 asymptomatic.</p> <p>23 PROFESSOR COOKE: It is worth just mentioning</p> <p>24 briefly again about the viral load in the CD4</p> <p>25 account, so we talked a bit about T helper cells</p>	<p style="text-align: right;">34</p> <p>1 and when we talk about CD4 counts, we're talking</p> <p>2 about a blood test which gives an estimate of</p> <p>3 the numbers of concentration of those CD4 cells.</p> <p>4 So the patient may be well and not have</p> <p>5 symptoms but their CD4 count may be declining</p> <p>6 and that is something that can be monitored.</p> <p>7 That tends to be the most useful test we can use</p> <p>8 to monitor that phase. The viral load is often</p> <p>9 measured. These days, patients would usually be</p> <p>10 on therapy but the viral load can give you some</p> <p>11 sense of progression risk but it doesn't tend to</p> <p>12 be what we use to monitor where things are</p> <p>13 going. CD4 count is the thing that's used</p> <p>14 mostly.</p> <p>15 PROFESSOR ANDERSON: I think that's also important</p> <p>16 in the States and in some other countries of</p> <p>17 the world just having a CD4 count below 200</p> <p>18 allows a definition of AIDS to be made. In the</p> <p>19 UK, you are required to have one of these</p> <p>20 clinical indicator diseases as well. So that's</p> <p>21 a difference between the American classification</p> <p>22 and ours, although in actual fact I think in</p> <p>23 practical terms it probably makes little</p> <p>24 difference because, as Graham is saying, by that</p> <p>25 stage your immune system is weak and you are</p>
<p style="text-align: right;">35</p> <p>1 susceptible to major infections.</p> <p>2 MS SCOTT: When you talk about the clinical</p> <p>3 AIDS-defining diseases, what you mean is one of</p> <p>4 those in category C?</p> <p>5 PROFESSOR ANDERSON: Exactly.</p> <p>6 DR EDWARDS: It might be worth noting that although</p> <p>7 the classification doesn't change, you stay in</p> <p>8 that category. Of course, on a practical level</p> <p>9 you may well have medication and treatment that</p> <p>10 makes you feel better, your symptoms are less,</p> <p>11 but the classification remains the same.</p> <p>12 PROFESSOR ANDERSON: Exactly.</p> <p>13 DR EDWARDS: So you as a person may be treated and</p> <p>14 do not feel as sick as you were before but your</p> <p>15 immune system is still very compromised.</p> <p>16 PROFESSOR ANDERSON: This, of course, presupposes</p> <p>17 this is without anti-retroviral therapy. This</p> <p>18 is the progression as untreated infection.</p> <p>19 PROFESSOR TUDOR-WILLIAMS: Forgive me, we didn't put</p> <p>20 it into the report but there are different</p> <p>21 classification systems for children who, by and</p> <p>22 large, have rather more rapid disease</p> <p>23 progression than adults. The CDC came out with</p> <p>24 their classification system in 1994 and WHO</p> <p>25 revised that in about 2007 with a very</p>	<p style="text-align: right;">36</p> <p>1 comprehensive view of paediatric HIV that is</p> <p>2 different to this but it does fall into similar</p> <p>3 kinds of categories of ABC.</p> <p>4 MS SCOTT: Again, the sort of acute, chronic and</p> <p>5 then AIDS.</p> <p>6 PROFESSOR TUDOR-WILLIAMS: Yes, and recognising that</p> <p>7 actually in children it was even less common for</p> <p>8 the immune system to mount an effective response</p> <p>9 that produced symptomatic disease and indeed</p> <p>10 a less effective immune system that brought the</p> <p>11 viral load down, so that in perinatal infected</p> <p>12 children we were still seeing viral loads above</p> <p>13 100,000 very commonly two years after -- you</p> <p>14 know, aged two.</p> <p>15 Again, as you go through childhood beyond</p> <p>16 the age of 5 the CD4 counts broadly fall in line</p> <p>17 with adult CD4 counts. Below 5 children have</p> <p>18 very much higher lymphocyte counts and CD4</p> <p>19 populations, so we had a whole different series</p> <p>20 of thresholds for recognising severe immune</p> <p>21 suppression in children.</p> <p>22 We haven't put that all into the report.</p> <p>23 I am very happy to provide it in a supplemental</p> <p>24 report, if that's felt to be relevant.</p> <p>25 MS SCOTT: So for children it takes them a lot</p>

<p style="text-align: right;">37</p> <p>1 longer because their immune systems are</p> <p>2 immature?</p> <p>3 PROFESSOR TUDOR-WILLIAMS: No, I wish that was true.</p> <p>4 No, quite the opposite, because you have got so</p> <p>5 many target cells, HIV replicates very, very</p> <p>6 efficiently in young children and, no, the fact</p> <p>7 that you've got a higher CD4 count just means</p> <p>8 that we had to adjust the threshold. I remember</p> <p>9 very clearly being told by my adult colleagues</p> <p>10 when we were diagnosing AIDS-defining conditions</p> <p>11 like pneumocystis pneumonia, in children with</p> <p>12 CD4 counts of 1,200 my colleagues would say,</p> <p>13 "Oh, don't be stupid. You paediatricians don't</p> <p>14 know what you're talking about". Actually, it</p> <p>15 turned out that the average six month old ought</p> <p>16 to have a CD4 count of about 3,000, and so 1,200</p> <p>17 was already severely immunocompromised -- with</p> <p>18 all due respect.</p> <p>19 MS SCOTT: Turning then back to the clinical</p> <p>20 category B, can we -- because the Inquiry has</p> <p>21 received and heard a lot of evidence about the</p> <p>22 different experiences that people had in the</p> <p>23 early days of HIV infection.</p> <p>24 Can I ask you to go -- to explain what</p> <p>25 these clinical conditions are and how they would</p>	<p style="text-align: right;">38</p> <p>1 be experienced by patients and what the</p> <p>2 treatment for them would be and, again, what the</p> <p>3 symptoms and side effects of those treatments</p> <p>4 would be.</p> <p>5 PROFESSOR ANDERSON: You are talking here about the</p> <p>6 things that don't reach a classification of</p> <p>7 AIDS.</p> <p>8 MS SCOTT: I am talking here about the clinical</p> <p>9 category B matters that are set out in this</p> <p>10 table 1.</p> <p>11 PROFESSOR ANDERSON: So there's a variety here of</p> <p>12 conditions that are coming into play because the</p> <p>13 immune system is weakening, and it will depend,</p> <p>14 again, on each individual person which of these</p> <p>15 conditions comes to the forefront and which ones</p> <p>16 they experience. There are some particularly</p> <p>17 common things. Shingles (herpes zoster) is</p> <p>18 quite a common thing for people to experience</p> <p>19 early on because most people have at some point</p> <p>20 in their lives had a chicken pox infection, so</p> <p>21 it is a common virus that is in most people and</p> <p>22 actually it's quite pathogenic, so you don't</p> <p>23 have to be that immunosuppressed for it to</p> <p>24 reoccur. So it is one of the things that can</p> <p>25 come quite early on in category B, if you like,</p>
<p style="text-align: right;">39</p> <p>1 because it is quite a powerful pathogen, it's</p> <p>2 common and you don't have to be that</p> <p>3 immunosuppressed to find it.</p> <p>4 We noticed things like -- I have put</p> <p>5 here -- we have here idiopathic thrombocytopenic</p> <p>6 purpura. That is when your platelets in your</p> <p>7 blood are low and that's probably an autoimmune</p> <p>8 problem that's going on in the background so</p> <p>9 people might notice easy bruising or on</p> <p>10 a routine blood test, oh, the platelet count is</p> <p>11 looking low. The patient may actually not</p> <p>12 necessarily notice that but it may come up as</p> <p>13 a signal, if you like, there's something going</p> <p>14 on.</p> <p>15 The sense of constitutional symptoms,</p> <p>16 people talking about just really not feeling</p> <p>17 great in quite a non-specific way and in</p> <p>18 those -- certainly in the days before</p> <p>19 anti-retroviral therapy it was not uncommon for</p> <p>20 people to talk about sweats, particularly night</p> <p>21 sweats, fevers and feeling just generally out of</p> <p>22 sorts and not well in quite a non-specific way.</p> <p>23 Infections of the skin are quite common,</p> <p>24 flaky skin, seborrheic dermatitis on the face,</p> <p>25 quite common and infections of the mouth like</p>	<p style="text-align: right;">40</p> <p>1 thrush and viruses that can affect the tongue</p> <p>2 causing oral hairy leukoplakia, which is an</p> <p>3 Epstein Barr virus. They are all quite</p> <p>4 non-specific in that setting but they are</p> <p>5 indicators of an immune suppression going on in</p> <p>6 the background.</p> <p>7 Many of these now, and we come to this</p> <p>8 a bit later in the report, could be, if you</p> <p>9 like, indications as sign posts, signalling</p> <p>10 conditions, people who maybe go to their doctor</p> <p>11 with some of these conditions should be ringing</p> <p>12 alarm bells that there's something else in the</p> <p>13 background. But these are often not</p> <p>14 particularly specific to HIV but they are there</p> <p>15 as a marker that the body is not strong and</p> <p>16 something is going on in the background.</p> <p>17 MS SCOTT: In the 1980s were treatments available</p> <p>18 for most of these conditions?</p> <p>19 PROFESSOR ANDERSON: In the 1980s, it would depend.</p> <p>20 It was variable.</p> <p>21 MS SCOTT: 1990s?</p> <p>22 PROFESSOR COOKE: Some of them, but limited I think.</p> <p>23 Acyclovir would have been available, which would</p> <p>24 be a standard treatment for shingles (herpes</p> <p>25 zoster) and some of these would have responded</p>



<p style="text-align: right;">41</p> <p>1 to standard antibiotics like penicillin. The</p> <p>2 more common things -- and thrush and I think, in</p> <p>3 fact, there would have been some therapies, but</p> <p>4 when we come on to talk about the conditions of</p> <p>5 more advanced disease, those therapies were ones</p> <p>6 that needed to be develop specifically for HIV</p> <p>7 infected patients.</p> <p>8 So of this list I would say actually</p> <p>9 probably most of them had some treatment</p> <p>10 available.</p> <p>11 DR WILLIAMS: I think things like constitutional</p> <p>12 symptoms, which are really disabling, things</p> <p>13 like feeling very tired, diarrhoea, weight loss,</p> <p>14 night sweats, there weren't specific treatments</p> <p>15 for that because they were the direct effect of</p> <p>16 HIV damaging the immune system. Things like</p> <p>17 peripheral neuropathy, which is numbness in the</p> <p>18 feet, again, they were direct effect of HIV so</p> <p>19 it is only when anti-retroviral therapy came</p> <p>20 along that that made a direct effect on those.</p> <p>21 Where you had a co-infection say with</p> <p>22 herpes zoster or with oral candida then that</p> <p>23 co-infection can be treated, but the</p> <p>24 constitutional symptoms were very difficult.</p> <p>25 PROFESSOR TUDOR-WILLIAMS: It is worth pointing out</p>	<p style="text-align: right;">42</p> <p>1 there's a second page to the document that is on</p> <p>2 the screen at the moment.</p> <p>3 MS SCOTT: Yes. Can we go down to the next page.</p> <p>4 PROFESSOR ANDERSON: Candida.</p> <p>5 MS SCOTT: Yes, so there are more conditions there</p> <p>6 on that second page. Can somebody speak to</p> <p>7 those.</p> <p>8 PROFESSOR ANDERSON: So we talked briefly about</p> <p>9 thrush in the mouth and again people can get</p> <p>10 thrush in the mouth for a variety of reasons --</p> <p>11 PROFESSOR TUDOR-WILLIAMS: Fluconazole was</p> <p>12 available.</p> <p>13 PROFESSOR ANDERSON: We would have used nystatin</p> <p>14 mouthwashes, anvitosin mouthwash, anvitosin</p> <p>15 lozenges, do you remember those? Actually,</p> <p>16 sometimes Canesten pessaries we would use for</p> <p>17 people to suck and it would often give people</p> <p>18 a very sore mouth, be very uncomfortable, white</p> <p>19 tongue, white around the cheeks, lining of the</p> <p>20 cheeks.</p> <p>21 The same for vaginal candida, again more</p> <p>22 difficult to treat than you would expect it to</p> <p>23 be, recurring, not shifting and, yes, there were</p> <p>24 certainly treatments available for vaginal</p> <p>25 candidiasis but not as effective as they are</p>
<p style="text-align: right;">43</p> <p>1 now, that's for sure.</p> <p>2 Cervical dysplasia was added later on as</p> <p>3 it became -- more women became affected or were</p> <p>4 living with HIV. It became clear that more of</p> <p>5 them had got cervical abnormalities on smear</p> <p>6 tests and again they would have had standard</p> <p>7 treatment for cervical dysplasia at the time.</p> <p>8 Now with anti-retroviral therapy often those</p> <p>9 cervical changes go once a woman starts</p> <p>10 anti-retroviral therapy. Peripheral neuropathy</p> <p>11 I think as Ian alluded to, often really hard to</p> <p>12 treat and pain we would have used a variety of</p> <p>13 sometimes Amitryptolene in those days. There</p> <p>14 were not great specific treatments for people at</p> <p>15 that point.</p> <p>16 MS SCOTT: Ms Edwards, did you want to out anything</p> <p>17 about nursing people at this stage of their HIV?</p> <p>18 DR EDWARDS: I think the expression earlier about</p> <p>19 these constitutional symptoms were so very</p> <p>20 difficult and so debilitating in many ways. You</p> <p>21 know, oral candida, for example, is probably the</p> <p>22 classic because I know it looks awful. It's</p> <p>23 awful to treat. It stops you eating. It looks</p> <p>24 horrible. It's a very psychologically difficult</p> <p>25 thing to deal with and, of course, although</p>	<p style="text-align: right;">44</p> <p>1 other people get these infections, people with</p> <p>2 HIV, their immune system wasn't improving so</p> <p>3 they kept coming back. It's not one, you know,</p> <p>4 you are not getting one of these things, you are</p> <p>5 getting one or two or three of them.</p> <p>6 My certain memories and I am sure lots</p> <p>7 have memories of the night sweats, that was</p> <p>8 really bad. You know, the night sweats at</p> <p>9 night, waking up drenched, candida, diarrhoea</p> <p>10 and things and this was before opportunistic</p> <p>11 infections really hit in. This was the early</p> <p>12 days of infection. So, yes, very debilitating</p> <p>13 and psychologically difficult.</p> <p>14 DR WILLIAMS: In terms of things like herpes zoster,</p> <p>15 which is shingles, as Sian said, become</p> <p>16 recurrent but the impact of shingles can leave</p> <p>17 you with very difficult to treat neuropathic</p> <p>18 pain where which is really debilitating. It is</p> <p>19 not just the herpes zoster that can be treated,</p> <p>20 it is the consequences that are really quite</p> <p>21 impactful.</p> <p>22 MS SCOTT: You have mentioned the psychological</p> <p>23 difficulties. Presumably knowing what is</p> <p>24 coming, what this means, adds to the burden</p> <p>25 tremendously to those that were suffering it.</p>

<p style="text-align: right;">45</p> <p>1 PROFESSOR ANDERSON: Absolutely, yes.</p> <p>2 MS SCOTT: I was going to move on now to the AIDS</p> <p>3 itself.</p> <p>4 SIR BRIAN LANGSTAFF: It sounds like something that</p> <p>5 we had better deal with at 12 o'clock, I think.</p> <p>6 12 o'clock.</p> <p>7 (11.30 am)</p> <p>8 (A short break)</p> <p>9 (12.04 pm)</p> <p>10 MS SCOTT: We were going to move on then from the</p> <p>11 chronic infection, so to the middle stage, to</p> <p>12 AIDS itself. You have already given some</p> <p>13 evidence about how you diagnose AIDS.</p> <p>14 We were looking at the table of -- a CDC</p> <p>15 table, where you -- which sets out the clinical</p> <p>16 category of AIDS-defining illnesses.</p> <p>17 You have explained that in order to meet</p> <p>18 a diagnosis of AIDS in the UK you need to have</p> <p>19 one of those AIDS-defining illnesses. Is that</p> <p>20 right?</p> <p>21 PROFESSOR ANDERSON: Correct.</p> <p>22 MS SCOTT: Can you just say a little about -- in</p> <p>23 your report you explain that the way that HIV</p> <p>24 relates to a person's immune system is reliant</p> <p>25 on three factors: the microbial exposure of the</p>	<p style="text-align: right;">46</p> <p>1 patient through life, the pathogenicity of</p> <p>2 organisms encountered, and the degree of</p> <p>3 immunosuppression of the host. Can you just</p> <p>4 explain why those three factors impact</p> <p>5 differently and give people different</p> <p>6 experiences of HIV going through to AIDS.</p> <p>7 PROFESSOR ANDERSON: So I will kick off on that. So</p> <p>8 from what we have discussed so far, I hope it's</p> <p>9 become clear that the clinical -- the things</p> <p>10 that happen to people living with HIV are</p> <p>11 a consequence of the immune system weakening.</p> <p>12 Now, all of us have been exposed to</p> <p>13 microbes and germs during our lives and many of</p> <p>14 us have got different exposures depending where</p> <p>15 in the world we've been or what we've had in our</p> <p>16 lives, and many of those infections may stay</p> <p>17 with us and we don't know we've got them and</p> <p>18 everything's fine until the immune system</p> <p>19 weakens, and then some of those things that we</p> <p>20 have been able to keep in check come back and</p> <p>21 re-emerge.</p> <p>22 So, for example, a particularly prominent</p> <p>23 one might be tuberculosis, and that then might</p> <p>24 be much more common in a person who has lived in</p> <p>25 an area where there is a lot of tuberculosis in</p>
<p style="text-align: right;">47</p> <p>1 the first place, whose immune system becomes</p> <p>2 weak enough for tuberculosis that may have been</p> <p>3 around for a while to emerge, and the pathogen</p> <p>4 itself, the problem that's going to emerge, how</p> <p>5 powerful it is. So a very powerful pathogen</p> <p>6 doesn't need so much immunosuppression to appear</p> <p>7 and it will depend if you have met that pathogen</p> <p>8 before.</p> <p>9 One particular example which we could use</p> <p>10 perhaps is cerebral toxoplasmosis. Now that is</p> <p>11 an infection that lives in meat, and actually if</p> <p>12 you look across the world, people in France, who</p> <p>13 are used to eating meat that is rare, often have</p> <p>14 got much higher levels of having met</p> <p>15 toxoplasmosis in a previous life than other</p> <p>16 parts of the world. So that's where we're</p> <p>17 thinking about the microbial exposure.</p> <p>18 If you have met toxoplasmosis in the past,</p> <p>19 then your immune system becomes weak, that is</p> <p>20 something that is going to come back, with</p> <p>21 a brain abscess most commonly.</p> <p>22 So when -- when -- and I am thinking back</p> <p>23 now, sometimes people will come with very</p> <p>24 undifferentiated symptoms, somebody will come</p> <p>25 with meningitis, which just suggests</p>	<p style="text-align: right;">48</p> <p>1 inflammation of the brain. So which sort of</p> <p>2 meningitis is it going to be? Well, where maybe</p> <p>3 have they lived? What exposure might they have</p> <p>4 had? How immunosuppressed are they? So those</p> <p>5 are the sorts of questions that would have</p> <p>6 guided the diagnostic process, if you like, in</p> <p>7 trying to work out what's happening.</p> <p>8 The more pathogenic things are happening</p> <p>9 at an earlier stage. And, for example,</p> <p>10 things -- there's a particular bacteria called</p> <p>11 microbacterium avium-intracellulare, normally it</p> <p>12 doesn't do anything much at all to people but</p> <p>13 once the immune system is so flat, then it can</p> <p>14 emerge and cause a problem. So that's something</p> <p>15 you would see probably much later on in the</p> <p>16 course.</p> <p>17 So it's trying to have a balance, as you</p> <p>18 are thinking this through, of what somebody</p> <p>19 might have met before, where they are in their</p> <p>20 immune system spectrum and what sort of pathogen</p> <p>21 power there is. But of course, by the time</p> <p>22 somebody's experiencing something from this list</p> <p>23 here, these are all things that really only</p> <p>24 happen when the immune system is really weak.</p> <p>25 They are a way of signalling how weak the immune</p>

<p style="text-align: right;">49</p> <p>1 system has become that somebody is able to have</p> <p>2 this as a problem in their lives, if that sets</p> <p>3 the scene.</p> <p>4 <b>MS SCOTT:</b> Yes.</p> <p>5 Henry, can we turn to Table 2, which is at</p> <p>6 page 33 of the report.</p> <p>7 <b>PROFESSOR ANDERSON:</b> I think the other thing I would</p> <p>8 just say at this point is if we think back,</p> <p>9 making a diagnosis of what it is that's the</p> <p>10 problem is absolutely critical here, because if</p> <p>11 you don't have anti-retroviral drugs to deal</p> <p>12 with the underlying HIV, then the work has to be</p> <p>13 done by the drugs you do have to treat whatever</p> <p>14 infection or tumour it is that is causing the</p> <p>15 problem. So the work of making a diagnosis is</p> <p>16 really important and so people would have gone</p> <p>17 through a lot of different tests and a lot of</p> <p>18 examinations to try and find out what pathogen</p> <p>19 it is if you can't deal with the underlying HIV</p> <p>20 itself.</p> <p>21 <b>MS SCOTT:</b> Sorry, Henry, can we go back to the</p> <p>22 previous page, the page -- yes --</p> <p>23 <b>PROFESSOR COOKE:</b> It is worth making the contrast to</p> <p>24 what we were discussing earlier about some of</p> <p>25 those more common conditions where there were</p>	<p style="text-align: right;">50</p> <p>1 some treatments. Many of these that we're going</p> <p>2 to go through are very severe conditions for</p> <p>3 which many treatments didn't exist, certainly in</p> <p>4 the 80s through to the 90s, and even quite</p> <p>5 recently. So the diagnosis was crucial but</p> <p>6 there was often a limitation in what could be</p> <p>7 offered in terms of intervention.</p> <p>8 <b>MS SCOTT:</b> So I understand from your report that</p> <p>9 there's now effective therapy for HIV and so</p> <p>10 these kinds of treat -- these kinds of</p> <p>11 conditions we're going to look at now aren't</p> <p>12 appearing, in this country in any event.</p> <p>13 <b>PROFESSOR COOKE:</b> Much less frequently.</p> <p>14 <b>MS SCOTT:</b> The Inquiry has heard evidence of people</p> <p>15 suffering these conditions and so can we do the</p> <p>16 same exercise, can we go through the symptoms</p> <p>17 here caused by the major HIV-associated</p> <p>18 pathogens and look at how that would have</p> <p>19 impacted people and the kinds of treatments, if</p> <p>20 any, that were available and the burden of that</p> <p>21 treatment on them.</p> <p>22 Can we start with candidiasis, please.</p> <p>23 <b>PROFESSOR ANDERSON:</b> I think Sian's already alluded</p> <p>24 to how painful it can be to have thrush in your</p> <p>25 mouth but if your immune system is even weaker</p>
<p style="text-align: right;">51</p> <p>1 then it can spread down into the oesophagus,</p> <p>2 which is the tube where you are swallowing food.</p> <p>3 So a very common complaint people would talk</p> <p>4 about was how difficult it was to swallow, to</p> <p>5 eat, a feeling of food being stuck, and pain,</p> <p>6 real pain as you are eating. Then that would</p> <p>7 be, I guess, a bit more (unclear) but you could</p> <p>8 actually find thrush getting into the lungs</p> <p>9 which would make you cough, short of breath. So</p> <p>10 imagining a sort of layer of fungus going</p> <p>11 through the hollow tubes in the oesophagus and</p> <p>12 the bronchi, which really -- pain, discomfort,</p> <p>13 difficulty breathing, and I think what you said</p> <p>14 earlier, the impact on appetite and your ability</p> <p>15 to enjoy food or eat properly.</p> <p>16 I don't know if -- Sian, you might want to</p> <p>17 add to that --</p> <p>18 <b>MS EDWARDS:</b> I don't think there's anything to</p> <p>19 add -- yes, I mean, I think anybody whose --</p> <p>20 most people who had AIDS actually had candida,</p> <p>21 it seemed incredibly common, and of course when</p> <p>22 people are sick, what you want to do is feed</p> <p>23 them, and no-one wanted to eat. And, you know,</p> <p>24 there's many, many reasons, including the virus</p> <p>25 itself, that people lost weight, but the extent</p>	<p style="text-align: right;">52</p> <p>1 of weight loss amongst this group of patients</p> <p>2 was extreme and debilitating and -- and</p> <p>3 extraordinarily painful for family members to --</p> <p>4 to watch.</p> <p>5 <b>PROFESSOR COOKE:</b> And even in -- in the setting of</p> <p>6 advanced immunosuppression, even a condition</p> <p>7 like candidiasis can be fatal. So you can have</p> <p>8 bloodstream infection, which can be fatal,</p> <p>9 and -- and many of these conditions we'll go</p> <p>10 through we'll talk about symptoms but in their</p> <p>11 most advanced conditions can be fatal.</p> <p>12 <b>MS SCOTT:</b> When it gets to this stage was there</p> <p>13 effective medication?</p> <p>14 <b>PROFESSOR COOKE:</b> There are usually medications now</p> <p>15 for this particular condition, candidiasis, that</p> <p>16 would be effective. We're seeing more</p> <p>17 resistance these days to some of these</p> <p>18 medications. But -- generally speaking -- but</p> <p>19 these are really relatively recent treatments</p> <p>20 that are available now.</p> <p>21 <b>MS SCOTT:</b> Going down the list then, cryptococcal</p> <p>22 infection.</p> <p>23 <b>PROFESSOR ANDERSON:</b> So cryptococcus is most</p> <p>24 common -- it's a fungus. It most commonly in</p> <p>25 the context of HIV infection would cause</p>



<p style="text-align: right;">53</p> <p>1 meningitis, a particularly nasty meningitis, so</p> <p>2 meningitis would then often present with -- in</p> <p>3 this particular case, awful headache, change in</p> <p>4 conscious level, very debilitating. And one</p> <p>5 particular feature of cryptococcus is it sort of</p> <p>6 clogs up -- the fluid round your brain can't</p> <p>7 flow properly down and away. So people often</p> <p>8 would get hydrocephalus and -- and very complex</p> <p>9 meningitis from a cryptococcal infection.</p> <p>10 <b>PROFESSOR TUDOR-WILLIAMS:</b> The treatment required</p> <p>11 repetitive lumbar punctures to decrease the</p> <p>12 pressure, which was highly invasive and</p> <p>13 discomfiting.</p> <p>14 <b>DR WILLIAMS:</b> And the consequence if you didn't do</p> <p>15 that properly was blindness. People would lose</p> <p>16 their sight. So cryptococcal infection is one</p> <p>17 of the most severe opportunistic infections we</p> <p>18 see, with a high mortality. Getting it early</p> <p>19 was very important. People presenting late had</p> <p>20 -- you know, their outcome was in the early days</p> <p>21 was not very good.</p> <p>22 <b>PROFESSOR ANDERSON:</b> I would also add the early --</p> <p>23 the treatments for fungal infections, they have</p> <p>24 advanced a lot but the sorts of treatment we</p> <p>25 might have had available when we were seeing</p>	<p style="text-align: right;">54</p> <p>1 a lot of -- they were really unpleasant for</p> <p>2 terosin, very toxic treatments. So even if</p> <p>3 there was an anti-fungal agent that you might</p> <p>4 want to use, it would have been very unpleasant.</p> <p>5 <b>PROFESSOR TUDOR-WILLIAMS:</b> It's also a really good</p> <p>6 example of a pathogen that's prevalent in the</p> <p>7 community. You didn't have to go further than</p> <p>8 a London park because pigeons, and pigeon poo</p> <p>9 particularly, was implicated in the spread of</p> <p>10 cryptococcosis, and again it comes back to the</p> <p>11 idea that probably all of us in this room who</p> <p>12 have ever walked through a London park have got</p> <p>13 exposed to cryptococcus but if your immune</p> <p>14 system is working okay it doesn't cause any</p> <p>15 problems but if your immune system is damaged it</p> <p>16 causes this really debilitating meningitis.</p> <p>17 <b>MS SCOTT:</b> I daren't try and pronounce the next one</p> <p>18 down. Does somebody else --</p> <p>19 <b>PROFESSOR ANDERSON:</b> Cryptosporidiosis.</p> <p>20 <b>MS SCOTT:</b> -- Want to have a go?</p> <p>21 <b>DR WILLIAMS:</b> Cryptosporidiosis.</p> <p>22 <b>MS SCOTT:</b> Can somebody speak to that?</p> <p>23 <b>PROFESSOR ANDERSON:</b> Well, again, it's another one</p> <p>24 of those infections that really indicates very,</p> <p>25 very profound immunosuppression, and it's --</p>
<p style="text-align: right;">55</p> <p>1 again, as Gareth has said, it's a thing that's</p> <p>2 knocking around in water supplies in any event,</p> <p>3 but we would see it in the context of HIV, often</p> <p>4 really debilitating, causing terrible, terrible</p> <p>5 diarrhea, very watery diarrhoea, which was</p> <p>6 sometimes with a crampy abdominal pain, but the</p> <p>7 cardinal feature was that people lost a huge</p> <p>8 amount of weight and fluid and -- and really,</p> <p>9 really intractable diarrhoea. And I think the</p> <p>10 most serious issues around cryptosporidial</p> <p>11 diarrhoea were really profound. And it was</p> <p>12 a horrid -- horrible infection to have.</p> <p>13 <b>MS SCOTT:</b> Do you want to add anything to that?</p> <p>14 <b>MS EDWARDS:</b> It -- I think it's -- this was the --</p> <p>15 I'm actually sitting here listening to this and</p> <p>16 memories are coming back. And I was a nurse so</p> <p>17 I can't imagine what people out there are</p> <p>18 feeling. I'm -- going through these is quite</p> <p>19 I think possibly quite traumatic because I think</p> <p>20 our memories of how people -- because, as I said</p> <p>21 earlier, it isn't one: people had candidiasis</p> <p>22 and they had cryptosporidiosis, you know, and</p> <p>23 they had something else. And these -- these --</p> <p>24 it is a ruthless -- in some countries it's</p> <p>25 a ruthless disease, and the impact of --</p>	<p style="text-align: right;">56</p> <p>1 certainly of cryptosporidiosis from a nursing</p> <p>2 and medical point of view to try and control</p> <p>3 this intractable diarrhoea meant that people</p> <p>4 couldn't go out, they couldn't leave the toilet.</p> <p>5 It was a dreadful, dreadful disease, which was</p> <p>6 very difficult from a medical point of view to</p> <p>7 control. And again, the families, it was a very</p> <p>8 hard thing to watch and care for people who had</p> <p>9 this condition, which was again common.</p> <p>10 <b>PROFESSOR TUDOR-WILLIAMS:</b> And we really didn't have</p> <p>11 useful treatment.</p> <p>12 <b>PROFESSOR COOKE:</b> Probably it's the best example of</p> <p>13 where we still really don't, and the thing</p> <p>14 that's transformed what we can do is the advent</p> <p>15 of HIV treatment itself. We still have very</p> <p>16 poor treatment for cryptosporidiosis and we</p> <p>17 desperately need it.</p> <p>18 <b>MS SCOTT:</b> CMV?</p> <p>19 <b>PROFESSOR ANDERSON:</b> Where shall we start? CMV.</p> <p>20 Goodness.</p> <p>21 Graham?</p> <p>22 <b>PROFESSOR COOKE:</b> So CMV is a viral infection. It</p> <p>23 belongs to a similar family to glandular fever</p> <p>24 and many people are exposed to it from early in</p> <p>25 life and increasingly exposed particularly</p>

<p style="text-align: right;">57</p> <p>1 through adolescence and early adulthood.</p> <p>2 Again, it's an infection that many of us</p> <p>3 handle without any great problem if our immune</p> <p>4 systems are good but in the setting of</p> <p>5 progressive weakening of the immune system, that</p> <p>6 virus can reactivate, and it's the reactivation</p> <p>7 of that virus that can be very debilitating.</p> <p>8 And often with advanced infection one of</p> <p>9 the more common thing we used to see, and</p> <p>10 thankfully see much less of, is -- was damage to</p> <p>11 the back of the eye, which can really affect</p> <p>12 vision. It's a good example of something that</p> <p>13 could then be long-term, even with effective</p> <p>14 treatment and recovery of the immune system</p> <p>15 subsequently.</p> <p>16 You can see reactivation in other parts of</p> <p>17 the body where it can also have very devastating</p> <p>18 effects in terms of the gut, and you can have</p> <p>19 a very inflamed gut, colon, and diarrhoea which</p> <p>20 can be very hard to treat and painful,</p> <p>21 reactivation at the upper end of the gut, in the</p> <p>22 oesophagus, where it can be very painful, and</p> <p>23 then reactivation in the lung as well where</p> <p>24 it -- a bit like pneumonia but you can have</p> <p>25 inflammation in the lung which can make</p>	<p style="text-align: right;">58</p> <p>1 breathing difficult and getting oxygen in.</p> <p>2 It is an example where we actually have</p> <p>3 got much better treatment now but some of those</p> <p>4 earlier treatments that we used were very toxic,</p> <p>5 and so, again, it's the sort of recurring theme</p> <p>6 of individuals who had very advanced HIV, were</p> <p>7 very sick, with really the only options for</p> <p>8 treatment being very toxic medications which we</p> <p>9 were obliged to use really.</p> <p>10 <b>PROFESSOR ANDERSON:</b> And because of the toxicity to</p> <p>11 the veins, we often used to have to implant what</p> <p>12 were called port-a-caths. So a person might</p> <p>13 have an in-dwelling venous access which would</p> <p>14 often be on the chest wall so that the drug</p> <p>15 could go into a big vein because it would be too</p> <p>16 damaging to the veins to go into the arm, and so</p> <p>17 often people would have to have a surgical</p> <p>18 procedure to have a thing called a port-a-cath</p> <p>19 fitted. And then that needed to be looked after</p> <p>20 and managed as well as getting the medication in</p> <p>21 as well. So it was really complicated.</p> <p>22 <b>PROFESSOR TUDOR-WILLIAMS:</b> Also cytomegalovirus is</p> <p>23 a really big issue for pregnant women. So in</p> <p>24 the context of HIV, that was a co-infection that</p> <p>25 we saw really too commonly in the infants born</p>
<p style="text-align: right;">59</p> <p>1 to mothers who were HIV infected. And I just</p> <p>2 remember treating far too many children who had</p> <p>3 gone blind as a result of HIV and CMV</p> <p>4 co-infection.</p> <p>5 And -- and I'm sure you remember that in</p> <p>6 the London Lighthouse, there may be people in</p> <p>7 this room who were involved in planting a garden</p> <p>8 that was full of different sensory plants with</p> <p>9 really lovely aromas because of -- because</p> <p>10 retinitis had blinded quite a number of people</p> <p>11 who were in-patients at the London Lighthouse.</p> <p>12 And I always remember thinking that was</p> <p>13 a tremendously wonderful humane response to what</p> <p>14 was a terrible co-infection.</p> <p>15 <b>DR WILLIAMS:</b> I think this is really -- the impact</p> <p>16 of CMV in the eye, you -- they would get -- one</p> <p>17 episode was treated, they would have to remain</p> <p>18 on treatments to try to prevent relapse, but</p> <p>19 then they -- ultimately they would then get</p> <p>20 other another relapse, and at each relapse there</p> <p>21 would be loss of vision. And so many people who</p> <p>22 developed CMV retinitis before they died they</p> <p>23 were -- they went completely blind. It was</p> <p>24 a really debilitating, horrible thing.</p> <p>25 <b>MS SCOTT:</b> Encephalitis?</p>	<p style="text-align: right;">60</p> <p>1 <b>PROFESSOR COOKE:</b> Not much better. So -- so HIV we</p> <p>2 know can directly affect the brain both in acute</p> <p>3 infection and chronic infection, and that can</p> <p>4 lead to quite a significant clinical</p> <p>5 manifestation of confusion or even more</p> <p>6 progressive cognitive sort of challenges with</p> <p>7 thinking and memory and so forth, which in</p> <p>8 advanced HIV could -- could be very profound.</p> <p>9 And even with the advent of effective HIV</p> <p>10 therapy, not always reversible. So it is still</p> <p>11 an issue that we have to contend with in clinics</p> <p>12 today, often to a milder degree thankfully but</p> <p>13 nonetheless still something that is sort of an</p> <p>14 active area of interest in particular.</p> <p>15 <b>MS SCOTT:</b> You touched on herpes zoster earlier.</p> <p>16 Here we have herpes simplex. Is there anything</p> <p>17 you want to add to that?</p> <p>18 <b>PROFESSOR ANDERSON:</b> Herpes simplex is an infection</p> <p>19 the skin, of the genitals, of the mucus</p> <p>20 membranes. You don't have to have had chicken</p> <p>21 pox to get it. So zoster is related to the</p> <p>22 chicken pox virus but herpes simplex -- many</p> <p>23 people have it and it doesn't cause problems,</p> <p>24 again, until the immune system becomes weak.</p> <p>25 But it's one of the ones that's quite a powerful</p>

<p style="text-align: right;">61</p> <p>1 pathogen. So, again, it would come quite early,  2 and I think the level of painful ulceration --  3 and, again, if you've got some oral candida and  4 herpes simplex in the mouth, exquisitely --  5 really painful and -- and very difficult, often,  6 to manage. So, again, it would be one of the  7 things that we did have some treatments which  8 you might then ask somebody to take long-term to  9 try and keep it under control. So, again,  10 adding another layer of medication, both to  11 treat and to try and prevent recurrence.</p> <p>12 <b>MS SCOTT:</b> Histoplasmosis.</p> <p>13 <b>PROFESSOR COOKE:</b> So it's another relatively unusual  14 infection in this case but, again, really we do  15 see occasionally in people who have healthy  16 immune systems but relatively rarely, but when  17 it -- when it did affect people with advanced  18 HIV -- we don't see it very often these days at  19 all, and now fortunately we have better  20 treatments for it, but it was a condition that  21 had a very bad outcome in the setting of some of  22 the treatments we had available at that time,  23 and these anti-fungal drugs we have already  24 mentioned were often, when they were available,  25 quite difficult to -- to take and -- and to use.</p>	<p style="text-align: right;">62</p> <p>1 And so -- yeah, so fortunately a condition  2 that we see much less of but nonetheless often  3 very serious, if not fatal.</p> <p>4 <b>MS SCOTT:</b> Kaposi's sarcoma. That's something  5 you've said in your report is not something  6 that's seen very much in this -- in those that  7 are infected through blood and blood products.  8 Is it seen in that cohort of patients?</p> <p>9 <b>MS EDWARDS:</b> It depends on the lifestyle.</p> <p>10 <b>PROFESSOR ANDERSON:</b> It depends whether people have  11 been exposed -- again, it has a viral origin.  12 It's related to some of the other herpes viruses  13 so it can present itself, but it is much less  14 common, but when it happens, it -- it's on the  15 skin, it's very prominent often. People have  16 lesions that appear that are very easy to see  17 and can get into the gut and into the lungs and  18 cause severe systemic problems as well.</p> <p>19 <b>MS EDWARDS:</b> It is, of course, the early look of  20 AIDS that many films are based on with people  21 who have these purple blotches all over their  22 bodies and skin, and was deeply stigmatising for  23 people. And if there was any way -- with some  24 of these things you were able not to be known as  25 having HIV infection and AIDS; if you had</p>
<p style="text-align: right;">63</p> <p>1 Kaposi's sarcoma, there's no way.</p> <p>2 <b>SIR BRIAN LANGSTAFF:</b> If my understanding is right,  3 then it was Kaposi's sarcoma that was  4 a particular identifier of the five adults who  5 gave rise to the first report in the western  6 world of AIDS.</p> <p>7 <b>PROFESSOR ANDERSON:</b> Indeed.</p> <p>8 <b>SIR BRIAN LANGSTAFF:</b> I think -- am I right in  9 thinking that it was because of a specific drug  10 that was really only used for that that the CDC  11 was able to investigate why that drug was being  12 used so much in that particular --</p> <p>13 <b>PROFESSOR ANDERSON:</b> That was pneumocystis, the next  14 one along.</p> <p>15 <b>MS EDWARDS:</b> But they came together.</p> <p>16 <b>PROFESSOR ANDERSON:</b> They came at the same time and  17 people would often have both.</p> <p>18 <b>SIR BRIAN LANGSTAFF:</b> Thank you.</p> <p>19 <b>MS SCOTT:</b> Lymphoma?</p> <p>20 <b>PROFESSOR ANDERSON:</b> Lymphoma, where shall we start?  21 Lymphoma is a cancer, it's a cancer that  22 has probably -- it's got an infectious aetiology  23 but again we're seeing it much more commonly.  24 These are cancers that can occur in people  25 without HIV but much more common with HIV, and</p>	<p style="text-align: right;">64</p> <p>1 the fact I think -- that the CNS, the brain --  2 the lymphoma that affected the brain  3 particularly relates to co-infection with HIV.</p> <p>4 Hard to treat and, again, going back to  5 some of the chemotherapeutic approaches, some of  6 the oncology approaches, often -- it was often  7 fatal.</p> <p>8 <b>DR WILLIAMS:</b> The other thing about lymphomas,  9 lymphomas are seen in people who have other  10 immunosuppression. So it's people who have had  11 transplants who get a much higher risk of  12 lymphoma. I mean, really, there is lymphoma --  13 when it presented, it usually presented with  14 advanced lymphoma rather than early, and  15 therefore the outcome of treatment was often  16 very difficult -- you know, it was very much  17 worse.</p> <p>18 But lymphoma -- now, treatment of lymphoma  19 in someone who is HIV positive, the outcome is  20 no different to someone who is HIV negative.  21 There has been, you know, a marked improvement  22 in that.</p> <p>23 <b>MS SCOTT:</b> Can I just invite you to look down the  24 rest of that list. Are there any of those  25 symptoms or pathogens that were particularly</p>



<p style="text-align: right;">65</p> <p>1 prevalent for those that have been affected by</p> <p>2 bleeding or blood disorders or -- that you would</p> <p>3 like to talk to because of the burden of</p> <p>4 treatment or the burden of the condition?</p> <p>5 <b>PROFESSOR ANDERSON:</b> I think we have to include</p> <p>6 pneumocystis here because that was probably one</p> <p>7 of the most common AIDS-defining conditions, and</p> <p>8 that is the one that you are referring to.</p> <p>9 A lung infection most commonly, although it</p> <p>10 could affect other parts, and often very slow to</p> <p>11 present itself. So quite a long time feeling</p> <p>12 short of breath, bit of a cough, not necessarily</p> <p>13 that much to see but actually by the time people</p> <p>14 came to medical attention often very short of</p> <p>15 breath, feeling very -- it was a very</p> <p>16 uncomfortable sensation, not bring able to</p> <p>17 breathe and shortness of breath on exertion.</p> <p>18 And it -- one -- perhaps one of the</p> <p>19 commonest opportunistic -- and earliest.</p> <p>20 <b>PROFESSOR TUDOR-WILLIAMS:</b> Rather than affecting the</p> <p>21 lung airway, it was affecting the lung tissue</p> <p>22 and preventing gas exchange so that certainly</p> <p>23 the children I looked after had very profound</p> <p>24 low levels of oxygen, which makes you feel</p> <p>25 horrible, and it was all part of that systemic</p>	<p style="text-align: right;">66</p> <p>1 illness.</p> <p>2 <b>PROFESSOR ANDERSON:</b> Yes.</p> <p>3 What I would say is that we quite -- even</p> <p>4 before we had very effective anti-retroviral</p> <p>5 therapy it became quite clear that taking</p> <p>6 regular low doses of an antibiotic called</p> <p>7 septrin could actually help prevent this</p> <p>8 occurring, and so, again, another drug that you</p> <p>9 might add in not to deal with the HIV but to try</p> <p>10 and prevent this particular infection, but would</p> <p>11 add to the burden of medications that people</p> <p>12 might be taking. And people with HIV often had</p> <p>13 high levels of allergies to drugs, and septrin</p> <p>14 in particular would be something -- people would</p> <p>15 start septrin and come out in a rash and have</p> <p>16 a drug reaction, as well as everything else.</p> <p>17 <b>MS SCOTT:</b> The oral evidence the Inquiry has</p> <p>18 received from some of the people who experienced</p> <p>19 some of these conditions and then recovered and</p> <p>20 are alive today is that they suffered ongoing</p> <p>21 damage as a result of these conditions. So,</p> <p>22 despite having been cured, they continue to</p> <p>23 exhibit symptoms. Is that right?</p> <p>24 <b>PROFESSOR COOKE:</b> I think that's particularly true</p> <p>25 for a couple of the conditions we haven't dealt</p>
<p style="text-align: right;">67</p> <p>1 with, so progressive multifocal</p> <p>2 leukoencephalopathy and toxoplasmosis are both</p> <p>3 conditions that affect the brain, caused by</p> <p>4 different pathogens that reactivate in advanced</p> <p>5 immunosuppression. But I -- you know, I still</p> <p>6 have patients in my clinic who are now</p> <p>7 relatively well, with -- with (unclear) vent</p> <p>8 strokes from conditions like this, so very</p> <p>9 profound long-term disability that can result</p> <p>10 even when HIV can be treated.</p> <p>11 <b>PROFESSOR ANDERSON:</b> Yes, so CMV, cryptococcus,</p> <p>12 toxoplasmosis, would all have long-term, lasting</p> <p>13 sequelae, even if the acute episode had been</p> <p>14 dealt with.</p> <p>15 <b>MS EDWARDS:</b> It may be just worth noting, as Jane</p> <p>16 said, each of these things it's not one, it's</p> <p>17 many, and for all of them there are treatments</p> <p>18 that are being tried and trials that are being</p> <p>19 tried on some of these infections in these early</p> <p>20 days but the treatments themselves were often</p> <p>21 very, very difficult, so there were further</p> <p>22 treatments, like anti-nausea treatments and such</p> <p>23 like to counteract the side effects of</p> <p>24 treatments. So it's just to sort of put in to</p> <p>25 perspective that people are very sick with</p>	<p style="text-align: right;">68</p> <p>1 multiple infections, with multiple treatments,</p> <p>2 and side effects and constant, and so the amount</p> <p>3 of treatments that people were taking was</p> <p>4 extraordinary.</p> <p>5 <b>PROFESSOR ANDERSON:</b> Following on from that as well,</p> <p>6 the numbers of times people would be in</p> <p>7 hospital. So this would, you know, you have --</p> <p>8 something would happen, you come into hospital,</p> <p>9 you would have an episode of treatment, go home,</p> <p>10 confirm back with something else. So there was</p> <p>11 a lot of people coming back and forth, as well</p> <p>12 as the treatment they were on, but hospitals as</p> <p>13 places of people coming back and forth to much</p> <p>14 more regularly.</p> <p>15 <b>DR WILLIAMS:</b> People might present with pneumocystis</p> <p>16 and you might be able to prevent that again but</p> <p>17 then they may then present with cytomegalovirus</p> <p>18 retinitis and you need treatment for that to</p> <p>19 prevent that and then they might present with</p> <p>20 toxoplasmosis and stroke and then lastly present</p> <p>21 with MEI, so there was a series of very severe</p> <p>22 opportunist infections that required</p> <p>23 preventative therapy and treatment but also very</p> <p>24 debilitating.</p> <p>25 <b>MS SCOTT:</b> You have also in your report drawn</p>

<p style="text-align: right;">69</p> <p>1 attention to the fact that there are other</p> <p>2 health complications which are observed in those</p> <p>3 that suffer from HIV. I am talking at page 25.</p> <p>4 So other than AIDS-defining illnesses, you</p> <p>5 observe that there are other health</p> <p>6 complications that are observed and one of the</p> <p>7 ones that you draw attention to is epilepsy,</p> <p>8 which can develop independently of HIV but also</p> <p>9 be caused to be linked to it.</p> <p>10 Can you tell us a little bit about that.</p> <p>11 It's the second paragraph down in page 25.</p> <p>12 <b>PROFESSOR ANDERSON:</b> So epilepsy is a seizure</p> <p>13 disorder and there can be multiple causes of</p> <p>14 seizures, so I think what we're trying to draw</p> <p>15 attention to here is that there are many of the</p> <p>16 clinical editions we have been discussing here</p> <p>17 which would be related to seizures, cryptococcal</p> <p>18 meningitis, toxoplasmosis, lymphoma, could all</p> <p>19 have an element of seizure activity around them</p> <p>20 and somebody may also have epilepsy, idiopathic</p> <p>21 epilepsy, as well or separately. So I think</p> <p>22 that the sense that we're trying to draw</p> <p>23 attention to here is you may find some of these</p> <p>24 sorts of disorders in parallel.</p> <p>25 <b>PROFESSOR COOKE:</b> I think it is worth clarifying for</p>	<p style="text-align: right;">70</p> <p>1 epilepsy that really we think of it, as you say,</p> <p>2 as a consequence of brain infection and a focus</p> <p>3 of brain infection generally in the HIV positive</p> <p>4 population.</p> <p>5 Clearly anyone can get epilepsy and it's</p> <p>6 relatively common in the general population but</p> <p>7 in the absence of an aggressive brain infection</p> <p>8 then it's relatively uncommon. Probably</p> <p>9 wouldn't be of greatly increased prevalence.</p> <p>10 <b>PROFESSOR TUDOR-WILLIAMS:</b> Certainly from</p> <p>11 a paediatric perspective, and I think it's true</p> <p>12 in adults, that HIV per se, the encephalopathy</p> <p>13 associated with the HIV virus did not cause</p> <p>14 seizures. It usually suggested there was</p> <p>15 something else going on. That was one of the</p> <p>16 problems, that you then do a CT or -- I can't</p> <p>17 remember when we started doing MRI scans, but</p> <p>18 you will see a lesion inside the brain that</p> <p>19 would be the likely cause but you didn't know</p> <p>20 whether that was lymphoma or toxoplasmosis and</p> <p>21 you'd take an educated guess and you'd treat the</p> <p>22 toxo and if it didn't go away then you presumed</p> <p>23 that it was probably lymphoma.</p> <p>24 I mean, it was not -- it doesn't lend</p> <p>25 itself to doing biopsies and getting tissue</p>
<p style="text-align: right;">71</p> <p>1 diagnoses. It was a really big diagnostic</p> <p>2 challenge back in the early '90s.</p> <p>3 <b>DR WILLIAMS:</b> I am not sure if we are going to go on</p> <p>4 to it later but the other important things,</p> <p>5 these are the AIDS-defining conditions, but</p> <p>6 there are other HIV associated co-morbidities</p> <p>7 which became much more apparent as treatment</p> <p>8 became more effective. I don't know whether you</p> <p>9 wanted to --</p> <p>10 <b>MS SCOTT:</b> Yes, now would be a good time. That was</p> <p>11 my next question. Can you tell us about that.</p> <p>12 <b>PROFESSOR ANDERSON:</b> We're now in the position that</p> <p>13 if you can with anti-retrovirals ensure that the</p> <p>14 immune system remains strong enough for the</p> <p>15 things we have been discussing just now to not</p> <p>16 occur, then there is time for other things to</p> <p>17 begin to emerge, which in the early days we</p> <p>18 didn't necessarily see because people died of</p> <p>19 some of the things we're talking about now.</p> <p>20 It's clear, I think one of the things that</p> <p>21 Ian referred to earlier, is that with HIV the</p> <p>22 body's in a constant state of inflammation, if</p> <p>23 you like. There's an excessive amount of</p> <p>24 inflammatory response going on. That seems to</p> <p>25 predispose to higher rates of cardiovascular</p>	<p style="text-align: right;">72</p> <p>1 disease, some of the metabolic disorders, bone</p> <p>2 disease, so there's a link between viral</p> <p>3 activity and inflammation.</p> <p>4 On top of that, we know that there are</p> <p>5 some behavioural aspects. So more people living</p> <p>6 with HIV tend to smoke, and that may predispose</p> <p>7 to some of these other -- there may be some</p> <p>8 lifestyle issues as well and we also know that</p> <p>9 the medications that are used in the treatment</p> <p>10 of HIV and its complications can themselves</p> <p>11 precipitate -- particularly, bone disorders are</p> <p>12 particularly common, so we see more long-term</p> <p>13 chronic conditions now because people are alive</p> <p>14 and this sort of low grade inflammatory response</p> <p>15 is continuing.</p> <p>16 <b>DR WILLIAMS:</b> Treatment is very effective in terms</p> <p>17 of getting the immune system better but there's</p> <p>18 still ongoing background of inflammation and</p> <p>19 that inflammation is thought to drive some of</p> <p>20 these increases in risk of other chronic</p> <p>21 diseases of ageing such as kidney disease, such</p> <p>22 as heart disease, such as liver disease and</p> <p>23 cancer, so we are seeing far more increase in</p> <p>24 non-AIDS defining cancers, like lung cancer is</p> <p>25 more common in people, skin cancers, and cancer</p>

<p style="text-align: right;">73</p> <p>1 is now by far the leading cause of death or</p> <p>2 mortality in someone with HIV not the</p> <p>3 AIDS-defining conditions.</p> <p>4 Equally, if you look at cardiovascular</p> <p>5 disease, HIV is thought to be equivalent to</p> <p>6 smoking in terms of risk of cardiovascular</p> <p>7 disease, to give you a sense of the risk.</p> <p>8 <b>MS SCOTT:</b> The increased risk of these other health</p> <p>9 complications is increased as against the</p> <p>10 HIV-negative population.</p> <p>11 <b>DR WILLIAMS:</b> Yes, absolutely. So yes we've dealt</p> <p>12 with very effectively in terms of improving the</p> <p>13 immune system but we're left with a population</p> <p>14 where they have a much more increased risk of</p> <p>15 other chronic diseases of ageing compared to the</p> <p>16 HIV negative population. So although life</p> <p>17 expectancy has improved this is often associated</p> <p>18 with increased frailty and impact of quality of</p> <p>19 life.</p> <p>20 <b>MS SCOTT:</b> Do you know what the increased risk is?</p> <p>21 I know it's on a population level but increased</p> <p>22 risk as against those that don't have HIV?</p> <p>23 <b>DR WILLIAMS:</b> In terms of, for example,</p> <p>24 cardiovascular disease then there's a sort of --</p> <p>25 it's the same as if you were a smoker so if you</p>	<p style="text-align: right;">74</p> <p>1 are a smoker then your risk of cardiovascular</p> <p>2 disease goes up. In chronic kidney disease your</p> <p>3 rate of decline in renal function is faster than</p> <p>4 in someone who is HIV negative.</p> <p>5 <b>MS SCOTT:</b> So you have set out the burden, if I can</p> <p>6 put it that way, of the virus itself, the</p> <p>7 AIDS-defining illnesses and the invasive</p> <p>8 treatments for those particularly in the '80s</p> <p>9 and '90s, the treatment for the medication for</p> <p>10 the ART, anti-retroviral medication, and these</p> <p>11 other associated health conditions. Can we talk</p> <p>12 now then about, and we've touched on this, the</p> <p>13 psychological impacts of having HIV and AIDS and</p> <p>14 again we've touched on the stigma of that as</p> <p>15 well.</p> <p>16 <b>DR JOHNSTON:</b> I think in the community for people to</p> <p>17 live with HIV, if we remember back, and I often</p> <p>18 think of the adverts that were on at that time,</p> <p>19 there was a huge stigma associated with it.</p> <p>20 I think if we think perhaps of the corona virus</p> <p>21 now and then think of the people who get corona</p> <p>22 virus being somehow stigmatised. It's that sort</p> <p>23 of thing. So there was a huge stigma attached</p> <p>24 to that.</p> <p>25 Dealing with a chronic life-shortening</p>
<p style="text-align: right;">75</p> <p>1 illness obviously has psychological impact and</p> <p>2 then you add to that dealing with the putting</p> <p>3 a stone in a pond the ripples, so you are</p> <p>4 dealing with your family, you are dealing with</p> <p>5 the lack of support, the lack of perceived</p> <p>6 sympathy from the wider population sometimes,</p> <p>7 and against the background of almost national</p> <p>8 hysteria. So all the elements were there</p> <p>9 I think to really make for a very</p> <p>10 psychologically damaging and difficult time and</p> <p>11 all of that enhanced the stigma that was</p> <p>12 associated with it.</p> <p>13 <b>MS EDWARDS:</b> Just to add, and I think Gareth can do</p> <p>14 this better than me, but of course the people we</p> <p>15 are dealing with are often children and</p> <p>16 adolescents who were going through, as we were</p> <p>17 talking about on Tuesday, huge changes in their</p> <p>18 own lives and separation which should have been</p> <p>19 a healthy separation from families, parents and</p> <p>20 trying to build their lives and their futures</p> <p>21 which, when we have just described what some</p> <p>22 people were going through, was near to</p> <p>23 impossible and also created great dependency, of</p> <p>24 course, on the family. Maybe Gareth can talk</p> <p>25 more about that.</p>	<p style="text-align: right;">76</p> <p>1 It's really important that we think that</p> <p>2 many of us dealt lots with adults who are</p> <p>3 infected but there is a high proportion of these</p> <p>4 young people, children and 16/15-year olds.</p> <p>5 <b>PROFESSOR TUDOR-WILLIAMS:</b> I like David's analogy of</p> <p>6 the ripples spreading outwards. Certainly we've</p> <p>7 seen -- we're always at pains to point out if</p> <p>8 a child was not infected, were they okay? No</p> <p>9 they were affected by what was going on within</p> <p>10 the family so, yes, it's a very real issue.</p> <p>11 <b>DR WILLIAMS:</b> I think we really can't overestimate</p> <p>12 the impact of psychological problems in somebody</p> <p>13 who is HIV positive. It really is something</p> <p>14 that is core and runs through the whole life of</p> <p>15 someone who is HIV positive. Some people will</p> <p>16 adjust to that diagnosis quite quickly. Others</p> <p>17 take a long time to adjust to that.</p> <p>18 Even in surveys of positive voices,</p> <p>19 a survey produced by Public Health England, even</p> <p>20 now looking at the incidence of mental health</p> <p>21 disorders, it is 30 per cent in the patient</p> <p>22 population with HIV compared to only</p> <p>23 a few per cent in HIV negative population, so</p> <p>24 I really cannot overestimate the impact of</p> <p>25 mental health problems.</p>



<p style="text-align: right;">77</p> <p>1 <b>PROFESSOR ANDERSON:</b> Absolutely.</p> <p>2 <b>MS EDWARDS:</b> Sorry, I was just going to say one</p> <p>3 other thing and I think that that is the issue</p> <p>4 of stigma, which again we talked about on</p> <p>5 Tuesday in great depth and mentioned is that</p> <p>6 people hid away. Most people when you have</p> <p>7 ill-health you see your doctor, your nurse, your</p> <p>8 community, and you are able to share this pain</p> <p>9 whether it be physical or psychological and you</p> <p>10 get support and people care and sympathise, and</p> <p>11 HIV is unusual and awful because of the fact</p> <p>12 that so many people were unable to do that</p> <p>13 sharing and often lived in isolated communities</p> <p>14 and were unable to share what they were going</p> <p>15 through and the psychological burden on not</p> <p>16 being able to share the pain psychologically or</p> <p>17 physically going is huge for people.</p> <p>18 <b>PROFESSOR ANDERSON:</b> That in the next, now we're</p> <p>19 down to that, the sense that the fear that you</p> <p>20 might transmit or pass the HIV on to somebody</p> <p>21 else might actually make people go into</p> <p>22 themselves even more and avoid forming new</p> <p>23 relationships and so becoming even further</p> <p>24 isolated.</p> <p>25 <b>MS SCOTT:</b> The impact of going through those</p>	<p style="text-align: right;">78</p> <p>1 psychological and emotional processes while one</p> <p>2 is physically unwell, what is the impact on</p> <p>3 one's physical health of those mental</p> <p>4 psychological and emotional difficulties?</p> <p>5 <b>DR JOHNSTON:</b> Sorry, just before we go on to that</p> <p>6 I would also say for the wider family members,</p> <p>7 because quite often people who had HIV and</p> <p>8 subsequently AIDS and would have been seen at</p> <p>9 that time as having a terminal diagnosis came</p> <p>10 home to die and they often came to more isolated</p> <p>11 communities or where there was even more stigma</p> <p>12 attached and those people subsequently died and</p> <p>13 it left a huge impact on their family.</p> <p>14 So not just for the people who had the</p> <p>15 illness but for the family and I think that</p> <p>16 continues to this day. I certainly know of</p> <p>17 families where they have lost a loved one and</p> <p>18 that still has a huge impact. So sorry then to</p> <p>19 return to your specific question, I mean, the</p> <p>20 whole gambit of mental illness, obviously</p> <p>21 depression, I think of cases of self-harm, right</p> <p>22 up to suicide, absolutely, and again I think for</p> <p>23 the whole family circle because people did feel</p> <p>24 a lack of support, perceived a lack of support,</p> <p>25 I think healthcare professionals sometimes we</p>
<p style="text-align: right;">79</p> <p>1 felt powerless and that may have transmitted to</p> <p>2 people feeling that we, even in extreme cases,</p> <p>3 didn't care but sometimes it was the lack of</p> <p>4 support and because people didn't neatly fit</p> <p>5 into a particular area where they could be</p> <p>6 referred for treatment or whatever, so you might</p> <p>7 have been depressed but then you were referred</p> <p>8 maybe to mental health services but it was sort</p> <p>9 of thought, well, this person has HIV or AIDS so</p> <p>10 that's obviously the cause of that and that was</p> <p>11 sort of almost compartmentalised.</p> <p>12 Again, an actual lack of support and</p> <p>13 a perceived lack of support meant that those</p> <p>14 mental health outcomes were perhaps worse.</p> <p>15 <b>MS EDWARDS:</b> I think one other thing as a community</p> <p>16 nurse, I think I have to acknowledge the fact</p> <p>17 that we were attached to big specialist centres</p> <p>18 often and where that did exist sometimes the</p> <p>19 care and support was there and we were able to</p> <p>20 help people to an extent, but outside those</p> <p>21 specialist centres and to the more rural</p> <p>22 regional -- I'm using my Australian terms now --</p> <p>23 the regional areas of the UK, sometimes the</p> <p>24 support and the medicine and the doctors were</p> <p>25 not there. They just were not. The ones that</p>	<p style="text-align: right;">80</p> <p>1 were there may not have had the education and</p> <p>2 the knowledge and so people were let down and so</p> <p>3 they had to survive in a family unit often alone</p> <p>4 with very little support.</p> <p>5 <b>DR JOHNSTON:</b> I think it is important to remember</p> <p>6 this was the pre-internet age which, of course,</p> <p>7 has revolutionised and people can keep in</p> <p>8 contact with self-help groups and other people</p> <p>9 who are suffering in a similar way. People</p> <p>10 really did feel very isolated at that time.</p> <p>11 <b>PROFESSOR ANDERSON:</b> I think there's also, this is</p> <p>12 still, there is some truth in what you are</p> <p>13 saying today. We know, as Ian's alluded to,</p> <p>14 that the burden of mental ill health is higher</p> <p>15 amongst people with HIV.</p> <p>16 Actually, many of the specialist centres,</p> <p>17 we ensure that we have psychologists on the</p> <p>18 team. We have very, very close working</p> <p>19 relationships with our mental health colleagues,</p> <p>20 but those may not be available outside those HIV</p> <p>21 specialist centres, even today. So we know the</p> <p>22 pressure on mental health services in general</p> <p>23 means that there is still unmet need and so even</p> <p>24 in an era of anti-retroviral therapy we are</p> <p>25 still seeing poor mental health and still seeing</p>

<p>81</p> <p>1 unmet need for its improvement.</p> <p>2 <b>PROFESSOR COOKE:</b> That was an issue that came up</p> <p>3 after yesterday as well in that, as you say, we</p> <p>4 still have limited access to mental health</p> <p>5 support for payments with HIV but actually it's</p> <p>6 probably better than for patients who have</p> <p>7 mono-infection with viral hepatitis where often</p> <p>8 there isn't even funding for psychological</p> <p>9 support services. Sometimes people can access</p> <p>10 that through HIV services where it's available</p> <p>11 but it's still a big issue in terms of accessing</p> <p>12 that.</p> <p>13 <b>MS SCOTT:</b> Professor Anderson, I know you were</p> <p>14 involved in a study in 2004 which looked at</p> <p>15 stigma in a population in North London. One of</p> <p>16 the findings from that report was that a lot of</p> <p>17 the participants were reporting that they had</p> <p>18 been discriminated against within the Health</p> <p>19 Service itself and they reported discriminatory</p> <p>20 behaviour as a result of their HIV status from</p> <p>21 GPs, from dentists, and from hospital workers</p> <p>22 outside HIV.</p> <p>23 I just wondered if that is something that</p> <p>24 patients have reported to the panel and what</p> <p>25 challenges and consequences arise for patients</p>	<p>82</p> <p>1 and for those trying to care for them.</p> <p>2 <b>PROFESSOR ANDERSON:</b> So the study you are alluding</p> <p>3 to, we carried out, a group of us carried out</p> <p>4 a questionnaire-based study for people who were</p> <p>5 living with HIV attending clinical services in</p> <p>6 north-east London particularly. It was asking</p> <p>7 a whole array of questions about what life was</p> <p>8 like and the challenges that people were facing</p> <p>9 but the particular question I think that you are</p> <p>10 alluding to, we asked a particular question,</p> <p>11 "Have you experienced discrimination consequent</p> <p>12 upon your HIV infection?"</p> <p>13 The thing we have to say is this was</p> <p>14 a questionnaire-based study, so people were</p> <p>15 ticking a box. We weren't interviewing people</p> <p>16 like Sian was describing yesterday, so it is</p> <p>17 a subjective response to a question with a tick</p> <p>18 box, "Have you felt that experience?" A very</p> <p>19 high proportion of people said that they had.</p> <p>20 Digging into that, you are absolutely right, it</p> <p>21 was an array of healthcare workers amongst</p> <p>22 others. I mean, it wasn't exclusively there, it</p> <p>23 was within a greater societal picture.</p> <p>24 I think the thing that comes from that was</p> <p>25 that the people who had been living with HIV for</p>
<p>83</p> <p>1 the longest experienced the highest risk of</p> <p>2 experiencing discrimination, often because they</p> <p>3 had been through more episodes of care or they</p> <p>4 had longer time living with HIV for that to have</p> <p>5 happened.</p> <p>6 The question was quite specifically asked,</p> <p>7 "Do you feel that this was a result of your HIV</p> <p>8 infection?" and people's answer to that was yes.</p> <p>9 I think there have been strides made. We did</p> <p>10 the study in 2004. We wrote it up in 2008 but,</p> <p>11 as Ian has alluded to, we have the positive</p> <p>12 voices survey, which, the most recent iteration</p> <p>13 was 2015/16 and still we're seeing, okay, the</p> <p>14 proportion has gone down slightly but we're</p> <p>15 still seeing people who are afraid to access</p> <p>16 healthcare because of the response that they may</p> <p>17 have. Ours was the first quantitative study to</p> <p>18 actually demonstrate this happening.</p> <p>19 Again, it has led to a whole array of</p> <p>20 attempts going, "What do we do? How do we move</p> <p>21 this forward", because it's unacceptable and</p> <p>22 every possible which way, and again going back</p> <p>23 to Ian's point about long-term conditions, more</p> <p>24 people with HIV are meeting more healthcare</p> <p>25 practitioners in more settings, so it's</p>	<p>84</p> <p>1 absolutely crucial that we address this.</p> <p>2 <b>PROFESSOR TUDOR-WILLIAMS:</b> I think also just</p> <p>3 remembering back the difficulties that we had</p> <p>4 persuading parents that their children's best</p> <p>5 interests were served by their GPs knowing.</p> <p>6 There was a great deal of resistance to the idea</p> <p>7 of general practitioners being informed about</p> <p>8 the HIV diagnosis.</p> <p>9 That was partly driven by experience that,</p> <p>10 I don't know whether David can attest to this,</p> <p>11 but we would get reports back from the families</p> <p>12 saying their notes and their children's notes</p> <p>13 would have a big red spot or something to</p> <p>14 identify them as HIV infected.</p> <p>15 It was vaguely covert in that it wasn't</p> <p>16 actually written HIV but it was -- everybody in</p> <p>17 the practice knew and they didn't want to</p> <p>18 experience that. They didn't want their</p> <p>19 children to experience that and the other</p> <p>20 element was in the school system as well, that,</p> <p>21 you know, there were sometimes advantages in the</p> <p>22 school understanding why the child was missing</p> <p>23 a lot of school because of hospital appointments</p> <p>24 and in-patient care episodes. But then, you</p> <p>25 know, there was an issue about the transfer of</p>

<p style="text-align: right;">85</p> <p>1 that knowledge from primary education into</p> <p>2 secondary education, without anybody thinking to</p> <p>3 ask the family and the child in particular</p> <p>4 whether they agreed with that. It was a really</p> <p>5 big issue for us.</p> <p>6 <b>DR JOHNSTON:</b> I think that's absolutely true and</p> <p>7 I think in primary care there would have been</p> <p>8 a concern that people were stigmatised at one</p> <p>9 end of the spectrum. I think at the other end</p> <p>10 of the spectrum people didn't want it recorded</p> <p>11 on their notes because it did have implications</p> <p>12 for life insurance and, you know, getting</p> <p>13 a mortgage and all sorts of things like that, in</p> <p>14 terms of school, you know, I've certainly</p> <p>15 experience of kids being asked not to take part</p> <p>16 in sport because, you know, there was some risk</p> <p>17 of something and just again the whole stigma</p> <p>18 that, you know, general practice, may be</p> <p>19 a smaller community and though, of course,</p> <p>20 confidentiality is at the heart of everything</p> <p>21 that we try to do, there is always just that</p> <p>22 concern that some information might become</p> <p>23 known. So absolutely I think that's a valid</p> <p>24 point.</p> <p>25 <b>MS EDWARDS:</b> At the same time as Jane was doing the</p>	<p style="text-align: right;">86</p> <p>1 quantitative data, we were collecting</p> <p>2 qualitative data from people with haemophilia</p> <p>3 and HIV, as I have mentioned the other day, and</p> <p>4 then secondly their families.</p> <p>5 I think what's just to add on to that,</p> <p>6 that I've recently done a same situation life</p> <p>7 history with healthcare workers and I just on</p> <p>8 this subject I talked to a number of community</p> <p>9 HIV specialists. Their job was to refer to GPs</p> <p>10 and services in the community outside of the</p> <p>11 hospitals that they had been treated so that</p> <p>12 they could go home and many of those nurses who</p> <p>13 were trying to make those referrals came to huge</p> <p>14 resistance from many services who didn't want to</p> <p>15 take on board the care of the patient with HIV</p> <p>16 infection and that, of course, also things like</p> <p>17 undertakers, you know, people who struggled with</p> <p>18 trying to get their partner being taken away who</p> <p>19 had died and the resistance of undertakers.</p> <p>20 It was widespread discrimination across</p> <p>21 the board and, like we keep saying, there were</p> <p>22 some very specialist centres where people even</p> <p>23 today, people walk into a clinic that I work in</p> <p>24 because it is a sexual health clinic and they</p> <p>25 say, "when I walk through this door I feel safe,</p>
<p style="text-align: right;">87</p> <p>1 but out there I never feel safe". I think</p> <p>2 that's today, so ...</p> <p>3 <b>MS SCOTT:</b> Professor Anderson, you touched on this,</p> <p>4 the consequence of that to those who are</p> <p>5 infected and affected not only is psychological</p> <p>6 and emotional but a reluctance to access</p> <p>7 healthcare services, so a direct impact on their</p> <p>8 physical health.</p> <p>9 <b>PROFESSOR ANDERSON:</b> Absolutely, no question. Even</p> <p>10 today I can think of a woman who said, "Oh, I've</p> <p>11 been called for a mammogram but I just don't</p> <p>12 want to go because they are going to start</p> <p>13 asking about my HIV. I think I won't go".</p> <p>14 <b>MS SCOTT:</b> Before we break for lunch can I just pick</p> <p>15 up a few questions about co-infection. We</p> <p>16 covered some of it yesterday, co-infection with</p> <p>17 hepatitis, covered some of it yesterday and</p> <p>18 I don't want to go back over that but that was</p> <p>19 primarily from the point of view of hepatitis</p> <p>20 but from what I've understood, co-infection with</p> <p>21 hepatitis B or C when one has HIV, doesn't alter</p> <p>22 the natural course of the -- doesn't alter the</p> <p>23 way the HIV develops. It's really, the</p> <p>24 alteration is all on the hepatitis.</p> <p>25 <b>PROFESSOR COOKE:</b> In general, yes, that's correct.</p>	<p style="text-align: right;">88</p> <p>1 <b>MS SCOTT:</b> What I would like to ask you is the</p> <p>2 difficulties of managing the two infections</p> <p>3 together, so whether it's hepatitis B and HIV or</p> <p>4 hepatitis C and HIV.</p> <p>5 Are there difficulties in taking</p> <p>6 medication for both infections at the same time?</p> <p>7 <b>PROFESSOR COOKE:</b> So certainly that is true and</p> <p>8 I think we touched on some of that yesterday.</p> <p>9 It's slightly different issues, depending on</p> <p>10 whether you are infected with HIV and hepatitis</p> <p>11 C or HIV and hepatitis B but we talked at length</p> <p>12 yesterday about interferon and the challenges of</p> <p>13 taking interferon. We know that for individuals</p> <p>14 infected with HIV, particularly those with</p> <p>15 advanced immunosuppression, who needed hepatitis</p> <p>16 C treatment, the treatment was more difficult,</p> <p>17 it was more prolonged and it was less likely to</p> <p>18 work, so all of those factors combined alongside</p> <p>19 the additional toxicity that you might have</p> <p>20 taking those medications with medications for</p> <p>21 HIV were huge challenges which were a barrier to</p> <p>22 both starting care and having successful</p> <p>23 treatment for hepatitis C.</p> <p>24 We know that without effective HIV</p> <p>25 treatment that hepatitis C progresses more</p>



<p style="text-align: right;">89</p> <p>1 quickly and the incidence of serious liver</p> <p>2 disease is higher. That has changed in recent</p> <p>3 years but it was certainly the case for a very</p> <p>4 long time.</p> <p>5 Similarly, for hepatitis B we know</p> <p>6 progression was more aggressive. Until</p> <p>7 relatively recently we didn't have specific</p> <p>8 treatments for hepatitis B that could be taken</p> <p>9 easily with HIV treatment. That's improved</p> <p>10 slightly with the advent of some medications</p> <p>11 which were active against both viruses but we</p> <p>12 still have a few patients who require different</p> <p>13 sets of medications for their HIV and</p> <p>14 hepatitis B.</p> <p>15 So certainly additional challenges and</p> <p>16 particularly around the additional toxicities of</p> <p>17 drugs that were being used.</p> <p>18 <b>MS SCOTT:</b> The choice for the patient is whether or</p> <p>19 not to take the medication for the hepatitis,</p> <p>20 rather than whether or not to take the</p> <p>21 medication for the HIV, if I can put it that</p> <p>22 way.</p> <p>23 <b>PROFESSOR COOKE:</b> Yes, that's changed over time and</p> <p>24 I think that that balance of, in particular, so</p> <p>25 obviously these days we have not really touched</p>	<p style="text-align: right;">90</p> <p>1 on this yet but we would be recommending</p> <p>2 everybody with HIV to be on treatment. That</p> <p>3 wasn't always the case but, by and large, by the</p> <p>4 late '80s early '90s most patients would be on</p> <p>5 some treatment which would be quite toxic. The</p> <p>6 decision of whether to take hepatitis C</p> <p>7 treatment would be a balance of how likely it</p> <p>8 was to be effective, balanced against the</p> <p>9 toxicity risks of taking it and, yes, so that</p> <p>10 would have been an issue, particularly before</p> <p>11 the advent of highly active anti-retroviral</p> <p>12 therapy.</p> <p>13 <b>MS SCOTT:</b> To reassure you I am going to be asking</p> <p>14 questions about treatment for HIV after lunch.</p> <p>15 Is there anything anyone wants to add to that</p> <p>16 discussion?</p> <p>17 <b>SIR BRIAN LANGSTAFF:</b> That brings us neatly I think</p> <p>18 to our lunch break. Two o'clock, please.</p> <p>19 <b>(1.00 pm)</b></p> <p>20 <b>(Luncheon Adjournment)</b></p> <p>21 <b>(2.00 am)</b></p> <p>22 <b>SIR BRIAN LANGSTAFF:</b> Yes, Ms Scott.</p> <p>23 <b>MS SCOTT:</b> As promised before lunch, I am going to</p> <p>24 turn to treatment but before I do, I just want</p> <p>25 to make a comment really by way of explanation</p>
<p style="text-align: right;">91</p> <p>1 both to the panel and to those that are</p> <p>2 listening, particularly for those that weren't</p> <p>3 here yesterday, and that's just to say that in</p> <p>4 your report you were asked to and have provided</p> <p>5 a short chronology of key events, and consistent</p> <p>6 with the approach that has been taken with other</p> <p>7 expert groups I am not going to ask you what was</p> <p>8 or should have been known by clinicians during</p> <p>9 the first half of the 1980s about risks of HIV</p> <p>10 or HTLV-3 or AIDS, or -- and I am not going ask</p> <p>11 you about medical and scientific work being done</p> <p>12 then in relation to the virus or to the</p> <p>13 development of a test for what we now know as</p> <p>14 HIV.</p> <p>15 The reason for that is because it is</p> <p>16 a question of fact for the Inquiry, which the</p> <p>17 Inquiry is investigating by reference to</p> <p>18 contemporaneous material, and so those</p> <p>19 questions -- any questions in relation to that</p> <p>20 will not form part of what happens for the rest</p> <p>21 of this afternoon.</p> <p>22 <b>SIR BRIAN LANGSTAFF:</b> And, I should add, by -- in</p> <p>23 relation to the evidence of those who can give</p> <p>24 their own contemporaneous testimony.</p> <p>25 <b>MS SCOTT:</b> Indeed.</p>	<p style="text-align: right;">92</p> <p>1 Moving, then, on to the treatment for HIV</p> <p>2 itself, we've touched on treatment for some of</p> <p>3 the AIDS-defining conditions, but moving on to</p> <p>4 the treatment for HIV itself, can somebody give</p> <p>5 us a brief outline of the first generation</p> <p>6 treatments, the mono-therapies.</p> <p>7 <b>DR WILLIAMS:</b> The first therapies basically targeted</p> <p>8 a particular enzyme in the virus replication</p> <p>9 cycle called reverse transcriptase. So reverse</p> <p>10 transcriptase was responsible for manufacturing</p> <p>11 a double-stranded DNA copy from</p> <p>12 a single-stranded RNA copy in the infected T</p> <p>13 cell. So it's an enzyme that was encoded by the</p> <p>14 virus to be able to do that. So what -- the</p> <p>15 initial first generation drugs, the nucleoside</p> <p>16 reverse transcriptase inhibitors targeted that</p> <p>17 drug -- targeted that enzyme by basically</p> <p>18 binding to the active site of that enzyme and</p> <p>19 inserting into the elongating DNA chain and</p> <p>20 terminating it. Right? So that's the way it</p> <p>21 worked.</p> <p>22 Now, the first drugs were a drug like AZT,</p> <p>23 or azidothymidine, now called zidovudine, which</p> <p>24 was not -- wasn't developed specifically for HIV</p> <p>25 but came off-the-shelf. It was actually</p>

<p style="text-align: right;">93</p> <p>1 a previous drug that had been tested for cancer</p> <p>2 but it did -- had been known to have an effect</p> <p>3 on -- on reverse -- on this -- what was -- being</p> <p>4 able to stop the reverse transcriptase enzyme.</p> <p>5 So that was originally made available in</p> <p>6 the UK in mid-1987 following the results of</p> <p>7 a trial in the United States which looked at</p> <p>8 giving patients who already had AIDS or severe</p> <p>9 disease zidovudine, and the trial was stopped</p> <p>10 after a few months because there was a much</p> <p>11 higher mortality in those who didn't -- who</p> <p>12 didn't receive the drug compared to those who</p> <p>13 did.</p> <p>14 So the death -- mortality fell from</p> <p>15 14 per cent down to 1 per cent. In fact, there</p> <p>16 were 19 deaths in the -- in the control arm and</p> <p>17 only one death in then -- based off that,</p> <p>18 zidovudine was licenced, right, as something</p> <p>19 that would be able to reduce or improve</p> <p>20 mortality.</p> <p>21 The problem with zidovudine mono-therapy</p> <p>22 is that at the time we didn't have the same</p> <p>23 knowledge of the virus as we do now, but it</p> <p>24 became apparent that the clinical benefit of</p> <p>25 that was short-lived. And it came at a high</p>	<p style="text-align: right;">94</p> <p>1 cost of toxicity in terms of side effects. The</p> <p>2 dose that was used in the early trials was twice</p> <p>3 the dose that is now licensed for it to be used,</p> <p>4 and so, yes, there was a desperate need to try</p> <p>5 and develop drugs and get treatments out there,</p> <p>6 and -- and this was made available, but it was</p> <p>7 by far -- you know, it was a drug that really</p> <p>8 was not fit for purpose in terms of being able</p> <p>9 to deliver what we wanted to achieve.</p> <p>10 There were other nucleoside reverse</p> <p>11 transcriptase inhibitors that became available</p> <p>12 in the early 1990s. That included zalcitabine,</p> <p>13 or DDC, or didanosine, DDI, and stavudine, D4T.</p> <p>14 They all worked in the same way, in that they</p> <p>15 basically inhibited this enzyme in the -- the</p> <p>16 virus needed to be able to infect a T cell and</p> <p>17 for it to replicate itself within the activated</p> <p>18 T cell. All these D drugs, as they were shown,</p> <p>19 equally had side effects that were quite marked</p> <p>20 and quite severe. So, for example, zalcitabine</p> <p>21 was a drug that had a high level of neuropathy,</p> <p>22 causes a toxic neuropathy in about 30 per cent</p> <p>23 of patients. Stavudine did the same. DDI was</p> <p>24 very difficult to take. It was, first of all,</p> <p>25 available as a powder, then became quite</p>
<p style="text-align: right;">95</p> <p>1 a chalky big tablet, before it became a capsule.</p> <p>2 So they were -- they were drugs that were</p> <p>3 developed in response to a crisis, in terms of</p> <p>4 trying to find something that would work, but</p> <p>5 they were drugs that were really quite -- had</p> <p>6 high level of side effects.</p> <p>7 MS SCOTT: Henry, can I ask you to turn to page 51</p> <p>8 of the report. It is probably 52 of the</p> <p>9 document. There's a table, table 4 there. Yes,</p> <p>10 so it's the page before that.</p> <p>11 The Inquiry has received a lot of evidence</p> <p>12 from people that took AZT in particular in the</p> <p>13 80s. You've created a table there that sets out</p> <p>14 some of the common side effects:</p> <p>15 "Blood disorders: anaemia, neutropaenia,</p> <p>16 nausea, vomiting, diarrhea, abdominal pain,</p> <p>17 headache, malaise, dizziness, myalgia, and</p> <p>18 lipotrophy."</p> <p>19 Does anyone -- can anyone else add</p> <p>20 anything about -- in particular in relation to</p> <p>21 AZT but all of those first generation</p> <p>22 medications that you have just spoken to, about</p> <p>23 the burden of the side effects and the</p> <p>24 difficulties that Dr Williams has raised about</p> <p>25 how difficult they were to actually take as</p>	<p style="text-align: right;">96</p> <p>1 a medication.</p> <p>2 PROFESSOR ANDERSON: Can I just add something there,</p> <p>3 and I think that my very strong memory of AZT --</p> <p>4 at the beginning -- I think you were right --</p> <p>5 the dosing was very high, it caused anaemia in</p> <p>6 a lot of people, and it got to the point</p> <p>7 actually where some people were blood</p> <p>8 transfusion dependent. So we used to have</p> <p>9 people who would take their AZT and then come in</p> <p>10 to have blood transfusions to deal with the</p> <p>11 anaemia that was the side effect of the drug.</p> <p>12 So given there wasn't anything else on the</p> <p>13 market, we were managing side effects, really</p> <p>14 serious, serious side effects, in order to try</p> <p>15 and continue to -- this drug. But we also had</p> <p>16 timers, I remember people used to get up in the</p> <p>17 middle of the night to take a dose, which was</p> <p>18 incredibly disruptive. So as well as feeling</p> <p>19 terrible, it really had a lot of implications</p> <p>20 for life, for how you lived your life.</p> <p>21 PROFESSOR COOKE: A lot of those side effects, as we</p> <p>22 have heard, were very common and very severe,</p> <p>23 and some of those very long-lasting. So, for</p> <p>24 those fortunate enough to survive, and still</p> <p>25 some of those patients in clinic now will still</p>

<p style="text-align: right;">97</p> <p>1 have some of those side effects as a result. So</p> <p>2 peripheral neuropathy for example. And there's</p> <p>3 a condition listed here which is a bit like</p> <p>4 cirrhosis which we see in a small number of</p> <p>5 patient who had didanosine, that is still an</p> <p>6 issue today, so many of these things ...</p> <p>7 PROFESSOR TUDOR-WILLIAMS: But can I just put the</p> <p>8 counter-argument, that the -- trials of the use</p> <p>9 of zidovudine, AZT, to prevent or decrease the</p> <p>10 risk of transmission from mothers to their</p> <p>11 babies was extraordinarily successful. I mean,</p> <p>12 really ground-breaking work, and it is still</p> <p>13 used today. I mean, we still use zidovudine to</p> <p>14 protect newborn infants to reduce the risk of</p> <p>15 transmission of HIV. So it's not -- whilst you</p> <p>16 read these lists of toxicities -- and indeed in</p> <p>17 the beginning I remember we were -- that very</p> <p>18 first child I looked after in 1988 we put onto</p> <p>19 continuous infusion of zidovudine, round the</p> <p>20 clock, 24 hours a day, because we knew so little</p> <p>21 about how the drug was distributed. But</p> <p>22 I wouldn't want to leave everybody with the</p> <p>23 impression that this is a -- such a toxic drug</p> <p>24 that it has no place in modern medicine, because</p> <p>25 it is still being used today.</p>	<p style="text-align: right;">98</p> <p>1 PROFESSOR ANDERSON: The caveat to that, of course,</p> <p>2 is we have learnt, and -- and so the doses that</p> <p>3 we are using are much lower, and we are able</p> <p>4 I think, now, to manage that drug in a useful</p> <p>5 way, whereas at the time Ian's described I think</p> <p>6 we hadn't got that knowledge of how to best use</p> <p>7 the benefits it could bring, and so we saw quite</p> <p>8 a lot of toxicity.</p> <p>9 PROFESSOR COOKE: But over -- saying, further, that</p> <p>10 there may be people -- and we still have</p> <p>11 patients who are still on it --</p> <p>12 PROFESSOR ANDERSON: Absolutely.</p> <p>13 PROFESSOR COOKE: -- and there is a minority of</p> <p>14 patients who don't get the side effects, and if</p> <p>15 they have been on the treatment for a very long</p> <p>16 time, it may still be an appropriate treatment</p> <p>17 to be on.</p> <p>18 DR WILLIAMS: And I think it's important -- this is</p> <p>19 desperate times in terms of people trying to</p> <p>20 find something that would stop the progression</p> <p>21 to AIDS and to -- for people dying, and so</p> <p>22 people would take medications in the hope that</p> <p>23 that would buy them some time in the way that</p> <p>24 people might take cancer chemotherapy to buy</p> <p>25 them some time.</p>
<p style="text-align: right;">99</p> <p>1 So -- but -- yeah, I mean, undoubtedly</p> <p>2 particularly -- you know, studied DDI and</p> <p>3 zalcitabine were all associated with quite</p> <p>4 severe major side effects. They are no longer</p> <p>5 particularly used these days. They were -- in</p> <p>6 fact, zalcitabine has been withdrawn, although</p> <p>7 zidovudine still has a role to play for some</p> <p>8 patients.</p> <p>9 PROFESSOR TUDOR-WILLIAMS: Yeah, and that's very</p> <p>10 short-term treatment, for the infants.</p> <p>11 MS SCOTT: Can I ask you in particular about</p> <p>12 lipoatrophy, subcutaneous fat loss, because that</p> <p>13 is a stigmatising side effect.</p> <p>14 DR WILLIAMS: It is. That is still something a lot</p> <p>15 of people who -- who were treated with these</p> <p>16 first generation therapies still have a problem</p> <p>17 with, and I still see lots of patients who have</p> <p>18 had -- developed lipoatrophy and who continue to</p> <p>19 need to have dermatological fillers to help</p> <p>20 alleviate that. The reason why lipoatrophy</p> <p>21 occurred is because, like I said, normally these</p> <p>22 drugs had an effect on the mitochondrial cells.</p> <p>23 The mitochondria are the -- are the energy parts</p> <p>24 of the cell, and they would affect how these</p> <p>25 cells functioned and it is in fat cells -- it</p>	<p style="text-align: right;">100</p> <p>1 made them lose fat cells, and so they developed</p> <p>2 this lipoatrophy which was, as you rightly say,</p> <p>3 quite stigmatising in terms of appearances.</p> <p>4 PROFESSOR COOKE: Again, we still have specialist</p> <p>5 services that are operating now providing what</p> <p>6 we call "New-Fill", to try to fill in that</p> <p>7 lipoatrophy for patients. It is still a very</p> <p>8 common issue and one that patients find very</p> <p>9 difficult I think.</p> <p>10 MS EDWARDS: Is it worth just mentioning the</p> <p>11 actual -- what happens. This is a very --</p> <p>12 what's the expression -- where sunken, sunken</p> <p>13 cheeks -- and it's actually the other drugs with</p> <p>14 the buffalo hump, isn't it? Yeah.</p> <p>15 So -- and unfortunately, what that looked</p> <p>16 like was like having AIDS, and so for lots of</p> <p>17 people that was very difficult because they'd</p> <p>18 got through the point where they didn't look</p> <p>19 like the pictures of people with having -- who</p> <p>20 had AIDS, and then when you go on these drugs</p> <p>21 you did, despite the fact you were actually</p> <p>22 maybe being treated and potentially getting a</p> <p>23 little bit better. So -- thus the stigma.</p> <p>24 MS SCOTT: Then came dual therapy. Again, can you</p> <p>25 give us an outline of how that came about.</p>



<p style="text-align: right;">101</p> <p>1 DR WILLIAMS: Well, again, the virology of HIV, how  2 it developed -- along the same lines we don't  3 know everything about the virus, but it became  4 apparent that if -- virus isolated from people  5 being on zidovudine for six months was more  6 resistant to zidovudine than virus taken at  7 baseline. So it became apparent that the virus  8 was changing. It was mutating to become  9 resistant. And we know now that's because of,  10 as I said earlier, the virus replicates at  11 a high level, it does not proofread itself, it  12 introduced errors, and so it can -- and so any  13 mutations that are introduced which are --  14 are -- mean that it is less susceptible to that  15 drug will emerge. So dual therapy came along  16 when we had access to didanosine and  17 zalcitabine, and people then looked to see  18 whether maybe taking two drugs as combination  19 therapy, and there was a large trial which  20 showed that there was, again, impact in terms of  21 30 to 40 per cent improvement in mortality  22 associated with taking dual therapy, but people  23 still -- as I said in the report, people still  24 continued to die. It wasn't -- you know, it  25 wasn't a -- it was an improvement but there</p>	<p style="text-align: right;">102</p> <p>1 were -- people still progressed and people still  2 failed therapy. We know now it's because the --  3 yes, it improved -- it stopped -- at the end,  4 the viral load went down further but it wasn't  5 sufficient to stop the virus from mutating,  6 developing resistance, and then for the viral  7 load coming back up again, and then ongoing  8 damage to the immune system recurring and people  9 fell ill.</p> <p>10 So these effects were time-limited in  11 terms of it's rather like you say cancer  12 chemotherapy might work out then you have  13 a relapse. It's the same sort of thing.</p> <p>14 MS SCOTT: In terms of the burden of the medication,  15 once patients are taking dual therapy can you  16 speak to that.</p> <p>17 DR WILLIAMS: Zidovudine used to be taken every four  18 hours as Jane said that was the first sort of  19 dosing schedule. But now for -- largely these  20 were taken twice a day but some of the earlier  21 formulations I sort of said before were very  22 difficult. DDI in particular was very difficult  23 drug to take because of its formulation. It  24 improved over time but when the other drugs came  25 along, then the pill burden increased. This is</p>
<p style="text-align: right;">103</p> <p>1 on a background of people who also had to take  2 drugs to prevent them from getting the  3 opportunistic infections that they had already  4 experienced, so they were often on multiple, you  5 know, doses of different drugs that made it  6 impacted on their quality of life.</p> <p>7 MS SCOTT: They are taking two toxic drugs now with  8 side effects. Does that double the chances of  9 having side effects?</p> <p>10 DR WILLIAMS: I think it is important to understand  11 these drugs were made available based on  12 a randomised clinical trial that showed clinical  13 outcome was better. So I think it's important  14 to say that. Yes, they had side effects that we  15 were no longer -- you know in today's regimens  16 we would no longer tolerate but at the time  17 large randomised control trials showed that  18 taking two drugs was better than one in terms of  19 improved mortality and people wanted to -- these  20 things are taken one step at a time and you need  21 evidence from randomised control trials to be  22 able to say, well, that is a better standard of  23 care. This is how you then move on.</p> <p>24 MS SCOTT: In terms of the patient experience of  25 taking those two drugs, was two worse than one</p>	<p style="text-align: right;">104</p> <p>1 commonly?</p> <p>2 DR WILLIAMS: They clearly may have side effects one  3 from the other, absolutely right. So as Graham  4 has indicated some would have no side effects  5 but side effects were very common. So diarrhoea  6 from DDI, nausea and vomiting from zidovudine.  7 So definitely it impacted on them, definitely.</p> <p>8 MS SCOTT: Then we move on to triple therapy. Can  9 you tell us about that.</p> <p>10 DR WILLIAMS: Triple therapy came along when other  11 classes of anti-retroviral drugs became  12 available. So nevirapine was the first  13 non-nucleoside reverse transcriptase inhibitor  14 so it basically impacted on reverse  15 transcriptase and then the integrase  16 inhibitors -- sorry, not the integrase the  17 protease inhibitors, particularly adenovir and  18 adenovir came along in '96/97. These were drugs  19 that acted in a different way to the NRTIs and  20 trials of three drugs versus two drugs again  21 showed an incremental improvement in terms of  22 clinical benefit, and so trials of adenovir plus  23 two nucleoside analogues were shown to be better  24 than just two nucleoside analogues on their own.  25 At that point in time also trials had</p>

<p>105</p> <p>1 a clinical end point. They are looking to 2 determining mortality rates and on progression 3 to AIDS but then as we got to -- as the virology 4 improved and we were able to measure the virus 5 directly with new technologies, looking at virus 6 detection assays, we could then monitor the 7 effects of these drugs and the amount of virus 8 in blood and it became -- from those large 9 clinical end point studies it became apparent 10 that if you are able to press the virus and keep 11 it down and keep it suppressed then that 12 resulted in an improvement in the immune system 13 and so the changes in the surrogate markers, the 14 CD4 count in the viral load captured the 15 clinical benefits.</p> <p>16 So it's rather like, for example, if you 17 are treating high blood pressure, the outcome of 18 high blood pressure is stroke but to do a 19 clinical trial you do a clinical trial to look 20 at its effect on blood pressure. So it wasn't 21 until 1998, until the 1990s, that we moved from 22 doing large clinical end point studies to doing 23 trials which looked out viral load as an end 24 point and, at that point in time, with triple 25 combination therapy, I remember going to the</p>	<p>106</p> <p>1 Vancouver conference in 1996 and it was 2 a fantastic experience and to be able to see 3 these improvements with the triple combination 4 therapy. It made a huge impact in being able to 5 suppress the virus and the virus did not emerge 6 with resistance. It maintained viral 7 suppression.</p> <p>8 MS SCOTT: In terms again of the patient experience 9 of taking these medications, if we go back to 10 the table that we were just looking at, there 11 are an enormous amount of medications on this 12 table and I don't want to go through all of them 13 but could you just pick out for us where some 14 particular medications or where a medication 15 came on the market that seemed to be better 16 tolerated by patients than the previous ones.</p> <p>17 DR WILLIAMS: I suppose that didn't really come 18 until the mid-2000s. Adenovir was a protease 19 inhibitor but it was very difficult to take you 20 had to take it three times a day, you had to 21 have a high fluid intake. There were dietary 22 restrictions and a high incidence of renal 23 stones with it.</p> <p>24 Ritonavir was originally a drug that was 25 used for treatment of HIV infection but it was</p>
<p>107</p> <p>1 a really difficult drug to take in terms of its 2 now only used as a booster for that.</p> <p>3 Nevirapine was associated with quite 4 severe rashes and people sometimes were admitted 5 to hospital because of a severe rash associated 6 with Nevirapine, although it is still a drug 7 that we use quite a lot in people who can 8 tolerate it.</p> <p>9 As we move sort of through, so between 10 1998 through to the mid-2000s, other newer drugs 11 emerged and new nucleoside analogues emerged in 12 Lamivudine and in terms of tenofovir which 13 didn't have this sort of side effect profile of 14 the D drugs that I mentioned before and newer 15 protease inhibitors became available such as 16 darunavir and atazanavir which were better 17 tolerated.</p> <p>18 Protease inhibitors generally, 19 particularly drugs like Kaletra contain 20 lopinavir and ritonavir had really -- people had 21 huge problems with gastrointestinal side effects 22 like diarrhoea, a chronic problem and drugs like 23 saquinavir also high dose burden, but also a lot 24 of problems with diarrhea. Lopinavir, another 25 one, these are early generation protease</p>	<p>108</p> <p>1 inhibitors. They advanced our knowledge. They 2 helped us move forward, but they were by far 3 ideal in terms of their pill burden, in terms of 4 their side effect profile.</p> <p>5 So it helped people but they had to deal 6 with -- a lot of people had to deal with side 7 effects. It wasn't really until I would say the 8 mid-2000s and a bit later when the integrase 9 inhibitors became available that we truly got 10 drugs that were much better tolerated.</p> <p>11 PROFESSOR ANDERSON: Just to add to what did Ian 12 said, particularly about the early protease 13 inhibitors, it was very hard to get a high 14 enough level in the blood to get a therapeutic 15 level, so you either had to take a lot of pills 16 a lot of times a day or you had to take 17 something which would help you boost it, which 18 was where ritonavir came in. I have a very 19 strong memory days of saquinavir when grapefruit 20 juice was found to be something that could boost 21 so people would have -- I can't remember -- 22 handfuls of pills three times a day out and be 23 advised to have grapefruit juice all in order to 24 keep the -- just to get the drug level into the 25 therapeutic range. So there was quite a lot</p>

<p style="text-align: right;">109</p> <p>1 of -- and even if it wasn't necessarily causing</p> <p>2 a side effect it was certainly causing</p> <p>3 a lifestyle effect and then gastrointestinal</p> <p>4 symptoms which we are talking about as well.</p> <p>5 There were difficulties in actually</p> <p>6 managing, in the early days of DDI you described</p> <p>7 those chalky pills. Before that it came as</p> <p>8 a sort of sachet of powder and you dispensed</p> <p>9 sort of a great shoe box of stuff and then we</p> <p>10 tried to make it flavoursome. Do you remember</p> <p>11 there was cherry flavour you could add and they</p> <p>12 were revolting. But again, as Ian has</p> <p>13 explained, they were making some sort of</p> <p>14 difference and so people put up with -- and we</p> <p>15 put up with prescribing things that were really</p> <p>16 difficult to take, not just making you feel</p> <p>17 unwell as well.</p> <p>18 DR WILLIAMS: I still have patients I look after who</p> <p>19 accessed triple therapy for the first time in</p> <p>20 1997 and it saved their lives. There's no doubt</p> <p>21 about that. Let's be very clear about that, the</p> <p>22 advent of triple therapy, high anti-retroviral</p> <p>23 therapy despite the problems of pill burden and</p> <p>24 side effects made a huge difference to some</p> <p>25 people.</p>	<p style="text-align: right;">110</p> <p>1 PROFESSOR ANDERSON: Yes, absolutely echo that and</p> <p>2 I think we learnt -- we were seeing, after that,</p> <p>3 the Vancouver conference and the introduction of</p> <p>4 protease inhibitors, a really dramatic shift and</p> <p>5 actually the cost in lifestyle and all the other</p> <p>6 things, there was a benefit, a really clear</p> <p>7 benefit.</p> <p>8 PROFESSOR TUDOR-WILLIAMS: I'd just like to say one</p> <p>9 thing. If you scroll right back to the top,</p> <p>10 there's a little line there that says for the</p> <p>11 interests of space we haven't included</p> <p>12 enfuvirtide but actually it's worth remembering</p> <p>13 that back in the '90s there was that interlude</p> <p>14 when we really had run out of new agents and T20</p> <p>15 or enfuvirtide, which is right at the top of the</p> <p>16 table and not in the table, there, that line</p> <p>17 there, enfuvirtide, my memory was this was</p> <p>18 a twice daily injection that was subcutaneous</p> <p>19 and very difficult to reconstitute and actually</p> <p>20 caused a lot of injection sites reactions. But</p> <p>21 we were using it and our patients were --</p> <p>22 I mean, again it bought time. It actually did</p> <p>23 tide people over long enough to survive to take</p> <p>24 advantage of triple combination therapy. But it</p> <p>25 was a really difficult drug to tolerate.</p>
<p style="text-align: right;">111</p> <p>1 MS EDWARDS: Can I just make a couple of things and</p> <p>2 maybe again Gareth will pick up on this. It was</p> <p>3 a bit of a different game when you were talking</p> <p>4 about children and many of these drugs were not</p> <p>5 available for children, although some were, and</p> <p>6 you can expand on this. But I think from the</p> <p>7 nursing point of view, how do you get these</p> <p>8 young children to take these drugs? We've</p> <p>9 already described how hideous they were to take</p> <p>10 as an adult when you can rationalise what you're</p> <p>11 doing. But to try and give a child something so</p> <p>12 revolting was very, very challenging.</p> <p>13 I think the second thing to say is the</p> <p>14 confidentiality issue that is associated with</p> <p>15 drugs because people had fridges full of drugs.</p> <p>16 They had bags full of drugs. I still know</p> <p>17 people today who make sure they take their drugs</p> <p>18 before they go to work and they are on one or</p> <p>19 two tablets a day. People were taking about 28</p> <p>20 drugs. It was enormous amount of drugs,</p> <p>21 including some of the prophylaxis for</p> <p>22 opportunistic infections which reduced. But</p> <p>23 maybe you want to expand on the children because</p> <p>24 it was a different scenario for the families who</p> <p>25 had small children.</p>	<p style="text-align: right;">112</p> <p>1 PROFESSOR TUDOR-WILLIAMS: Yes, okay. I don't want</p> <p>2 to spend too long but it was a challenge. The</p> <p>3 pharmaceutical industry, in fairness, was trying</p> <p>4 to work very hard with us to produce</p> <p>5 formulations that children could take but they</p> <p>6 were often -- I mean, ritonavir as a suspension</p> <p>7 is utterly repulsive and I wouldn't allow any</p> <p>8 doctor in our team, any new doctor joining,</p> <p>9 junior doctor, to prescribe ritonavir if they</p> <p>10 hadn't tasted it first so that they knew what</p> <p>11 they were actually having to -- what we were</p> <p>12 asking parents to do with their children.</p> <p>13 We ended up quite often inserting</p> <p>14 gastrostomy tubes, a tube that will sit in the</p> <p>15 stomach wall, through which you can give the</p> <p>16 medicines simply because you can then avoid all</p> <p>17 the taste but pathways that were so difficult</p> <p>18 for parents to manage. It was a challenge.</p> <p>19 MS SCOTT: I think you say in the report,</p> <p>20 Professor Tudor-Williams, that the drugs that</p> <p>21 did come on for children, the trials that did</p> <p>22 take place for children, were far better than</p> <p>23 for adults.</p> <p>24 PROFESSOR TUDOR-WILLIAMS: We tended to lag behind.</p> <p>25 We tended to have less choices and therefore</p>



<p style="text-align: right;">113</p> <p>1 children were kept on their first line therapy</p> <p>2 for longer and these were not easy therapies, so</p> <p>3 we ended up with, in those early days,</p> <p>4 unfortunately selecting out highly resistant</p> <p>5 virus in the population of the early-treated</p> <p>6 young people.</p> <p>7 MS EDWARDS: Just to add one quick thing, which is</p> <p>8 of course some of these side effects also had</p> <p>9 issues around bleeding so if you had haemophilia</p> <p>10 you weren't able to take some of these drugs</p> <p>11 because of the problems with bleeding. I think</p> <p>12 we've written that somewhere.</p> <p>13 MS SCOTT: Can you then take us up to the present</p> <p>14 day in terms of the history of the medications.</p> <p>15 DR WILLIAMS: Yes, triple therapy definitely made a</p> <p>16 huge impact and certainly from '97/98 the</p> <p>17 incidence of new opportunistic infections --</p> <p>18 this is when AIDS started to fall. We saw</p> <p>19 dramatic impact on both that and mortality. But</p> <p>20 the drugs were not ideal, so during the 2000s</p> <p>21 the integrase inhibitors became available,</p> <p>22 raltegravir in 2007 and the other integrase</p> <p>23 inhibitors later on and safer drugs in terms of</p> <p>24 nucleoside analogues like tenofovir also became</p> <p>25 available.</p>	<p style="text-align: right;">114</p> <p>1 The pill burden started to fall and people</p> <p>2 started to be able to take regimens that were</p> <p>3 once a day and now regimens that are in a single</p> <p>4 pill. They became more tolerable.</p> <p>5 A pivotal study was the smart study done</p> <p>6 in the mid-2000s which looked -- because we --</p> <p>7 to sort of consider what the thinking was at</p> <p>8 that time there was a sense that some of these</p> <p>9 drugs had sufficient high adverse events that</p> <p>10 maybe taking them periodically would be better</p> <p>11 than taking them all the time and, therefore,</p> <p>12 there was a trial that was done, randomised</p> <p>13 people to take medications periodically</p> <p>14 depending on their level of CD4 count in their</p> <p>15 blood versus taking therapy all the time. But</p> <p>16 that was -- the result of that trial was pivotal</p> <p>17 because it showed the control of virus all the</p> <p>18 time was far better. It had a far better</p> <p>19 improvement in clinical outcome.</p> <p>20 So that changed the way we looked at</p> <p>21 things and then as medications become more</p> <p>22 tolerable, not to say they didn't have side</p> <p>23 effects but they became more tolerable in terms</p> <p>24 of people being able to take them better, then</p> <p>25 it became clear that we needed to start therapy</p>
<p style="text-align: right;">115</p> <p>1 in everybody.</p> <p>2 It wasn't until 2015 where the outcome of</p> <p>3 a trial looking at early versus deferred</p> <p>4 anti-retroviral therapy where we showed that</p> <p>5 actually even if you take therapy as soon as you</p> <p>6 are diagnosed with a high CD4 count you have</p> <p>7 clinical benefit in terms of risk of progression</p> <p>8 to severe disease. It went down -- it sort of</p> <p>9 halved but it was only in terms of 4 per cent</p> <p>10 down to 1.6 per cent but told us that treatment</p> <p>11 at any level of CD4 count was important.</p> <p>12 From 2015 guidelines changed which then</p> <p>13 indicated that people should be offered therapy</p> <p>14 at the point of diagnosis and Public Health</p> <p>15 England now monitor the outcome of therapy and</p> <p>16 we know that from recent data that there are</p> <p>17 those patients who are diagnosed and attending</p> <p>18 clinical care, 97 per cent of them on therapy,</p> <p>19 and of those who are on therapy 97 per cent have</p> <p>20 undetectable viral loads in terms of viral</p> <p>21 suppression.</p> <p>22 Having said all that, that is a biological</p> <p>23 outcome in terms of viral suppression and that</p> <p>24 doesn't then -- one needs then to acknowledge</p> <p>25 what are the quality of life issues around</p>	<p style="text-align: right;">116</p> <p>1 taking therapy all the time and the other things</p> <p>2 that we talked about co-morbidities and</p> <p>3 everything else and mental health disease.</p> <p>4 MS SCOTT: Just sticking then on the burden and the</p> <p>5 side effects of the modern treatment where are</p> <p>6 we in relation to that? How is that</p> <p>7 experienced?</p> <p>8 DR WILLIAMS: Certainly someone starting</p> <p>9 anti-retroviral therapy today will have a much</p> <p>10 different experience than someone starting</p> <p>11 therapy 20 years ago. There's no doubt about</p> <p>12 that. Most people starting therapy for the</p> <p>13 first time today will take a regimen that mainly</p> <p>14 contained one or two pills and there will be</p> <p>15 drugs that are better tolerated. Their</p> <p>16 discontinuation rate due to side effects will</p> <p>17 be only a few per cent at one year and they will</p> <p>18 tend to continue to live normal lives and go to</p> <p>19 work. Yes, people experience side effects, as</p> <p>20 people experience side effects from any</p> <p>21 medication prescribed for any condition, but</p> <p>22 generally it is much better, right.</p> <p>23 Having said that, those people who have</p> <p>24 been treated before they will often be on much</p> <p>25 more complex regimens because of issues around</p>

<p style="text-align: right;">117</p> <p>1 resistance. They may have developed resistance</p> <p>2 to the previous drugs and therefore their</p> <p>3 regimens may be more complex. They may be twice</p> <p>4 a day, maybe more medications within them and</p> <p>5 they may therefore have more sides effects</p> <p>6 associated with that. So it's very different</p> <p>7 from someone starting therapy for the first</p> <p>8 time, diagnosed today, compared to someone who</p> <p>9 has been on therapy to 20 years where they have</p> <p>10 the burden of problems from over 20 years and</p> <p>11 what may be needed from therapy in them compared</p> <p>12 to someone who is naive to therapy.</p> <p>13 MS SCOTT: You have said that the 97 per cent of</p> <p>14 those who take anti-retroviral therapy have</p> <p>15 reduced their viral load to undetectable</p> <p>16 amounts. Can you explain what that actually</p> <p>17 means.</p> <p>18 DR WILLIAMS: Again, that's a very biological</p> <p>19 outcome to a certain extent and I think it's</p> <p>20 important not to -- one needs to emphasise the</p> <p>21 other impacts of taking therapy for a long time</p> <p>22 are, and we've alluded to that earlier.</p> <p>23 What that means is that if you suppress</p> <p>24 the virus, the virus is therefore unable to</p> <p>25 change and mutate, right, and therefore you</p>	<p style="text-align: right;">118</p> <p>1 don't develop resistance. Resistance is not</p> <p>2 developed and therefore people -- there's no --</p> <p>3 there's absolutely no reason that as long as you</p> <p>4 continue to suppress the virus through</p> <p>5 anti-retroviral therapy that people will not</p> <p>6 fail therapy, and the main reason why that will</p> <p>7 occur, as long as people are able to adhere to</p> <p>8 therapy, so one of the things that is very</p> <p>9 important about treatment is that people need to</p> <p>10 take -- need to adhere to therapy at a high</p> <p>11 level so more than 90 per cent of medications,</p> <p>12 so that in itself is also something, you know,</p> <p>13 important to emphasise and to do.</p> <p>14 But what the viral suppression allows, it</p> <p>15 allows the immune system to improve, right.</p> <p>16 I alluded to earlier rather like being on</p> <p>17 a treadmill. If you can come off the treadmill</p> <p>18 you recover and that's exactly what happens. It</p> <p>19 allows the immune system to recover, it allows</p> <p>20 it to rebuild itself and it allows the immune</p> <p>21 system to do what it should do, which is to</p> <p>22 prevent other infections occurring.</p> <p>23 Clearly starting therapy at a higher</p> <p>24 level, at a higher CD4 count, is better than</p> <p>25 starting therapy at a lower CD4 count because</p>
<p style="text-align: right;">119</p> <p>1 you are having to rebuild the immune system from</p> <p>2 a very low level, not that it can't be done but</p> <p>3 it's associated with more morbidity.</p> <p>4 MS SCOTT: Once a viral load is undetectable, is it</p> <p>5 right that it's been known for some time that</p> <p>6 that means that it's not untransmittable?</p> <p>7 DR WILLIAMS: Yes. I mean, the viral undetectable</p> <p>8 is basically based on an assay we use to monitor</p> <p>9 viral load in blood. It's not to say the virus</p> <p>10 has gone away. The virus is still there in</p> <p>11 cells that are quiescent and latent and if you</p> <p>12 were to stop therapy, the viral load comes back</p> <p>13 up again.</p> <p>14 MS SCOTT: So it has not been cured.</p> <p>15 DR WILLIAMS: Not been cured. It's about</p> <p>16 maintaining viral suppression through drugs that</p> <p>17 inhibit viral replication. It's not about cure</p> <p>18 or eradication. That is very much the goal that</p> <p>19 scientists are trying to look at but at the</p> <p>20 moment in time therapy is lifelong and people</p> <p>21 have to take lifelong therapy every day. So</p> <p>22 that's again that has an impact in terms of how</p> <p>23 people see also themselves as a chronic</p> <p>24 condition.</p> <p>25 PROFESSOR ANDERSON: I think it is also important to</p>	<p style="text-align: right;">120</p> <p>1 just reinforce Ian's point about undetectable.</p> <p>2 So the terminology has come from the laboratory</p> <p>3 assays and the level that we call undetectable</p> <p>4 has changed as the assays have got more</p> <p>5 sophisticated, so it is about looking in the</p> <p>6 blood to see how much viral replication is going</p> <p>7 on. In the early tests, you could find 400</p> <p>8 copies per ml and that would be the level of</p> <p>9 detection. Then it went down to 100 with a more</p> <p>10 sensitive assay, then it went down to 50, then</p> <p>11 it went down to 20, so it's about -- it's</p> <p>12 referring to what the laboratory tests can do</p> <p>13 and the sensitivity of it. So it is</p> <p>14 undetectable on the test and, again, it's really</p> <p>15 important it's not gone away, it's just that the</p> <p>16 test has got to the limit of its sensitivity.</p> <p>17 DR WILLIAMS: Also you alluded to the question about</p> <p>18 being untransmittable. Certainly, if you reduce</p> <p>19 the virus in blood, you reduce the genital</p> <p>20 fluids, therefore, we now know again from</p> <p>21 randomised control trials that if you go on to</p> <p>22 therapy you reduce the risk of transmission to</p> <p>23 an uninfected partner by 95/96 per cent.</p> <p>24 MS SCOTT: Through having sexual relations?</p> <p>25 DR WILLIAMS: That's right.</p>

<p style="text-align: right;">121</p> <p>1 PROFESSOR ANDERSON: Again, from vertical</p> <p>2 transmission, the greatest risk factor for</p> <p>3 vertical transmission is how viral load in the</p> <p>4 mother and a woman who has an undetectable viral</p> <p>5 load, the risk of transmission is -- well,</p> <p>6 Gareth?</p> <p>7 PROFESSOR TUDOR-WILLIAMS: Less than 1 in 1,000.</p> <p>8 MS SCOTT: How about transmission via blood?</p> <p>9 MS EDWARDS: Wherever, it's all about viral load.</p> <p>10 DR WILLIAMS: I would have to say there isn't the</p> <p>11 same level of evidence, right, in terms of, for</p> <p>12 example, in injecting drug users and clearly</p> <p>13 blood products are now screened for that, so</p> <p>14 there isn't that evidence. So the evidence</p> <p>15 comes from sexual transmission because that's</p> <p>16 where ongoing transmission occurred.</p> <p>17 PROFESSOR COOKE: It's difficult to really</p> <p>18 underestimate the transformative impact triple</p> <p>19 therapy has had, not just to individuals but for</p> <p>20 the management of the whole disease. It</p> <p>21 transformed what was essentially a disease of</p> <p>22 progressed immunosuppression which was managed</p> <p>23 in hospitals, in my own setting, where</p> <p>24 I imagine, James, at the time, there may have</p> <p>25 been 40 or 50 patients in that hospital and we</p>	<p style="text-align: right;">122</p> <p>1 now have maybe three or four.</p> <p>2 What has happened is we have moved HIV to</p> <p>3 a much more of a chronic disease, like we might</p> <p>4 manage diabetes or hypertension, which is in</p> <p>5 outpatient clinics and we now have maybe 100,000</p> <p>6 patients in the country on this treatment, and</p> <p>7 it was really in the middle of the 1990s that</p> <p>8 that transformative change came about.</p> <p>9 PROFESSOR ANDERSON: Absolutely. I think the other</p> <p>10 thing which Sian has alluded to is the need to</p> <p>11 continue therapy. Something I hear from</p> <p>12 patients is the reminder that every time you</p> <p>13 take a pill you're still living with HIV and</p> <p>14 sometimes that can be a barrier to adherence</p> <p>15 because it's a constant reminder. There are</p> <p>16 some drugs at the moment only in clinical trials</p> <p>17 which are looking at either longer term</p> <p>18 injectables or depots which may again change the</p> <p>19 field, but for the moment somebody will need to</p> <p>20 take tablets every day.</p> <p>21 MS SCOTT: You told us that the anti-retroviral</p> <p>22 therapy, for 97 per cent of those that take it,</p> <p>23 is highly effective. Is it more effective for</p> <p>24 somebody that is newly infected and goes on to</p> <p>25 those therapies, those modern therapies, rather</p>
<p style="text-align: right;">123</p> <p>1 than someone that has had HIV and been living</p> <p>2 with it for a long time?</p> <p>3 DR WILLIAMS: The thing that determines response is</p> <p>4 the ability to take the medication. It depends</p> <p>5 on your previous exposure and development of</p> <p>6 resistance and on other co-morbidities that</p> <p>7 might impact on side effects. So, yes, someone</p> <p>8 who is diagnosed now at an earlier age is likely</p> <p>9 to have a much better experience than someone</p> <p>10 diagnosed -- well, they will have a much better</p> <p>11 experience than someone diagnosed 20 years ago.</p> <p>12 PROFESSOR COOKE: We have now really touched on life</p> <p>13 expectancy, so we would expect that if someone</p> <p>14 was diagnosed now in a timely way, able to take</p> <p>15 that treatment every day, that their life</p> <p>16 expectancy would, as far as we can tell, be the</p> <p>17 same as if they didn't have HIV at this point</p> <p>18 and that was obviously not the case for a very</p> <p>19 long period of time.</p> <p>20 MS SCOTT: Just picking up on that point and</p> <p>21 developing it further, for someone who has been</p> <p>22 living with HIV for a long time but is on these</p> <p>23 modern drugs now and who has undetectable viral</p> <p>24 load, what is their life expectancy?</p> <p>25 DR WILLIAMS: Can I just -- I'll answer that</p>	<p style="text-align: right;">124</p> <p>1 question but quickly come back to the fact that</p> <p>2 in terms -- I don't want -- get the Inquiry to</p> <p>3 understand that although these modern drugs are</p> <p>4 much better tolerated, they are not without</p> <p>5 their issues for individual patients, so</p> <p>6 sleeping difficulties can still occur, anxiety</p> <p>7 for some patients, gastrointestinal symptoms,</p> <p>8 loss of bone density, so there are things that</p> <p>9 need to be monitored for even with these modern</p> <p>10 drugs, but they are, compared to the older</p> <p>11 drugs, are much better. So I would not want the</p> <p>12 Inquiry to think that they are absolutely</p> <p>13 perfect because they are not. They are much</p> <p>14 better than they used to be and people tolerate</p> <p>15 them much better but there are ongoing issues</p> <p>16 where we would like to improve on them.</p> <p>17 PROFESSOR COOKE: One of the more common things we</p> <p>18 still deal with is neural psychiatric effects,</p> <p>19 even from drugs today, often relatively mild and</p> <p>20 not as common as they used to be but nonetheless</p> <p>21 there and even with the new options we have we</p> <p>22 still sometimes have to rely on drugs which</p> <p>23 cause that and that obviously adds to issues</p> <p>24 that might exist before those drugs are started.</p> <p>25 MS EDWARDS: Can I add one thing onto this as well</p>



<p style="text-align: right;">125</p> <p>1 and that's I don't think we can underestimate</p> <p>2 adherence as an important component to this drug</p> <p>3 therapy. When you start newly on a drug and the</p> <p>4 doctor says you need to take this drug every day</p> <p>5 at the same time for the rest of your life, it</p> <p>6 is somewhat easier when you have been spending</p> <p>7 20 years plus taking all sorts of drugs that may</p> <p>8 not have worked, you are very sick, you have</p> <p>9 been very sick, and we were talking earlier on</p> <p>10 about psychological impacts and that we know</p> <p>11 that the psychological impact of depression and</p> <p>12 anxiety has a very negative effect on adherence.</p> <p>13 The second thing is about relationship</p> <p>14 with the healthcare service. I think that your</p> <p>15 relationship with your doctor, your relationship</p> <p>16 with your nurse, has a very strong correlation</p> <p>17 with how able you are, whether that is mentally</p> <p>18 or physically, to take these drugs and keep</p> <p>19 taking them at the same time every single day.</p> <p>20 So I think that history that you are talking</p> <p>21 about of people being unwell for a long time has</p> <p>22 a bigger impact on that aspect.</p> <p>23 PROFESSOR ANDERSON: I think there's another aspect</p> <p>24 which I think you might come to but the</p> <p>25 increasing understanding of the importance of</p>	<p style="text-align: right;">126</p> <p>1 peer support in things like adherence and so</p> <p>2 within clinical settings making sure that there</p> <p>3 are people with the experience of living with</p> <p>4 HIV and taking medication who can support people</p> <p>5 on that part of their treatment, because</p> <p>6 actually sometimes having experiential expertise</p> <p>7 is incredibly helpful in helping with that.</p> <p>8 MS SCOTT: I did ask a question about life</p> <p>9 expectancy but I can come back to that.</p> <p>10 PROFESSOR TUDOR-WILLIAMS: Just on that point,</p> <p>11 because you asked the question about if you had</p> <p>12 been living with this for 20 years how could</p> <p>13 you -- what's it like taking today's drugs.</p> <p>14 From my experience, every time we look at -- we</p> <p>15 are constantly trying to find the best possible</p> <p>16 tolerated options for the young people as they</p> <p>17 are growing up and as we have more things to</p> <p>18 offer, but every time we look at that</p> <p>19 decision-making we go back and look at the</p> <p>20 archived resistance.</p> <p>21 Ian mentioned this, the virus is very</p> <p>22 capable of lying dormant, latent inside cells</p> <p>23 for a very long time and the worry is that when</p> <p>24 you have been exposed to mono-therapy or dual</p> <p>25 therapy and you have archived those viruses with</p>
<p style="text-align: right;">127</p> <p>1 that resistance we take that into account when</p> <p>2 we think about the options today. I am sure it</p> <p>3 is true in adult practice, isn't it?</p> <p>4 PROFESSOR ANDERSON: Yes.</p> <p>5 MS SCOTT: What are the long-term effects of taking</p> <p>6 anti-retroviral therapy then, even the well</p> <p>7 tolerated drugs for people that are going to be</p> <p>8 taking it for a long time, for the rest of their</p> <p>9 lives?</p> <p>10 DR WILLIAMS: I think the answer to that is we don't</p> <p>11 know, to be honest. Clearly we have experience</p> <p>12 from some of the older drugs in that people --</p> <p>13 we have mentioned peripheral neuropathy. The</p> <p>14 symptoms of peripheral neuropathy can be</p> <p>15 long-standing and be a problem for many, many</p> <p>16 years, even after you have discontinued the drug</p> <p>17 that was causing the problem. Many of the</p> <p>18 earlier drugs like didanosine or stavudine have</p> <p>19 had long-term impacts on the liver and pancreas</p> <p>20 and people developing liver disease and</p> <p>21 pancreatic disease 10 or 15 years later. So</p> <p>22 there are those.</p> <p>23 If you asked me what are the long-term</p> <p>24 impacts of the current modern drugs, we haven't</p> <p>25 as yet seen any signals to be concerned but like</p>	<p style="text-align: right;">128</p> <p>1 any long-term medication we need to continue to</p> <p>2 be robust in being able to identify that. There</p> <p>3 are, for example, tenofovir, there are two forms</p> <p>4 of tenofovir: one is tenof DF and one is tenof</p> <p>5 AF. Tenof DF was the first version. That can</p> <p>6 be associated with chronic kidney disease but it</p> <p>7 can be also associated with bone loss, but</p> <p>8 that's largely been replaced by a newer version</p> <p>9 of tenofovir.</p> <p>10 Again, experience will tell us that there</p> <p>11 will be something down the line that may well --</p> <p>12 which has not been identified in clinical</p> <p>13 trials. Often you identify in clinical trials</p> <p>14 what are the most common side effects but when</p> <p>15 you put the drug into a larger patient</p> <p>16 population than a clinic population then you</p> <p>17 experience problems associated with that drug in</p> <p>18 a more diverse patient population.</p> <p>19 PROFESSOR COOKE: It's probably fair to say that we</p> <p>20 see these cycles of excitement and then</p> <p>21 tempering of that excitement over the years</p> <p>22 where we are dealing with patients with drugs</p> <p>23 where we know that they are not perfect,</p> <p>24 something new comes along, new and shiny, and</p> <p>25 everyone is excited about it and we tend to use</p>

<p style="text-align: right;">129</p> <p>1 more of it, and then gradually often we have</p> <p>2 found there are side effects and have had to</p> <p>3 rein back. Still there will be some patients</p> <p>4 who benefit but we learned a bit more about it,</p> <p>5 even with the newest drugs we are still going</p> <p>6 through that cycle I think.</p> <p>7 PROFESSOR ANDERSON: There's also an issue, I think</p> <p>8 Ian has alluded to it, the diversity, so as</p> <p>9 people who are living with HIV get older, not</p> <p>10 only are they taking drugs for a long time but</p> <p>11 they are physiologically changing, the human</p> <p>12 being is changing as well, so we don't</p> <p>13 necessarily know about how that's going to play</p> <p>14 out and there is far less data at the moment on</p> <p>15 women. They tend to be less well respected in</p> <p>16 clinical trials and older people are</p> <p>17 under-represented in clinical trials as well, so</p> <p>18 those are areas which we still have to find out</p> <p>19 about.</p> <p>20 MS SCOTT: That was a question in fact that somebody</p> <p>21 specifically asked me to ask the Panel, which is</p> <p>22 what impact is the ageing process going to have</p> <p>23 on the effectiveness of these medications?</p> <p>24 DR WILLIAMS: I don't think it's an impact on the</p> <p>25 effectiveness because I think as long as people</p>	<p style="text-align: right;">130</p> <p>1 take the medication then the viral replication</p> <p>2 cycle will be prevented. But clearly as people</p> <p>3 get older the dose of the drugs might need to be</p> <p>4 adjusted.</p> <p>5 We know little about that because of the</p> <p>6 way the drugs are handled by an individual as</p> <p>7 they get older, but I think there is an</p> <p>8 important issue about whether HIV has an impact</p> <p>9 on ageing in terms of the interface with someone</p> <p>10 getting older and the interface of disease</p> <p>11 associated with ageing and HIV, and whether</p> <p>12 there is -- HIV is associated with either</p> <p>13 accelerated ageing or attenuated ageing. It's</p> <p>14 generally thought to be associated with</p> <p>15 attenuation, that diseases of ageing tend to be</p> <p>16 more common in people with HIV rather than</p> <p>17 premature.</p> <p>18 MS SCOTT: Is there anything anyone else wanted to</p> <p>19 say about medication before I pick up on the</p> <p>20 life expectancy question?</p> <p>21 So we started to digress into life</p> <p>22 expectancy and I think what you said was for</p> <p>23 somebody who's newly infected and goes on to</p> <p>24 these modern drugs, life expectancy is pretty</p> <p>25 much as it is for somebody that doesn't have</p>
<p style="text-align: right;">131</p> <p>1 HIV.</p> <p>2 The next obvious question is, well, what's</p> <p>3 the impact on life expectancy compared to</p> <p>4 somebody that doesn't have HIV?</p> <p>5 SIR BRIAN LANGSTAFF: May I just ask a question</p> <p>6 about that. It really arises out of what you</p> <p>7 were saying about the long-term effects of</p> <p>8 having drugs. It must be the case that no-one</p> <p>9 in the UK has suffered from AIDS for longer than</p> <p>10 40 years, so so far as those with haemophilia</p> <p>11 are concerned the cohort may be largely younger</p> <p>12 than older. We just -- how do we know what the</p> <p>13 life expectancy is going to be?</p> <p>14 [Applause]</p> <p>15 PROFESSOR ANDERSON: I don't think we do.</p> <p>16 DR WILLIAMS: We don't know the answer, Sir Brian.</p> <p>17 I think that's a very important point and</p> <p>18 I think all we can do is estimate through</p> <p>19 epidemiological models. So people more with</p> <p>20 experience in epidemiology and life expectancy</p> <p>21 are the ones who have looked at this, but what</p> <p>22 they do is they look at the incidence of</p> <p>23 different diseases associated in someone who is</p> <p>24 aged 60 or 70 and they extrapolate from there in</p> <p>25 terms of what the deemed life expectancy is.</p>	<p style="text-align: right;">132</p> <p>1 But no, you are absolutely right, I don't</p> <p>2 know what's around the corner in terms of this.</p> <p>3 There could be something that could develop,</p> <p>4 that would be, you know, that is unexpected but</p> <p>5 experience to date suggests that once you --</p> <p>6 when people have been on therapy for a long time</p> <p>7 and they have recovered their immune system,</p> <p>8 then generally they live -- their incidence of</p> <p>9 disease associated with severe immunosuppression</p> <p>10 is no longer there but what there is a concern</p> <p>11 about is, which I alluded to earlier in the</p> <p>12 previous session, was the fact there are issues</p> <p>13 around diseases of ageing and cancers. That's</p> <p>14 what I am seeing now in my cohort, is that --</p> <p>15 SIR BRIAN LANGSTAFF: Presumably the effects of</p> <p>16 mental problems.</p> <p>17 DR WILLIAMS: Absolutely, yes. Absolutely right.</p> <p>18 So living longer is fine but living well and</p> <p>19 longer is really important and that's where we</p> <p>20 at this moment in time can't guarantee that.</p> <p>21 MS SCOTT: Turning then to those that have been</p> <p>22 living with HIV for many years, what do we know</p> <p>23 about life expectancy for those patients?</p> <p>24 DR WILLIAMS: I think it's a really -- I think it's</p> <p>25 a question that I can't answer to a certain</p>

<p style="text-align: right;">133</p> <p>1 extent because I don't think we have, you know, 2 as I said before, there are epidemiological 3 models which can model this but most people, the 4 way they publish this data, is looking at 5 someone diagnosed now, right. 6 Someone living, you know, who has been 7 living with HIV for 20/25 years has had a 8 damaged immune system for much longer time than 9 someone who is diagnosed now. Having a damaged 10 immune system for a long period of time has its 11 long-term sequelae, particularly in terms of 12 cancers. 13 As I said, frailty is a problem in people 14 getting older with HIV because it's not just 15 about living longer, it's about the quality of 16 life, and frailty in someone with HIV living 17 longer is much higher so they tend to have much 18 more conditions of ageing, the prevalence of 19 those conditions is higher, right. Therefore, 20 diabetes, blood pressure problems, 21 cardiovascular disease, renal disease, they tend 22 to have more complex problems. So, yes, life 23 expectancy has improved a great deal but where 24 we are now is trying to improve and reduce these 25 other conditions that impact on frailty and</p>	<p style="text-align: right;">134</p> <p>1 quality of life. 2 MS SCOTT: Professor Tudor-Williams, can I ask you 3 from your perspective the same question. In 4 your report you suggest that outcomes are better 5 for those that were infected between the ages of 6 5 to 15, as I understand it, in terms of life 7 expectancy. Is there any other information that 8 you are able to share with us about that? 9 PROFESSOR TUDOR-WILLIAMS: I mean, there is limited 10 data specifically from haemophiliac cohorts 11 looking at that kind of actuarial survival that 12 would suggest that those that were infected at 13 a younger age have done better than those 14 infected who were older. I think that just 15 simply reflects the older ones already had 16 co-morbidities that were prejudicial, that gave 17 a survival advantage if you are infected 18 younger. 19 I think -- I tried to put a little bit of 20 data into the report relating to our cohort of 21 young people who have been living with the virus 22 that was perinatally acquired, so they reached 23 their 20s having already lived with the virus 24 for 20 years. It's clear that there are 25 increased morbidities and mortality rate that is</p>
<p style="text-align: right;">135</p> <p>1 related to both an increased risk of cancer, 2 some increased risk of mental health issues. 3 Our own cohort data shows that there's probably 4 a 13 per cent higher incidence in our young 5 people up to the age of 24 compared to the 6 non-HIV infected same population and also 7 a 20 per cent likelihood of mental health issues 8 with three of our cohort who have attempted 9 suicide. 10 I think, therefore, it's incredibly 11 difficult, as I said, that the long-term 12 outcomes for the young people who are infected 13 surviving into adulthood are really difficult to 14 model at the moment and I think we would be 15 unwise to be drawn into guesswork about what the 16 future holds because whilst there may be some 17 unexpected and adverse problems lying ahead, 18 equally there may be some amazing breakthroughs 19 that everybody's going to be able to take 20 advantage of. 21 I remain an eternal optimist from that 22 point of view and I really hope that anything we 23 say today will be trumped by advances in therapy 24 tomorrow. 25 MS EDWARDS: Can I say that I don't think we can</p>	<p style="text-align: right;">136</p> <p>1 underestimate the mental health issues. We're 2 not talking about -- we're talking about mental 3 health issues about, certainly with people who 4 have had haemophilia who were diagnosed very 5 young, and all that whole issue of the problems 6 with schooling, the problems with lack of 7 interest or ability to have a future that they 8 could hold on to, they have been told that they 9 were going to die. They've watched their 10 friends die. They have watched their brothers 11 die, you know, and we've discussed all this on 12 Tuesday but I think that it's a profound effect 13 on the mental health. 14 So if you have been living not only with 15 these physical illnesses that we have been 16 talking about, these long-term treatments we 17 have been talking about but also a sort of 18 a loss and grief in your life that there's a lot 19 to deal with that interrelate with one another. 20 MS SCOTT: Sir, I note the time. If this is 21 a convenient time to break we will be collecting 22 questions from any core participants to be put 23 to the expert Panel in the last session. 24 SIR BRIAN LANGSTAFF: Thank you for that. I think 25 it probably is convenient. Let us be back here</p>



<p>1 then at 3.30.</p> <p>2 (3.01 pm)</p> <p>3 (A short break)</p> <p>4 (3.30 pm)</p> <p>5 SIR BRIAN LANGSTAFF: Before we turn to the next</p> <p>6 topic, could I just check something with you and</p> <p>7 see that I understand it correctly, and you can</p> <p>8 tell me where I've gone wrong.</p> <p>9 Can we have a look, please, Henry, at</p> <p>10 what -- the picture on page 3 -- it's a couple</p> <p>11 of pages further on. That's the one.</p> <p>12 This is described as HIV treatment</p> <p>13 explained. To understand what the explanation</p> <p>14 actually is, can I just see that my</p> <p>15 understanding is right.</p> <p>16 The little dot at the top, the -- labelled</p> <p>17 "HIV", that's where one starts with -- that's</p> <p>18 the viral particle. The big circle in the</p> <p>19 middle is the cell, with the nucleus in the</p> <p>20 centre, and the net result of the viral particle</p> <p>21 entering the cell is to render the cell</p> <p>22 ineffective at doing its job in the immune</p> <p>23 system and you end up with -- well, here it</p> <p>24 shows five little particles coming out of the</p> <p>25 far end.</p>	<p>1 I suspect that's representational but it</p> <p>2 means that one viral particle comes in, a number</p> <p>3 of -- more come out, it clones itself?</p> <p>4 You are nodding. That's for the</p> <p>5 transcript.</p> <p>6 PROFESSOR ANDERSON: Yes.</p> <p>7 DR WILLIAMS: If I can say that the nucleus is</p> <p>8 rather like a computer, and so what the virus is</p> <p>9 doing is basically encoding itself into the</p> <p>10 computer.</p> <p>11 SIR BRIAN LANGSTAFF: Can we just take it stage by</p> <p>12 stage.</p> <p>13 First of all, it's got -- this viral</p> <p>14 particle has got to get into the cell, through</p> <p>15 the cell wall.</p> <p>16 DR WILLIAMS: That's right.</p> <p>17 SIR BRIAN LANGSTAFF: You mentioned earlier, though</p> <p>18 I don't think it's shown directly on this, the</p> <p>19 importance of the CCR5 co-receptor, which some</p> <p>20 cells don't have, most do, and it's by binding</p> <p>21 to those, is it, that the particle can get in,</p> <p>22 into the centre of the cell?</p> <p>23 DR WILLIAMS: Yes, there's three stages to HIV</p> <p>24 entry. There's the -- the virus will bind</p> <p>25 directly to a CD4 molecule, through its gp120,</p>
<p>1 then it will then bind to the co-receptor, which</p> <p>2 is either CCR5 but there are others, and then it</p> <p>3 will -- then there's a third stage, which is</p> <p>4 fusion with the cell membrane, which then allows</p> <p>5 the virus to inject the viral RNA into the cell</p> <p>6 cytoplasm.</p> <p>7 SIR BRIAN LANGSTAFF: So here we are with the orange</p> <p>8 arrow showing the little particle coming through</p> <p>9 the cell wall, and then at stage 3 we have the</p> <p>10 HIV RNA. Now you have described how HIV</p> <p>11 consists of a strand of RNA, whereas what is in</p> <p>12 a cell, gives you the cellular message, is DNA.</p> <p>13 DR WILLIAMS: That's right.</p> <p>14 SIR BRIAN LANGSTAFF: Reverse transcription is</p> <p>15 creating DNA out of RNA, is it, rather than the</p> <p>16 other way round, which is transcription --</p> <p>17 DR WILLIAMS: Yes.</p> <p>18 SIR BRIAN LANGSTAFF: -- since reverse</p> <p>19 transcription?</p> <p>20 PROFESSOR ANDERSON: Yes, exactly.</p> <p>21 SIR BRIAN LANGSTAFF: So there we see the two</p> <p>22 separate strands of RNA combining to form a dual</p> <p>23 strand?</p> <p>24 DR WILLIAMS: It's a single strand of RNA.</p> <p>25 SIR BRIAN LANGSTAFF: Single strand? Right. So</p>	<p>1 there are two single strands but it curls itself</p> <p>2 round to the double helix.</p> <p>3 DR WILLIAMS: Absolutely, into a double strand of</p> <p>4 DNA.</p> <p>5 SIR BRIAN LANGSTAFF: So you've got a bit of DNA but</p> <p>6 it's not a complete bit of DNA, not a</p> <p>7 complete --</p> <p>8 DR WILLIAMS: It's the viral -- it's encode -- code</p> <p>9 -- it's all the viral genes, right, but it's in</p> <p>10 a DNA rather than RNA copy, and so it allows the</p> <p>11 DNA then to be encoded into the cell nucleus.</p> <p>12 SIR BRIAN LANGSTAFF: And if it is encoded in that</p> <p>13 way then it can replicate?</p> <p>14 DR WILLIAMS: Because when the cell is then</p> <p>15 activated, the cell reads its -- the DNA in its</p> <p>16 nucleus and will then produce new products as a</p> <p>17 result of reading its cell -- its cell DNA.</p> <p>18 PROFESSOR COOKE: It's that integration that makes a</p> <p>19 cure so difficult that is different from other</p> <p>20 viruses, so that that DNA is then integrated</p> <p>21 into a -- the human DNA.</p> <p>22 SIR BRIAN LANGSTAFF: Right. So the first stage is</p> <p>23 entry, the second stage is reverse</p> <p>24 transcription, and then once this bit of DNA has</p> <p>25 been produced, it has to be integrated into the</p>

<p style="text-align: right;">141</p> <p>1 centre of the cell. That's the integration 2 that's stage 4, I think, as shown here. 3 PROFESSOR ANDERSON: That's right. 4 SIR BRIAN LANGSTAFF: Then, having got in, the new 5 viral material is produced, and that's what you 6 say -- you say -- or the diagram says here it's 7 cut and assembled by protease, which is another 8 protein, is it? 9 DR WILLIAMS: Yes, it's the -- the virus encodes 10 with three enzymes. It has three enzyme 11 products, which is reverse transcriptase, 12 integrase and protease, and it needs all those 13 three enzymes to be able to produce a new virus 14 particle. 15 SIR BRIAN LANGSTAFF: And each of them at 16 a different stage. 17 DR WILLIAMS: That's right. 18 SIR BRIAN LANGSTAFF: So there's the entry first of 19 all, there's reverse transcriptase, the enzyme 20 which allows for reverse transcription. Then 21 you have integrase which allows it to integrate 22 into the centre of the cell. Having got into 23 the centre of the cell, it starts to reproduce, 24 and that's when you need protease -- 25 DR WILLIAMS: It will reproduce -- reproduce only if</p>	<p style="text-align: right;">142</p> <p>1 the cell is activated. So if the cell is not 2 activated, the viral DNA -- pro-viral DNA will 3 remain in the cell nucleus and the cell will 4 become a resting T cell which might live for 5 many tens of years. And that is the reservoir 6 of HIV infection that is so difficult to 7 eliminate. 8 SIR BRIAN LANGSTAFF: So the way in which -- or the 9 bits which the treatments you have told us about 10 target are, first of all, you have told us about 11 nucleoside and non-nucleoside reverse 12 transcriptase inhibitors, and what -- the way 13 that the non -- sorry, the way the nucleoside 14 work is -- is that by competing with the -- 15 DR WILLIAMS: So it competes -- when you -- when you 16 build a DNA you need to build it with building 17 blocks, nucleic acids, okay? So the things like 18 zidovudine are -- are analogues of nucleic 19 acids, right, and so they compete with the 20 active site of the enzyme to get -- to be put in 21 -- into the expanding DNA chain. So that is 22 part -- that's the sort of -- competitive sort 23 of inhibition. 24 SIR BRIAN LANGSTAFF: So to use a homely analogy, 25 it's a bit like elbowing the virus aside, trying</p>
<p style="text-align: right;">143</p> <p>1 to get -- trying to get into the queue. 2 DR WILLIAMS: That's right, yes, yes. 3 SIR BRIAN LANGSTAFF: Like a tube train, some people 4 may get on and some people may not. 5 DR WILLIAMS: Yes. 6 SIR BRIAN LANGSTAFF: So then the integration, 7 that's the integrase inhibitor, and -- oh, 8 I haven't asked you about the non-nucleoside. 9 Does that deal with the process of transcribing 10 -- (overspeaking) 11 DR WILLIAMS: What the non-nucleoside does is that 12 it binds something slightly separate to -- 13 slightly distant to the active site of the 14 enzyme, but in doing so it distorts the active 15 site and therefore the substrate, which is the 16 nucleic acid, can no longer be bound to it and, 17 therefore, it stops the work of the -- of the 18 enzyme. 19 PROFESSOR TUDOR-WILLIAMS: It sort of buckles the 20 door of the tube train. 21 SIR BRIAN LANGSTAFF: That's what I thought, yes, 22 thank you. 23 MS EDWARDS: It's like a zip, in fact, you know, the 24 teeth of a zip. And you can -- if you -- one of 25 the teeth of the zip doesn't work, then it won't</p>	<p style="text-align: right;">144</p> <p>1 go down. And the non-nucleosides, I -- this is 2 how I explain it to nurses anyway -- and the 3 non-nucleosides are like putting a bit of cotton 4 or fluff in the actual zip at the top so you're 5 not going to go anywhere at all. All those 6 little teeth of the zip are like the nucleosides 7 going down the zip. 8 SIR BRIAN LANGSTAFF: With the working of integrase, 9 do you have an explanation for that? 10 DR WILLIAMS: Well, integrase will allow the DNA in 11 a nucleus to be spliced and allow for the -- the 12 pro-viral DNA to be inserted. So what it does 13 is it stops that from happening and therefore 14 the pro-viral DNA just loops itself round and is 15 not encoded. 16 SIR BRIAN LANGSTAFF: So a sort of cut and paste. 17 DR WILLIAMS: That's right, yes. 18 SIR BRIAN LANGSTAFF: Then the -- 19 DR WILLIAMS: Not pasted, just cut. 20 SIR BRIAN LANGSTAFF: Just pasting -- 21 MS EDWARDS: No pasting. 22 SIR BRIAN LANGSTAFF: No, right. Sorry. 23 And then the protease, that's taking apart 24 proteins and reassembling them in the -- 25 DR WILLIAMS: Yes, so larger -- the -- when the cell</p>

<p style="text-align: right;">145</p> <p>1 is activated it will produce new viral RNA, and</p> <p>2 it will produce new messenger RNA, and the</p> <p>3 messenger RNA is responsible for producing large</p> <p>4 polyproteins, and those polyproteins need to be</p> <p>5 spliced in order to have a mature virus</p> <p>6 particle.</p> <p>7 SIR BRIAN LANGSTAFF: When we're talking about</p> <p>8 triple therapy, it might be thought that what</p> <p>9 the -- the aim of the therapy would be to have</p> <p>10 something which stopped reverse transcription,</p> <p>11 something which stopped integration, and</p> <p>12 something which hindered reassembly by protease.</p> <p>13 Is it like that or do we actually have two</p> <p>14 different drugs which have a go at reverse</p> <p>15 transcription?</p> <p>16 DR WILLIAMS: All -- all you really need to be able</p> <p>17 to do is to stop the virus from mutating.</p> <p>18 Right? So it stops -- you can stop the virus</p> <p>19 replicating, right, but what you don't want to</p> <p>20 do is allow the virus to slip out and transform</p> <p>21 itself to something that will still replicate in</p> <p>22 the presence of drug. If you have -- it's</p> <p>23 rather like if you treat TB, TB, if you only</p> <p>24 give it one drug, it will get resistance very</p> <p>25 quickly. You need combination therapy to treat</p>	<p style="text-align: right;">146</p> <p>1 TB. So it's the same sort of thing.</p> <p>2 Because the drugs are acting at different</p> <p>3 sites and there's more than -- there's three of</p> <p>4 them involved, then it's much more complex and</p> <p>5 much more difficult for the virus to escape that</p> <p>6 sort of hold.</p> <p>7 SIR BRIAN LANGSTAFF: Essentially that's how triple</p> <p>8 therapy works, is it?</p> <p>9 PROFESSOR COOKE: To answer your question there,</p> <p>10 then, it's unusual to have one of each.</p> <p>11 PROFESSOR ANDERSON: Exactly, yes.</p> <p>12 PROFESSOR COOKE: It does tend, partly for</p> <p>13 historical reasons, to be two drugs targeting</p> <p>14 the reverse transcriptase, combined with one</p> <p>15 other.</p> <p>16 PROFESSOR ANDERSON: Exactly.</p> <p>17 PROFESSOR COOKE: At least when you start.</p> <p>18 PROFESSOR ANDERSON: So the "triple" means three</p> <p>19 different molecular drugs, not three different</p> <p>20 sites of action.</p> <p>21 SIR BRIAN LANGSTAFF: But -- and one site of action</p> <p>22 will almost always be reverse transcriptase?</p> <p>23 PROFESSOR ANDERSON: At the moment, yes.</p> <p>24 SIR BRIAN LANGSTAFF: And it may be one of the other</p> <p>25 sites.</p>
<p style="text-align: right;">147</p> <p>1 PROFESSOR ANDERSON: Exactly.</p> <p>2 SIR BRIAN LANGSTAFF: Did I understand the -- from</p> <p>3 your report that -- we have four stages. Entry</p> <p>4 is the first stage, which is not necessarily the</p> <p>5 same sort of enzyme process as the other three,</p> <p>6 but that there are now drugs which are being</p> <p>7 developed or have been developed to stop entry</p> <p>8 as well?</p> <p>9 PROFESSOR ANDERSON: Yes.</p> <p>10 PROFESSOR COOKE: Yes.</p> <p>11 SIR BRIAN LANGSTAFF: Right.</p> <p>12 PROFESSOR TUDOR-WILLIAMS: T20 was an example of</p> <p>13 that totally new class of drugs, which is why it</p> <p>14 was effective in the face of a lot of resistance</p> <p>15 to reverse transcription, for instance.</p> <p>16 So once you got reverse transcriptase that</p> <p>17 was no longer inhibited by the existing drugs,</p> <p>18 T20 blocked that whole fusion process, and there</p> <p>19 are now new molecules in that class being</p> <p>20 developed.</p> <p>21 SIR BRIAN LANGSTAFF: But there's no point in doing</p> <p>22 all four together? All four stages?</p> <p>23 MS EDWARDS: We're getting down to two --</p> <p>24 PROFESSOR COOKE: Generally where there have been</p> <p>25 trials done of comparing four versus three,</p>	<p style="text-align: right;">148</p> <p>1 generally there hasn't been any advantage to</p> <p>2 doing that.</p> <p>3 MS EDWARDS: And in fact latest trials are actually</p> <p>4 going down to two drugs now.</p> <p>5 SIR BRIAN LANGSTAFF: Ah, right.</p> <p>6 PROFESSOR ANDERSON: And some of the other entry</p> <p>7 inhibitors block some of the co-receptors as</p> <p>8 well, so there are molecules that can block</p> <p>9 other bits of entry.</p> <p>10 DR WILLIAMS: What is important is the drug has</p> <p>11 a high genetic barrier to resistance. So if the</p> <p>12 virus needs to do a lot more work to get</p> <p>13 resistance to that drug, then that drug is then</p> <p>14 generally more successful. But it only needs</p> <p>15 one nucleic acid chain to get resistance, then</p> <p>16 that means that's a low genetic barrier drug.</p> <p>17 SIR BRIAN LANGSTAFF: Thank you very much indeed.</p> <p>18 I don't know if there are any questions which</p> <p>19 arise out of that for you, Ms Scott?</p> <p>20 MS SCOTT: I don't think so, sir.</p> <p>21 SIR BRIAN LANGSTAFF: That is all I am going to ask</p> <p>22 for the moment.</p> <p>23 MS SCOTT: In your report you've got a section on</p> <p>24 the impact of HIV on those with blood and</p> <p>25 bleeding disorders. Professor Rockstroh is</p>



<p style="text-align: right;">149</p> <p>1 coming to give evidence tomorrow, and you have</p> <p>2 told me that he is the best person to direct</p> <p>3 those questions to; is that right?</p> <p>4 DR WILLIAMS: Yes.</p> <p>5 MS SCOTT: So then can I move on then to the parts</p> <p>6 of your report that deal with the information</p> <p>7 that should be given to patients. I am going to</p> <p>8 ask you a couple of questions about diagnosis</p> <p>9 and then pick the report up at page 61 about</p> <p>10 what should be given to patients on diagnosis.</p> <p>11 The Inquiry has heard lots of evidence</p> <p>12 from different people infected by HIV that they</p> <p>13 were not told of their diagnoses at the time</p> <p>14 that the tests -- that it was discovered that</p> <p>15 they were HIV positive.</p> <p>16 Can I ask you whether or not it's ever</p> <p>17 been acceptable to withhold an HIV diagnosis</p> <p>18 from an adult patient with capacity?</p> <p>19 PROFESSOR ANDERSON: No.</p> <p>20 DR WILLIAMS: No.</p> <p>21 MS SCOTT: Has it ever been acceptable to withhold</p> <p>22 the diagnosis of HIV of a child from a parent</p> <p>23 with capacity?</p> <p>24 PROFESSOR TUDOR-WILLIAMS: No.</p> <p>25 MS SCOTT: Just sticking with then the diagnosis,</p>	<p style="text-align: right;">150</p> <p>1 Professor Tudor-Williams, you in your report --</p> <p>2 you, and your -- in the section of the report I</p> <p>3 believe you authored -- about children and</p> <p>4 adolescents make some comments about how</p> <p>5 practice has changed in terms of informing young</p> <p>6 people of their diagnosis of HIV themselves.</p> <p>7 Can you just tell us about that.</p> <p>8 PROFESSOR TUDOR-WILLIAMS: So back in the early days</p> <p>9 when we didn't have very great treatments to</p> <p>10 offer, there was a huge worry and -- I mean,</p> <p>11 obviously much of my experience was with young</p> <p>12 people who were infected as a result of vertical</p> <p>13 transmission rather than through blood products.</p> <p>14 Of the children in this country who were</p> <p>15 infected through blood products it was about</p> <p>16 10 per cent of the overall two and a half</p> <p>17 thousand or so children who have lived with HIV.</p> <p>18 So there were many reasons -- we were very</p> <p>19 often -- it was a family condition, we were very</p> <p>20 often dealing with parents whose were themselves</p> <p>21 infected, who were dealing with their own</p> <p>22 feelings about that diagnosis, and who found it</p> <p>23 extraordinarily difficult to allow us to tell</p> <p>24 the young people growing up with HIV that that</p> <p>25 was their diagnosis. And the result was that we</p>
<p style="text-align: right;">151</p> <p>1 very often, not through our own desire but out</p> <p>2 of respect with dealing with the family's</p> <p>3 wishes, delayed telling the young people. And</p> <p>4 they would say, "Well, no, no, no, they are just</p> <p>5 about to go to secondary school, that's not</p> <p>6 a good time for them to know", and "No, no, no,</p> <p>7 they're just -- you know, they're not really</p> <p>8 into puberty yet so let's do it when they have</p> <p>9 gone through puberty." All sorts of reasoning</p> <p>10 would postpone us from letting the young person</p> <p>11 themselves know the name of the virus.</p> <p>12 I think part of that was because of the</p> <p>13 whole tombstone campaign and the huge stigma</p> <p>14 that was associated with it. They didn't want</p> <p>15 their -- their children to be burdened with this</p> <p>16 knowledge. But what we learned over the years</p> <p>17 was that in fact if you discover that you are</p> <p>18 living with a sexually transmissible virus at</p> <p>19 the time that you go through puberty and at the</p> <p>20 time you start negotiating intimate</p> <p>21 relationships, that is a disaster. That is</p> <p>22 really difficult for young people to cope with.</p> <p>23 And it is much, much better if they assimilate</p> <p>24 that knowledge long before they are into any</p> <p>25 kind of those personal relationship issues.</p>	<p style="text-align: right;">152</p> <p>1 And over the years we've really come to</p> <p>2 the point where we are much more, I think,</p> <p>3 paternalistic in a way with the parents and say,</p> <p>4 "Actually, we really -- in this instance we</p> <p>5 really do know best, please trust us, it's going</p> <p>6 to be a great deal better if your young -- if</p> <p>7 the youngster -- you know, who's your</p> <p>8 responsibility, your child, knows about this at</p> <p>9 ten or eleven years of age", with the name of</p> <p>10 the virus when it's not really a big deal. You</p> <p>11 know, HIV, human immunodeficiency virus, it's</p> <p>12 just a name, and it doesn't carry an enormous</p> <p>13 amount of weight.</p> <p>14 And then all of the education that goes on</p> <p>15 from that time forward prepares them to</p> <p>16 understand both themselves, where they stand in</p> <p>17 relation to other people, and we help -- I mean,</p> <p>18 what has made an enormous difference is this</p> <p>19 equation U equals U, undetectable equals</p> <p>20 untransmissible, so that they grow up -- and --</p> <p>21 and we're already in that position to be able to</p> <p>22 say, "Look here on the computer is your viral</p> <p>23 load. It's undetectable. That's great. It</p> <p>24 means that you will not pass this on to anybody</p> <p>25 else, provided you can keep taking the</p>

<p style="text-align: right;">153</p> <p>1 medicine."</p> <p>2 So it sets them up for a much, much better</p> <p>3 psychological experience going through</p> <p>4 adolescence.</p> <p>5 MS SCOTT: You were talking about the name "HIV" not</p> <p>6 having any meaning to them because they are so</p> <p>7 young and they don't know about the stigma of</p> <p>8 the disease. Is that what you are describing?</p> <p>9 PROFESSOR TUDOR-WILLIAMS: Well, it gets -- it gets</p> <p>10 over that hurdle of, "Oh no, we mustn't tell</p> <p>11 them the name of the virus." And actually, for</p> <p>12 younger kids, it's not a big deal. You know,</p> <p>13 it's just the name of a virus and -- and they</p> <p>14 don't go home and have sleepless nights about</p> <p>15 it, if you are 10 or 11. If you are 15 and you</p> <p>16 are hearing about it for the first time, that is</p> <p>17 really hard.</p> <p>18 MS SCOTT: So that they learn about the virus and</p> <p>19 how to manage it before they understand the</p> <p>20 stigma and the psychosocial impacts and so on of</p> <p>21 it.</p> <p>22 PROFESSOR TUDOR-WILLIAMS: Yes.</p> <p>23 MS SCOTT: Can we then -- you deal with these issues</p> <p>24 in great detail in your report about the advice</p> <p>25 and the information that should be given to</p>	<p style="text-align: right;">154</p> <p>1 patients on first diagnosis and what should</p> <p>2 happen to them once they have been diagnosed in</p> <p>3 terms of signposting them on to secondary care,</p> <p>4 et cetera, referring them to secondary care, and</p> <p>5 you deal with it in great detail, and for those</p> <p>6 that have the report in front of them it starts</p> <p>7 at page 61. We can probably deal with this</p> <p>8 fairly quickly.</p> <p>9 But can I ask you to explain what</p> <p>10 information should be shared with patients when</p> <p>11 they are being diagnosed, how that should be</p> <p>12 done and where patients should go after first</p> <p>13 diagnosis in terms of their HIV care.</p> <p>14 PROFESSOR ANDERSON: So I think it depends. We've</p> <p>15 got two approaches we've put in the report. The</p> <p>16 first is -- David might want to speak to -- if</p> <p>17 somebody receives a new diagnosis outside</p> <p>18 a specialist setting and what happens in</p> <p>19 a specialist setting. Ultimately, the</p> <p>20 information is going to be the same; it's the</p> <p>21 pathway in, I guess.</p> <p>22 However, I think what I would want to</p> <p>23 preface everything with is that information</p> <p>24 sharing and for people learning about what's</p> <p>25 happening is a process. There won't be a moment</p>
<p style="text-align: right;">155</p> <p>1 when you know everything or you know nothing, it</p> <p>2 will be a continuing ongoing discussion over --</p> <p>3 after many different conversations. So this is</p> <p>4 a process for people to be able to have all the</p> <p>5 information that they need. But maybe if we</p> <p>6 were to say what would be happening to somebody</p> <p>7 who might have a test in a primary care setting</p> <p>8 where we would begin.</p> <p>9 DR JOHNSTON: So testing in primary care is more</p> <p>10 common now than it used to be. There was almost</p> <p>11 a desire not to test at one stage. That has</p> <p>12 changed so now we will be testing in appropriate</p> <p>13 circumstances, and sometimes the result can be</p> <p>14 a surprise when it comes back or sometimes</p> <p>15 people are more prepared for that.</p> <p>16 So what will simply happen is that you</p> <p>17 would try to see the patient, arrange an</p> <p>18 appointment for them to come in, and chat them</p> <p>19 through. And I think in primary care you would</p> <p>20 be giving them very basic information, showing</p> <p>21 them the support that any good primary care team</p> <p>22 should provide for any condition. So we're</p> <p>23 looking to provide care, understanding, respect,</p> <p>24 excellence, insofar as that is provided in</p> <p>25 primary care. But I think the key thing is</p>	<p style="text-align: right;">156</p> <p>1 communication, and assuring them that you are</p> <p>2 talking to their secondary care team, their</p> <p>3 specialist team, that they will be referred on,</p> <p>4 hopefully they will get a timely appointment to</p> <p>5 be seen. You give them advice. I think the key</p> <p>6 thing is also explaining that this is something</p> <p>7 that obviously will be a bit of a shock to them.</p> <p>8 If they do want to come back and have a chat</p> <p>9 with you, if they've any questions, often</p> <p>10 informing patients that it is entirely</p> <p>11 appropriate to write a little list -- often</p> <p>12 doctors are thought to hate lists, but in these</p> <p>13 circumstances, "Come back with a list, these are</p> <p>14 the questions that you may want answered." And</p> <p>15 then offering to see them after they have seen</p> <p>16 their specialist team. Because often they go</p> <p>17 and see the big doctor, the specialist doctor in</p> <p>18 the specialist centre, and that can seem quite</p> <p>19 daunting. And they will have questions because</p> <p>20 the experience can generate questions that can</p> <p>21 quite simply be answered in primary care.</p> <p>22 And also, increasingly with specialist</p> <p>23 teams being at a distance, then there is much</p> <p>24 more that can be done in primary care in terms</p> <p>25 of follow-up blood tests and so on, so that</p>

<p>157</p> <p>1 people don't have to make onerous journeys to</p> <p>2 have very basic tests done that can be done in</p> <p>3 their local setting. So it's facilitating the</p> <p>4 care and ensuring that patients know that they</p> <p>5 are on a sort of continuum of care and we're all</p> <p>6 on the one side trying to support them. I think</p> <p>7 that's what good care looks like now.</p> <p>8 MS SCOTT: Out of that arises a couple of issues,</p> <p>9 one of which I know you are dealing with in the</p> <p>10 supplemental letter of instruction, but what you</p> <p>11 are describing requires those that are</p> <p>12 delivering care in primary setting to have some</p> <p>13 knowledge of HIV.</p> <p>14 DR JOHNSTON: Yes.</p> <p>15 MS SCOTT: And to be able to impart that to the</p> <p>16 patient, and you are going to be dealing with</p> <p>17 what training and so on medics working in those</p> <p>18 settings should be -- will have in the letter</p> <p>19 of -- in supplemental report.</p> <p>20 PROFESSOR ANDERSON: Correct, yes.</p> <p>21 DR JOHNSTON: So, again -- sorry, do you want me to</p> <p>22 comment on that?</p> <p>23 MS SCOTT: Yes, please do.</p> <p>24 DR JOHNSTON: Primary care, I mean, the number of</p> <p>25 patients with HIV in any individual GP's</p>	<p>158</p> <p>1 practice will be relatively small. So, again,</p> <p>2 as a GP, you will go and perhaps just check</p> <p>3 what's available in your local service. You may</p> <p>4 even speak to the specialist team that you are</p> <p>5 going to be referring to, because I think</p> <p>6 satisfaction equals performance minus</p> <p>7 expectation, and if you say to somebody they are</p> <p>8 going to get an appointment in two days and they</p> <p>9 don't get it for three weeks, then they are</p> <p>10 feeling anxious. So you want to find out what</p> <p>11 you are offering them.</p> <p>12 And also I think we would refer them to</p> <p>13 good sources of information because people as</p> <p>14 soon as they get this nowadays of course will go</p> <p>15 straight on to the internet, and the sources of</p> <p>16 information that come up aren't always just the</p> <p>17 most authoritative.</p> <p>18 MS SCOTT: The other thing that comes out of there</p> <p>19 is the referral to secondary care.</p> <p>20 Professor Anderson, maybe you want to pick this</p> <p>21 up?</p> <p>22 PROFESSOR ANDERSON: Yes, so there are national</p> <p>23 standards. Essentially in any service where HIV</p> <p>24 testing is being carried out, wherever it may</p> <p>25 be, there needs to be a clear mechanism and</p>
<p>159</p> <p>1 a pathway for people who test positive to move</p> <p>2 into the appropriate care that they need after</p> <p>3 that. And that might be from primary care, it</p> <p>4 could be in community testing settings, it could</p> <p>5 be a variety of settings. But the national</p> <p>6 standard is that everybody with a new HIV</p> <p>7 diagnosis should see a specialist team within</p> <p>8 two weeks. If they have any symptoms whatsoever</p> <p>9 that should probably be within a couple of days,</p> <p>10 and if somebody is already in hospital and is</p> <p>11 having an HIV test as part of a work-up for</p> <p>12 a medical problem, the team with responsibility</p> <p>13 for HIV within that hospital should be seeing</p> <p>14 that patient within 24 hours. So those are the</p> <p>15 national standards that the British HIV</p> <p>16 association has set, and we would expect people</p> <p>17 then to be working within those confines.</p> <p>18 Now, obviously, if somebody has an HIV test</p> <p>19 within, for example, a sexual health setting, they may</p> <p>20 actually be immediately able to meet somebody, and</p> <p>21 increasingly with point of care testing, you may find</p> <p>22 somebody who is having a test who is able to go</p> <p>23 straight from having a fingerprick test which is</p> <p>24 positive to meeting a specialist team potentially</p> <p>25 within hours.</p>	<p>160</p> <p>1 So it depends a little bit on where testing is</p> <p>2 done but the bottom line standards are two weeks if</p> <p>3 you are well, two days if you are not, and 24 hours if</p> <p>4 you are sick.</p> <p>5 MS SCOTT: And to a specialist team. So why is it</p> <p>6 so important?</p> <p>7 PROFESSOR ANDERSON: So a specialist team -- and by</p> <p>8 that I am -- I am particularly talking about</p> <p>9 a specialist team because you won't necessarily</p> <p>10 meet a consultant immediately or a doctor but</p> <p>11 the team will have that special -- specialist</p> <p>12 knowledge.</p> <p>13 So often somebody will -- the first person</p> <p>14 they will meet will be a specialist nurse who</p> <p>15 would be able to assess where people are at,</p> <p>16 because actually often some of the questions</p> <p>17 that people have require a bit more information</p> <p>18 than just a positive test. So to actually find</p> <p>19 out, start to take a full history, an</p> <p>20 examination, work out more information that's</p> <p>21 going to be able to inform the process going</p> <p>22 forward. So meeting a team of people who have</p> <p>23 got different skills, and -- and the majority of</p> <p>24 HIV care now is delivered through</p> <p>25 multidisciplinary teams. And we would also</p>



<p style="text-align: right;">161</p> <p>1 hope, and it's not universal, we would also hope</p> <p>2 that very early on -- and I am not going to say</p> <p>3 exactly how quickly -- somebody who has a new</p> <p>4 diagnosis of HIV would meet somebody else who's</p> <p>5 living with HIV and would be able to engage not</p> <p>6 only with professional expertise but with lived</p> <p>7 experience expertise as well.</p> <p>8 MS SCOTT: So this was what you referred to earlier,</p> <p>9 the peer support, and the importance of that in</p> <p>10 getting people to understand the importance of</p> <p>11 taking medication and coming to terms with their</p> <p>12 diagnosis, et cetera.</p> <p>13 PROFESSOR ANDERSON: Exactly, exactly. And</p> <p>14 a specialist team because -- and I think we've</p> <p>15 alluded to this before, there are -- we know now</p> <p>16 that the best outcomes, not only for the person</p> <p>17 but for public health as well, are to be</p> <p>18 starting therapy relatively quickly, and so</p> <p>19 having that specialist advice very early about</p> <p>20 what's available, what you might choose to take,</p> <p>21 what drugs would suit you best, when you want to</p> <p>22 start, do you want to start, how's it going to</p> <p>23 work, that's quite specialist information, but</p> <p>24 it needs to be given quite quickly. So it is</p> <p>25 really important that people have access to high</p>	<p style="text-align: right;">162</p> <p>1 quality, up-to-date, specialist knowledge about</p> <p>2 what is the best treatment for them which will</p> <p>3 allow them to have the best outcomes.</p> <p>4 MS SCOTT: Then the wider issues that arise with an</p> <p>5 infection like HIV, transmission to partners,</p> <p>6 children, et cetera, the psychosocial impacts</p> <p>7 and so on, at what point do those get picked up?</p> <p>8 And again, what specialist skills are required</p> <p>9 in order to give patients care in respect of</p> <p>10 those issues?</p> <p>11 PROFESSOR ANDERSON: So again, I think this -- it</p> <p>12 goes back to what I said at the very beginning:</p> <p>13 this is about a process, and there will be</p> <p>14 a multidisciplinary team with a variety of</p> <p>15 skills, and actually people probably need to</p> <p>16 meet a number of different professionals, either</p> <p>17 as a team together or one-to-one separately, but</p> <p>18 nonetheless it is really important that that</p> <p>19 expertise is available quickly but it may need</p> <p>20 multiple conversations before you cover</p> <p>21 absolutely everything.</p> <p>22 So I think at the very beginning people</p> <p>23 usually want to know: okay, I have HIV, what's</p> <p>24 stage is it at? Am I ill? What's going to</p> <p>25 happen? What treatment would you -- shall</p>
<p style="text-align: right;">163</p> <p>1 I start? Those are the sort of -- you know,</p> <p>2 I guess, the immediate things, but actually the</p> <p>3 next question is: what sort of relationship are</p> <p>4 you in? Are you having sex with somebody? What</p> <p>5 are you going to do about -- what do you want to</p> <p>6 do about telling other people?</p> <p>7 And where does that disclosure lie and how</p> <p>8 does that happen?</p> <p>9 There are specialist health advisers</p> <p>10 within sexual health clinics, and the BHIVA</p> <p>11 standards recommend that everybody should have</p> <p>12 the expertise of somebody to talk about how you</p> <p>13 might want to have the conversations with</p> <p>14 significant others, including family members,</p> <p>15 and to start to think about children as well.</p> <p>16 But those are big conversations. They</p> <p>17 don't happen, you know, in your first half-hour</p> <p>18 appointment. They are going to evolve over</p> <p>19 a period of time. Normally, and within our</p> <p>20 clinic and I think most clinics, somebody with</p> <p>21 a new diagnosis of HIV would be allocated about</p> <p>22 an hour of clinic time, and during that time</p> <p>23 would have probably a confirmatory test, they</p> <p>24 would have tests taken for CD4 count viral load,</p> <p>25 other baseline investigations. They're rarely</p>	<p style="text-align: right;">164</p> <p>1 -- they would rarely be done in primary care,</p> <p>2 although they might be. So not only would you</p> <p>3 be sharing information, you probably also would</p> <p>4 be gathering information at the same time, and</p> <p>5 then trying to piece the jigsaw together over</p> <p>6 subsequent visits.</p> <p>7 MS SCOTT: And then once that initial prescription</p> <p>8 of medication has taken place, what does the</p> <p>9 care look like from then on in?</p> <p>10 PROFESSOR ANDERSON: Well, I think, first of all,</p> <p>11 I wouldn't say "that initial prescription",</p> <p>12 I think you have to, first of all, think</p> <p>13 through -- I mean, you have heard from Ian,</p> <p>14 there are now multiple drugs on the market,</p> <p>15 multiple combinations, we have national</p> <p>16 guidelines, and there are alternative regimes</p> <p>17 that people may start. And also, you know, the</p> <p>18 idea that you are going to have a blood test one</p> <p>19 day and start treatment that's going to be for</p> <p>20 the rest of your life the next day, it's a big</p> <p>21 step. So often you may want to have some time</p> <p>22 to -- actually, the first -- it might even be</p> <p>23 the first few appointments -- thinking about</p> <p>24 what it means to start treatment, which is the</p> <p>25 best regime for you, which mixture is going to</p>

<p style="text-align: right;">165</p> <p>1 work best, coming back with questions.</p> <p>2 So, although increasingly people are being</p> <p>3 treated very quickly, and in some clinics people</p> <p>4 are being treated on the same day, I think</p> <p>5 normally people would want some space. So</p> <p>6 I don't think yet, and I am not going to say</p> <p>7 this is yet normal, that -- most people would</p> <p>8 probably leave their first visit with treatment</p> <p>9 information but not necessarily a prescription,</p> <p>10 and that prescription for some people may not be</p> <p>11 for some time yet.</p> <p>12 Now, obviously, if somebody has got very</p> <p>13 advanced infection and they are very sick, or</p> <p>14 they are in a hospital bed, then things are</p> <p>15 going to move in a different way. I mean,</p> <p>16 I would also reflect back that if somebody has</p> <p>17 primary infection and a very high viral load,</p> <p>18 then that might be another indication to move</p> <p>19 very quickly with anti-retroviral therapy. But</p> <p>20 actually most people will want -- you have a lot</p> <p>21 to digest, as David was saying, and that</p> <p>22 requires processing before you go out with</p> <p>23 a prescription.</p> <p>24 So the first few visits will be dependent</p> <p>25 on what the person needs, there will be perhaps</p>	<p style="text-align: right;">166</p> <p>1 visits more close together, where blood test</p> <p>2 results taken on visit one would be discussed</p> <p>3 a week or so later, but it will not necessarily</p> <p>4 be all the same in one package for everybody.</p> <p>5 I don't know if that resonates with your</p> <p>6 practice --</p> <p>7 DR WILLIAMS: No, I was going to say there were</p> <p>8 a couple of things where there -- if someone is</p> <p>9 diagnosed with an advanced disease with an</p> <p>10 opportunist infection, actually it may not be</p> <p>11 safe to start anti-retroviral therapy --</p> <p>12 PROFESSOR ANDERSON: Actually, that's a good point.</p> <p>13 DR WILLIAMS: -- straight away, because they --</p> <p>14 starting anti-retroviral therapy improves the</p> <p>15 immune system and people can get what's known as</p> <p>16 immune reconstitution syndrome and people can be</p> <p>17 unwell. So, for example, in patients presenting</p> <p>18 with TB with advanced disease, we treat the TB</p> <p>19 first and then start the anti-retroviral</p> <p>20 therapy.</p> <p>21 The other thing I think which drives,</p> <p>22 often, why people want to start therapy</p> <p>23 immediately is because they are concerned about</p> <p>24 onward transmission to others, and that is often</p> <p>25 a big drive in wanting to -- you know, they are</p>
<p style="text-align: right;">167</p> <p>1 concerned about their own health but they're</p> <p>2 concerned about the health of their partners and</p> <p>3 such like.</p> <p>4 MS SCOTT: Then once one's got over the initial</p> <p>5 stage, then what does -- what does the care look</p> <p>6 like? Is that delivered in specialist centres</p> <p>7 on an ongoing basis or do you discharge back to</p> <p>8 primary care once the viral load is --</p> <p>9 PROFESSOR ANDERSON: So for most adult patients,</p> <p>10 let's assume -- excluding somebody who is very</p> <p>11 sick and in hospital with complications or has</p> <p>12 got an opportunistic infection, the initial</p> <p>13 period, probably a number of weeks -- and I'm</p> <p>14 not going to be more specific than that -- where</p> <p>15 the assessment, the baseline tests are done,</p> <p>16 a treatment plan is -- is considered, and things</p> <p>17 start to move forward. If somebody starts</p> <p>18 anti-retroviral therapy, you would want to see</p> <p>19 them again at least two -- four weeks later, so</p> <p>20 that you are able to see if the medication is</p> <p>21 suiting somebody, how it's going, checking in.</p> <p>22 Often there would be a phone line or a nurse or</p> <p>23 a pharmacist who you could talk to as you start</p> <p>24 treatment as you go through. And most people</p> <p>25 would have met a specialist pharmacist before</p>	<p style="text-align: right;">168</p> <p>1 they start treatment to help with adherence.</p> <p>2 So that period of starting treatment will</p> <p>3 require different amounts of support from</p> <p>4 different people. It would normally be</p> <p>5 happening in a specialist centre. And then once</p> <p>6 somebody is stable on their therapy, by which</p> <p>7 I mean they are happy with it, and it is</p> <p>8 working, viral load's going down, you would see</p> <p>9 them less often. But once the viral load is</p> <p>10 completely undetectable, you would be starting</p> <p>11 to think about even six-monthly appointments.</p> <p>12 But it will depend on each person individually</p> <p>13 what support they need in that first bit. But</p> <p>14 that will nearly always happen within specialist</p> <p>15 care with, if somebody has been tested in</p> <p>16 primary care, liaison with letters, assuming the</p> <p>17 patient gives consent for that to happen.</p> <p>18 PROFESSOR TUDOR-WILLIAMS: Just one very quick point</p> <p>19 about the small kids. When we start</p> <p>20 anti-retroviral therapy on very young children,</p> <p>21 because their weight is changing and the drug</p> <p>22 dosage is dependent on the child's weight and</p> <p>23 their growth, we see them very frequently. We</p> <p>24 will be seeing them -- I mean, if we are</p> <p>25 starting under a year of age, we'll probably see</p>

<p style="text-align: right;">169</p> <p>1       them once a month, and then only as their growth</p> <p>2       stabilises out can we stretch those appointments</p> <p>3       out a bit more. But I mean most of my teenagers</p> <p>4       we're seeing maybe three times a year, in school</p> <p>5       holidays typically.</p> <p>6       PROFESSOR ANDERSON: And I think there's also a sort</p> <p>7       of interesting phenomenon: if somebody has a new</p> <p>8       HIV diagnosis, a lot of other worries may come</p> <p>9       up. So something which before they had an HIV</p> <p>10      diagnosis they might have dismissed as a minor</p> <p>11      thing and not been worried about may become</p> <p>12      a much bigger worry: oh, is this a sign of</p> <p>13      something?</p> <p>14             So having the availability of a phone</p> <p>15      line, somebody to be able to come in for an</p> <p>16      extra appointment if worries emerge is also very</p> <p>17      important. But that, again, will usually happen</p> <p>18      in a specialist setting.</p> <p>19      MS SCOTT: The targets for referral on to specialist</p> <p>20      secondary care are short, ambitious ones. Are</p> <p>21      you aware of any resource -- of funding issues</p> <p>22      nationally which affect the provision of</p> <p>23      services or care or treatment for HIV?</p> <p>24      PROFESSOR ANDERSON: Well, for the moment HIV</p> <p>25      treatment and care is free for everybody who is</p>	<p style="text-align: right;">170</p> <p>1       resident in the UK, and that's -- that's</p> <p>2       absolutely clear. So there should be no barrier</p> <p>3       for people to be able to access specialist care</p> <p>4       on a financial basis. And nobody should have to</p> <p>5       pay for that.</p> <p>6             However, I do think it's important --</p> <p>7       I would defer to David here -- how to access --</p> <p>8       if your nearest specialist centre is an hour and</p> <p>9       a half on the bus, how do you want -- there are</p> <p>10      actual physical access barriers, which I think</p> <p>11      are important. And I think there's also the --</p> <p>12      the disconnect, if you like, the way the funding</p> <p>13      flows, which is a bit more complicated, the</p> <p>14      money is there for specialist HIV treatment and</p> <p>15      care, but that's a different budget from some of</p> <p>16      the other care that people living with HIV might</p> <p>17      need. But in the short-term, there should not</p> <p>18      be a financial barrier for anybody having the</p> <p>19      HIV care that is appropriate for them.</p> <p>20      DR WILLIAMS: Just quickly, it is a self-referral so</p> <p>21      you don't need actually a GP referral. So</p> <p>22      people can attend any centre they want. Which</p> <p>23      is unusual compared to other conditions.</p> <p>24      PROFESSOR ANDERSON: That's really important. So</p> <p>25      the choice of where you might choose to go is</p>
<p style="text-align: right;">171</p> <p>1       also open, and the ability to go to either</p> <p>2       somewhere near you where you work or where you</p> <p>3       live is in your hands. You can choose.</p> <p>4       MS EDWARDS: Can I just add one thing that -- that</p> <p>5       we wrote in the report as well, is that the</p> <p>6       centres and all the conversations that Jane just</p> <p>7       said, it's not just even the patient; you know,</p> <p>8       the families or significant person in somebody's</p> <p>9       life is very much part of this decision-making.</p> <p>10      And -- and we wrote this -- you know, if</p> <p>11      somebody wants to bring their partner, their</p> <p>12      child to this consultation, that would be</p> <p>13      encouraged as a holistic way of caring for</p> <p>14      somebody with HIV these days.</p> <p>15      MS SCOTT: And one of the questions that's been left</p> <p>16      over to the supplemental report is: what is the</p> <p>17      optimum level -- what is the optimum model of</p> <p>18      care for somebody with HIV? And you are going</p> <p>19      to be addressing that in the supplemental</p> <p>20      letter.</p> <p>21      PROFESSOR ANDERSON: We are going to be addressing</p> <p>22      that. I think in summary now though we can say</p> <p>23      that it needs to be person-centred,</p> <p>24      co-ordinated, joined up.</p> <p>25      DR WILLIAMS: And integrated.</p>	<p style="text-align: right;">172</p> <p>1       PROFESSOR ANDERSON: And integrated with all the</p> <p>2       other things that are happening for somebody.</p> <p>3       And we will go into that in more detail.</p> <p>4       MS SCOTT: That was the end of the questions that</p> <p>5       I had. I have a handful of questions from core</p> <p>6       participants.</p> <p>7             Why do some people not contract HIV when</p> <p>8       exposed to the virus through blood products or</p> <p>9       blood transfusion? I think we addressed this</p> <p>10      briefly this morning.</p> <p>11      PROFESSOR COOKE: I think we addressed this this</p> <p>12      morning to some extent. So it's a balance</p> <p>13      between the exposure risk, how much virus might</p> <p>14      be in whatever exposure there is, balanced with</p> <p>15      the susceptibility an individual might have,</p> <p>16      which might vary partly for genetic reasons and</p> <p>17      partly for other reasons. So those factors</p> <p>18      interplay.</p> <p>19      MS SCOTT: Is it possible for somebody to clear HIV?</p> <p>20      MS EDWARDS: Two cases?</p> <p>21      DR WILLIAMS: There are people who are known as</p> <p>22      elite controllers who effectively -- through</p> <p>23      their immune systems, they are able to control,</p> <p>24      and if you're -- and they may well have an</p> <p>25      undetectable viral load but not on treatment,</p>



<p style="text-align: right;">173</p> <p>1 and they are at the one end -- another end of  2 the spectrum. There have only been two cases of  3 people who have eradicated HIV, usually through  4 a bone -- a stem cell transplant -- stem cell  5 transplantation for treatment of relapsed  6 lymphoma. And this comes down to the -- using  7 stem cells which don't express CCR5, which is  8 what we alluded to earlier. They -- those stem  9 cells mature into -- into the new immune system  10 but they are -- cannot -- but they are no longer  11 infected, cannot be infected by HIV because they  12 don't express CCR5.</p> <p>13 So there is a patient in Berlin, well  14 publicised, called Mr Brown, and there's a case  15 from The London Clinic, the clinic that I work  16 at, which was publicised in February last year.</p> <p>17 PROFESSOR COOKE: So we have a section in the report  18 about cure, and I know it's a common question  19 for patients in clinic continually. That's part  20 of the answer to that, is that in theory it can  21 be possible in a very small number of patients  22 to have cure but it relies on quite extensive  23 chemotherapy which can really only be justified  24 when you have another reason to have it, in  25 particular, cancer. So the cases Ian describes</p>	<p style="text-align: right;">174</p> <p>1 are both cases where patients needed  2 chemotherapy and then had to have a bone marrow  3 transplant. One of the research areas at the  4 moment is to understand how that process could  5 be done for more patients safely and at the  6 moment we're not in a position to be doing that.</p> <p>7 MS SCOTT: Just on the question of cure, yesterday  8 when evidence was being given by the hepatitis  9 expert panel, they were talking about curing  10 hepatitis C. What is the difference between HIV  11 and hepatitis C? Why is it HIV can't be cured?</p> <p>12 PROFESSOR COOKE: It's partly as we were discussing  13 earlier, this is a virus that integrates into  14 the host DNA and can also pass between cells,  15 even when therapy is effective. So trying to  16 get rid of that virus completely from the body  17 is very difficult, and it requires at the moment  18 this quite radical approach to try and do that.  19 Even in these two cases, and these are well  20 publicised, they are undergoing close monitoring  21 still because we don't really know the  22 durability of these procedures when they happen,  23 and Timothy Brown, I think, it has been over ten  24 years now, but the most recent case is less than  25 two years. So we're still learning what the</p>
<p style="text-align: right;">175</p> <p>1 durability of that could be in terms of whether  2 the virus could come back. We think probably  3 not but we don't know.</p> <p>4 MS SCOTT: You mentioned elite controllers. Did I  5 understand from your report that even elite  6 controllers will, if they don't have treatment,  7 they will lose control of the viral load  8 ultimately?</p> <p>9 PROFESSOR COOKE: Not necessarily, although  10 a proportion will do. But there is a group of  11 patients who appear to have an antibody response  12 but don't have virus that re-emerges or at least  13 hasn't yet.</p> <p>14 PROFESSOR TUDOR-WILLIAMS: You just asked about  15 hep C those in terms of what is the difference.</p> <p>16 PROFESSOR COOKE: So hepatitis C doesn't have that  17 process, as we discussed yesterday, where it  18 would integrate into the host DNA, for example.  19 So it's possible to eradicate the infection  20 completely from the body more straightforwardly  21 than it would be for HIV or hepatitis B, in  22 fact.</p> <p>23 MS SCOTT: Are the risks of infection in blood  24 products and blood transfusions greater than  25 exposure to the virus in other ways?</p>	<p style="text-align: right;">176</p> <p>1 DR WILLIAMS: The answer is yes.</p> <p>2 PROFESSOR ANDERSON: We talked about that earlier.</p> <p>3 DR WILLIAMS: It's a larger inoculum and directly  4 given into blood. So if you are looking at  5 sexual transmission, it's an inoculum within  6 genital fluids and the amount of HIV is going to  7 be smaller compared to a blood transfusion.</p> <p>8 PROFESSOR COOKE: You will sometimes see that quoted  9 as 100 per cent risk. I think, for reasons  10 we've discussed, it's probably not that but some  11 the reference texts that we have looked do  12 actually use that sort of level of risk.</p> <p>13 MS SCOTT: Does the fact of co-infection between HIV  14 and HCV or HBV mean that the -- I think evidence  15 was given or I asked a question about whether or  16 not the fact of co-infection between hepatitis  17 and HIV alters the natural course of HIV, to  18 which the answer was no. The question that's  19 asked is: does the fact of that co-infection,  20 which means that the immune system is fighting  21 the HIV, mean that in fact the patient is  22 impacted indirectly? So the immune system is  23 having to do more, it's on the treadmill, having  24 to run twice as fast because it's got hepatitis  25 and it's got HIV. So is it impacted in that</p>

<p style="text-align: right;">177</p> <p>1 way?</p> <p>2 PROFESSOR COOKE: I mean, in a sense, it's difficult</p> <p>3 to be clear about that but certainly in terms of</p> <p>4 the sort of activity we talked about before,</p> <p>5 then the exhaustion and the weakening of the</p> <p>6 immune system has consequences for the control</p> <p>7 of other viruses as a result of HIV and that's</p> <p>8 both true for hepatitis B and for hepatitis C.</p> <p>9 MS SCOTT: A similar-ish question: does the fact</p> <p>10 that someone is infected with HBV or HCV give</p> <p>11 rise to a greater statistical risk that they</p> <p>12 will also be infected with HIV?</p> <p>13 PROFESSOR COOKE: In general, yes. It depends very</p> <p>14 much on the routes of exposure and the risks and</p> <p>15 the geographic location. But there are many</p> <p>16 shared routes of transmission. So overall there</p> <p>17 is an increased risk.</p> <p>18 PROFESSOR ANDERSON: We would also say that</p> <p>19 hepatitis B and hepatitis C would be an</p> <p>20 indication for an HIV test. So it's really</p> <p>21 important.</p> <p>22 PROFESSOR COOKE: Absolutely. Everybody with either</p> <p>23 of those the infections needs to be tested for</p> <p>24 HIV and potentially more than once if there's</p> <p>25 ongoing exposure risk.</p>	<p style="text-align: right;">178</p> <p>1 MS SCOTT: The virus HHV8 which causes Kaposi's</p> <p>2 sarcoma, is that transmitted via blood and blood</p> <p>3 products?</p> <p>4 PROFESSOR COOKE: I'm not sure that's something</p> <p>5 we've looked at at this point.</p> <p>6 DR WILLIAMS: It's largely transmitted sexually.</p> <p>7 That's the reason why it's much more common in</p> <p>8 men who have sex with men than in other affected</p> <p>9 patient populations. It is something we can</p> <p>10 certainly comment on.</p> <p>11 PROFESSOR TUDOR-WILLIAMS: It can certainly be</p> <p>12 transmitted perinatally as well.</p> <p>13 PROFESSOR ANDERSON: We can explore and put into our</p> <p>14 supplementary report any data that we find.</p> <p>15 MS SCOTT: There are some questions that are of</p> <p>16 particular interest to women with bleeding</p> <p>17 disorders and they are this, that the Inquiry</p> <p>18 has heard evidence, that they experience more</p> <p>19 blood during menstruation so there's more blood</p> <p>20 on household items and more blood during</p> <p>21 intercourse. The questions that arise from that</p> <p>22 are: is there a higher risk of infection for</p> <p>23 those individuals through sex because there's</p> <p>24 more blood present?</p> <p>25 PROFESSOR ANDERSON: If people are living with HIV</p>
<p style="text-align: right;">179</p> <p>1 with an undetectable viral load, it should make</p> <p>2 no difference. Graham, would you agree with</p> <p>3 that?</p> <p>4 PROFESSOR COOKE: Yes. I think in the setting where</p> <p>5 people aren't on effective treatment it's harder</p> <p>6 to say. But generally where there is more blood</p> <p>7 during sexual intercourse we would imagine there</p> <p>8 would be more transmission. I don't think we</p> <p>9 have very good data on that.</p> <p>10 PROFESSOR ANDERSON: I don't think there's very much</p> <p>11 data at all but, nonetheless, a woman who is on</p> <p>12 effective therapy should not be worrying about</p> <p>13 a sexual transmission during menstruation.</p> <p>14 MS SCOTT: Equally, the concern about there's more</p> <p>15 blood around generally in their life, and so</p> <p>16 more blood on household items and so on, is are</p> <p>17 there an increased risk of infection to family</p> <p>18 from blood on household items.</p> <p>19 DR WILLIAMS: No.</p> <p>20 PROFESSOR ANDERSON: No, I don't know.</p> <p>21 MS EDWARDS: Reiterate: it's the viral load. If the</p> <p>22 treatment which everybody should have access to</p> <p>23 now is effective and viral load is undetectable,</p> <p>24 whatever circumstances we're talking about will</p> <p>25 be the same answer.</p>	<p style="text-align: right;">180</p> <p>1 PROFESSOR ANDERSON: Yes.</p> <p>2 MS SCOTT: These questions, as I understand it, are</p> <p>3 concerned with the historic position, so before</p> <p>4 effective treatment where the viral load was</p> <p>5 undetectable.</p> <p>6 PROFESSOR COOKE: I think we haven't addressed</p> <p>7 issues in the report here related to specific</p> <p>8 haematological conditions and I think --</p> <p>9 MS SCOTT: Perhaps those could be asked tomorrow of</p> <p>10 Professor Rockstroh.</p> <p>11 MS EDWARDS: We know that the presence of blood that</p> <p>12 contains HIV can be transmitted to another</p> <p>13 person if it is blood-to-blood contact.</p> <p>14 DR WILLIAMS: During sexual intercourse but, in</p> <p>15 terms of household items or looking after</p> <p>16 somebody with HIV, no. If people take</p> <p>17 precautions, you know, if they -- there have</p> <p>18 been reports of someone who is a carer who has</p> <p>19 acquired HIV through handling infected material</p> <p>20 from a patient, but that should not happen in</p> <p>21 terms of with universal precautions.</p> <p>22 MS SCOTT: So one of the other questions that they</p> <p>23 ask is: how long does the virus live outside the</p> <p>24 body?</p> <p>25 PROFESSOR ANDERSON: Very short.</p>

<p style="text-align: right;">181</p> <p>1 MS SCOTT: What does that mean? Seconds, minutes,</p> <p>2 hours?</p> <p>3 MS EDWARDS: It doesn't -- effectively when you are</p> <p>4 talking about viral transmission, if you have</p> <p>5 blood on a kitchen table, even if it did live</p> <p>6 for two hours, you have still got to get it into</p> <p>7 a bloodstream. So what you have got to think is</p> <p>8 the issue about inoculum.</p> <p>9 DR WILLIAMS: Often transmission occurs through</p> <p>10 cells. So cells are responsible for -- because</p> <p>11 the cells that are infected with HIV get</p> <p>12 transferred across and cells will die very</p> <p>13 quickly outside the body and blood.</p> <p>14 MS EDWARDS: I think it's worth mentioning, though,</p> <p>15 that we may be talking in domestic situations</p> <p>16 where for something like children who had</p> <p>17 haemophilia who were bleeding, and I know that</p> <p>18 the bleeding is often joint bleeding but just</p> <p>19 something like profuse nosebleeds, which really</p> <p>20 are very, very profuse, the concern around</p> <p>21 a parent becoming infected from a child with HIV</p> <p>22 is a very real concern before we're talking</p> <p>23 about treatment when the idea of HIV in the</p> <p>24 blood is up there. That's a very real concern</p> <p>25 for people.</p>	<p style="text-align: right;">182</p> <p>1 PROFESSOR TUDOR-WILLIAMS: Can I add, it was a very</p> <p>2 real concern but actually the data as it emerged</p> <p>3 became immensely reassuring that there were, as</p> <p>4 far as I'm aware, absolutely zero transmissions</p> <p>5 to people caring for HIV infected youth. That's</p> <p>6 not true for hepatitis B.</p> <p>7 MS EDWARDS: I was going to say hepatitis is</p> <p>8 different.</p> <p>9 MS SCOTT: So another question they ask is would</p> <p>10 standard washing procedures destroy the virus,</p> <p>11 so we're talking about the blood on the table,</p> <p>12 wiping it away, that would get rid of the virus.</p> <p>13 PROFESSOR ANDERSON: Yes.</p> <p>14 MS EDWARDS: Absolutely.</p> <p>15 MS SCOTT: Then questions about treatment. Were</p> <p>16 there any additional side effects of HIV</p> <p>17 treatment and I think here we're talking about</p> <p>18 the older treatments for those who were</p> <p>19 co-infected with hepatitis C.</p> <p>20 PROFESSOR COOKE: There were and we also saw</p> <p>21 additive side effects from having combinations</p> <p>22 of treatments that would be potentially</p> <p>23 problematic and in the setting of inflammation</p> <p>24 in the liver, often one of the more common side</p> <p>25 effects we have not really touched on is</p>
<p style="text-align: right;">183</p> <p>1 inflammation in the liver as response to drugs,</p> <p>2 and in the setting of where there's already</p> <p>3 inflammation that can be exacerbated by the</p> <p>4 addition of HIV medication. So that's certainly</p> <p>5 something we recognise and in general patients</p> <p>6 who have co-infection with hepatitis tend to</p> <p>7 have more side effects from the same medication.</p> <p>8 MS SCOTT: Is there a link between Truvada and</p> <p>9 kidney infection?</p> <p>10 MS EDWARDS: Not kidney infection.</p> <p>11 DR WILLIAMS: But one of the drugs in Truvada, that</p> <p>12 is a branded name, it contains emtricitabine and</p> <p>13 one of the early versions of tenofovir,</p> <p>14 tenofovir can have, as a side effect, can affect</p> <p>15 part of the functioning of the kidney and cause</p> <p>16 kidney -- renal toxicity. But it's something we</p> <p>17 monitor for.</p> <p>18 MS EDWARDS: Yes, I was going to say people are</p> <p>19 monitored for all of these side effects very</p> <p>20 regularly.</p> <p>21 PROFESSOR ANDERSON: But it's not related to kidney</p> <p>22 infection.</p> <p>23 MS SCOTT: Another question about AZT treatment, the</p> <p>24 question is why would someone not have been</p> <p>25 given it, so were there any clinical</p>	<p style="text-align: right;">184</p> <p>1 contraindications for being prescribed AZT in</p> <p>2 the early days around 1987.</p> <p>3 DR WILLIAMS: In 1987 it was given to people who</p> <p>4 were ill with AIDS and people who had</p> <p>5 symptomatic disease. If someone was HIV</p> <p>6 antibody positive but otherwise well there</p> <p>7 wasn't any evidence that giving AZT to those</p> <p>8 patients they would have benefit. So it's</p> <p>9 largely targeted for people who had life</p> <p>10 threatening illnesses and infections.</p> <p>11 If, for example, someone had severe</p> <p>12 anaemia, then giving -- many people have thought</p> <p>13 about giving AZT because AZT can cause anaemia.</p> <p>14 PROFESSOR ANDERSON: Actually there was quite</p> <p>15 a prominent and very disappointing trial called</p> <p>16 the concord trial because we had seen these</p> <p>17 improvements in life expectancy in people who</p> <p>18 were very sick, a trial was put into place to</p> <p>19 see if it would help people who were less sick</p> <p>20 and the results showed that it didn't make</p> <p>21 a difference and so that was where the</p> <p>22 decision-making came from about it being used in</p> <p>23 sicker patients at that stage.</p> <p>24 MS EDWARDS: The side effects of AZT at the time,</p> <p>25 many people, it had a lot of bad press, and lots</p>



<p style="text-align: right;">185</p> <p>1 of people -- there was terminology around at the</p> <p>2 time, you know, it was the AZT that kills you,</p> <p>3 which obviously was not correct but people died</p> <p>4 despite being on treatment and so for some</p> <p>5 people they felt strongly they did not want to</p> <p>6 go on this drug and some medics would discourage</p> <p>7 it because of the fair comments around about the</p> <p>8 toxicities and the side effects.</p> <p>9 MS SCOTT: That's another question. What is known</p> <p>10 about deaths being caused by that treatment?</p> <p>11 DR WILLIAMS: I think -- in terms of what is known,</p> <p>12 as we were saying earlier, a lot of people felt</p> <p>13 that or it was put about that AZT was causing</p> <p>14 the deaths but it wasn't that, it was the AZT</p> <p>15 effect that was transient and, therefore, it</p> <p>16 only had short-term benefit and therefore people</p> <p>17 relapsed and went on to become ill as a result</p> <p>18 of their underlying HIV because the effective</p> <p>19 treatment of AZT was very short-term.</p> <p>20 That's not to say, for example, someone</p> <p>21 having AZT and becoming anaemic did not, you</p> <p>22 know, have side effects from that. I mean,</p> <p>23 there have been deaths associated with</p> <p>24 anti-retroviral therapy, for example, some of</p> <p>25 the non-nucleosides can cause very severe rash,</p>	<p style="text-align: right;">186</p> <p>1 a Stevens-Johnson rash, which can result in</p> <p>2 a really -- rash and a severe hepatitis, and</p> <p>3 people have died as a result of that. I think</p> <p>4 there's --</p> <p>5 PROFESSOR TUDOR-WILLIAMS: A sensitivity reaction,</p> <p>6 which is rather specific and genetically</p> <p>7 determined. But I think -- I mean, I remember</p> <p>8 very clearly coming back from the States in 1994</p> <p>9 full of enthusiasm to use AZT to try and prevent</p> <p>10 or decrease the risk of mother to child</p> <p>11 transmission and finding my colleagues being</p> <p>12 vilified for using AZT because it was regarded</p> <p>13 at that moment in history as a poison, and that</p> <p>14 was an echo chamber that led to people</p> <p>15 demonstrating outside Great Ormond Street</p> <p>16 Hospital accusing people there who were very</p> <p>17 well meaning physicians, paediatricians, trying</p> <p>18 to do their very best to help, being castigated</p> <p>19 as murderers. I mean, it really was a very</p> <p>20 uncomfortable time.</p> <p>21 PROFESSOR COOKE: I think it's worth emphasising, we</p> <p>22 have touched on this but it's worth emphasising,</p> <p>23 that because there was so much recognised</p> <p>24 toxicity with AZT and those early treatments</p> <p>25 they did tend to be left until they were really</p>
<p style="text-align: right;">187</p> <p>1 needed at a late stage, and so it was often</p> <p>2 quite a close association in time between</p> <p>3 starting them and death and I think it's quite</p> <p>4 understandable how people became concerned they</p> <p>5 were causing deaths but generally the evidence</p> <p>6 didn't support that directly.</p> <p>7 MS SCOTT: Excuse me, I have one question on my</p> <p>8 phone so I am not just reading my text messages.</p> <p>9 So this is a question in relation to the</p> <p>10 evidence that you gave about the immune system</p> <p>11 remaining hyper-activated and not returning to</p> <p>12 a resting state and we have a very, very visual</p> <p>13 image of being on the running machine.</p> <p>14 So the question is what effects would this</p> <p>15 hyper-activation have on the autonomic nervous</p> <p>16 system and what kind of cognitive psychological</p> <p>17 or physiological consequences might that have</p> <p>18 for the patient? And what might modulate those</p> <p>19 consequences?</p> <p>20 DR WILLIAMS: I think there are a couple of things</p> <p>21 there. One is that immune activation is</p> <p>22 a feature of untreated chronic infection and</p> <p>23 that leads to release from the immune system of</p> <p>24 lots of cytokines which are produced as</p> <p>25 inflammation. That's bad, generally bad. In</p>	<p style="text-align: right;">188</p> <p>1 terms of the autonomic nervous system,</p> <p>2 definitely like the peripheral nervous system</p> <p>3 and the central nervous system, it can be</p> <p>4 affected by HIV.</p> <p>5 Neurons are not directly infected by HIV</p> <p>6 but the support cells that support those</p> <p>7 neurones are and so you can -- people can get an</p> <p>8 autonomic neuropathy as well as getting</p> <p>9 peripheral neuropathy, and so the features of an</p> <p>10 autonomic neuropathy would be dizziness when you</p> <p>11 get up or a fast pulse rate and things like</p> <p>12 that. Certainly those are features that we</p> <p>13 have -- I have seen in patients with HIV.</p> <p>14 In terms of cognitive, it comes back to</p> <p>15 Graham's description of HIV in the brain and</p> <p>16 so -- the drugs don't -- how drugs get across</p> <p>17 the blood brain barrier may be different but</p> <p>18 definitely if you look at how -- if you look at</p> <p>19 HIV in the brain in people who died from HIV,</p> <p>20 HIV definitely affects the brain in terms of the</p> <p>21 support cells and results in loss of neurones</p> <p>22 and can affect people's cognition. There's no</p> <p>23 doubt about that.</p> <p>24 PROFESSOR COOKE: I agree completely, but I think</p> <p>25 the direct link between inflammation and those</p>

<p style="text-align: right;">189</p> <p>1 two things I think is less clear, although those</p> <p>2 two things are definitely recognised</p> <p>3 complications of HIV and its treatment.</p> <p>4 MS SCOTT: What is the norm to do? Is it the norm</p> <p>5 to do autopsies on all HIV deaths and, if so,</p> <p>6 when was that the norm and does it remain the</p> <p>7 norm?</p> <p>8 PROFESSOR ANDERSON: It's no longer the norm. There</p> <p>9 was a time I think early in the epidemic when</p> <p>10 there were a lot of very important and</p> <p>11 unanswered questions and people would die</p> <p>12 without us understanding why and we would ask,</p> <p>13 if we could ask for an autopsy, to understand</p> <p>14 better.</p> <p>15 I think now we're doing -- I think there</p> <p>16 are fewer but there was a period of time when</p> <p>17 there was a real uncertainty about what had</p> <p>18 happened and trying to find out.</p> <p>19 PROFESSOR COOKE: It differs slightly from centre to</p> <p>20 centre but I think generally there is an</p> <p>21 ambition still to do autopsies when the cause of</p> <p>22 death is unclear.</p> <p>23 PROFESSOR ANDERSON: Absolutely.</p> <p>24 PROFESSOR COOKE: Partly for the benefit of learning</p> <p>25 and knowledge, partly for the benefit of family,</p>	<p style="text-align: right;">190</p> <p>1 and there are still conditions that are emerging</p> <p>2 that we still need to learn about, so it's</p> <p>3 definitely something that's often requested</p> <p>4 although I think actually progressing to autopsy</p> <p>5 is less common than it used to be.</p> <p>6 MS SCOTT: What is known about the prevalence of HIV</p> <p>7 infection in, first of all, the United Kingdom</p> <p>8 population and, secondly, the UK blood donor</p> <p>9 population since 1970? Are you able to answer</p> <p>10 those questions?</p> <p>11 PROFESSOR ANDERSON: I don't know the date of the</p> <p>12 blood donor so --</p> <p>13 MS EDWARDS: Blood donors?</p> <p>14 MS SCOTT: The UK population and the UK blood donor</p> <p>15 population.</p> <p>16 PROFESSOR COOKE: I think we have a figure in the</p> <p>17 report. I think the figure quoted here is --</p> <p>18 no, that is C ... there's a figure in the report</p> <p>19 I think if someone could find it.</p> <p>20 PROFESSOR ANDERSON: The thing I think I would</p> <p>21 emphasise here is the prevalence of HIV is very</p> <p>22 variable across the UK, so there are areas where</p> <p>23 the prevalence is high and that is considered to</p> <p>24 be greater than 2 per 1,000 population, areas</p> <p>25 where it's very high where it's greater than 5</p>
<p style="text-align: right;">191</p> <p>1 per 1,000 population, but if you take the</p> <p>2 generality or the average across the UK, then</p> <p>3 that's lower.</p> <p>4 But I think what's important is looking at</p> <p>5 each particular area because things change very</p> <p>6 dramatically. The centre of London is quite</p> <p>7 different from a rural population outside</p> <p>8 London, for example, so there are some very</p> <p>9 clear prevalence diagrams from Public Health</p> <p>10 England where the exact prevalence can be seen.</p> <p>11 I think those will have been mapped. I'm not</p> <p>12 sure they have gone back to the -- we would have</p> <p>13 to look back. I think their records began in</p> <p>14 about '88/89.</p> <p>15 PROFESSOR TUDOR-WILLIAMS: Yes, I think so.</p> <p>16 PROFESSOR ANDERSON: But Public Health England are</p> <p>17 the guardians of that data.</p> <p>18 PROFESSOR COOKE: I think the figure we have in the</p> <p>19 report for the prevalence in first time donors</p> <p>20 is 0.03 per cent and I just need to check that</p> <p>21 because I think there might be a typo in the</p> <p>22 report which we will check in relation to HCV.</p> <p>23 That is I think the figure for HIV.</p> <p>24 PROFESSOR ANDERSON: In terms of, just going back to</p> <p>25 the prevalence in our supplementary report we</p>	<p style="text-align: right;">192</p> <p>1 have been asked some very specific questions on</p> <p>2 datasets, what is known, where it is, and we</p> <p>3 will put this into there.</p> <p>4 PROFESSOR TUDOR-WILLIAMS: The other source of data</p> <p>5 is from the national surveillance of HIV in</p> <p>6 pregnancy and childhood, the NHS -- there's</p> <p>7 a website that is very accessible that documents</p> <p>8 all of the seroprevalence rates and, as Jane</p> <p>9 says, these vary considerably across the nation.</p> <p>10 MS SCOTT: Just two more questions. Do you know</p> <p>11 what the death rates are for babies infected</p> <p>12 perinatally? So those that would have been</p> <p>13 infected perinatally.</p> <p>14 PROFESSOR TUDOR-WILLIAMS: Yes, I mean, I can tell</p> <p>15 you the death rates from cohort studies for the</p> <p>16 perinatal cohorts in Europe were published,</p> <p>17 natural history data, modified by a little bit</p> <p>18 of AZT use, but published in about 1996 showed</p> <p>19 that 15 per cent of the perinatal infected</p> <p>20 children were dead by the age of 6, whereas the</p> <p>21 same analysis performed in Malawi showed that</p> <p>22 15 per cent of the children were still alive at</p> <p>23 age 3 so, again, it comes back to all the other</p> <p>24 co-morbidities that children around the world</p> <p>25 with HIV are facing, but in Europe the overall</p>

<p style="text-align: right;">193</p> <p>1 survival rate was about 85 per cent at the age</p> <p>2 of 6.</p> <p>3 MS SCOTT: Are you aware of any studies on the</p> <p>4 infected blood community? So those that have</p> <p>5 been infected with HIV through blood and blood</p> <p>6 products?</p> <p>7 PROFESSOR ANDERSON: We are. We have referenced</p> <p>8 those in the report and I think tomorrow you</p> <p>9 will be able to get more detail from the team,</p> <p>10 the expert panel. We have put this in.</p> <p>11 MS SCOTT: We can pick that up tomorrow.</p> <p>12 PROFESSOR ANDERSON: Yes, the author of that</p> <p>13 particular point will be on the panel tomorrow.</p> <p>14 MS SCOTT: The second part of that question is are</p> <p>15 you aware of any studies on the stigma -- on</p> <p>16 those living with HIV for long-term, the</p> <p>17 consequences of that, and in particular in</p> <p>18 relation to stigma.</p> <p>19 PROFESSOR ANDERSON: Long-term ...?</p> <p>20 MS SCOTT: The long-term consequences of living with</p> <p>21 HIV.</p> <p>22 PROFESSOR ANDERSON: As whether people experience</p> <p>23 more stigma or less stigma or the impact of</p> <p>24 stigma in the long term?</p> <p>25 MS SCOTT: May I just turn behind me because I am</p>	<p style="text-align: right;">194</p> <p>1 not quite sure. <i>(Pause)</i></p> <p>2 I think there are two parts to that second</p> <p>3 part of the question. So I think studies on</p> <p>4 stigma in HIV and studies on the long-term</p> <p>5 consequences of living with HIV.</p> <p>6 PROFESSOR ANDERSON: So the long-term consequences</p> <p>7 of HIV, there are a number of studies ongoing at</p> <p>8 the moment and I think there's a couple with</p> <p>9 particular relevance. One is called the POPPY</p> <p>10 Study, which is looking at people who are both</p> <p>11 older with HIV and people who are older who do</p> <p>12 not have HIV, and the data that's coming from</p> <p>13 that is able to have a very close look at the</p> <p>14 differences between people who are living with</p> <p>15 HIV and getting older and the people who aren't.</p> <p>16 Data is coming from that study now and that's</p> <p>17 being co-ordinated by Imperial and UCL.</p> <p>18 In terms of stigma, there's a lot of work</p> <p>19 going on, I think one of the things I would say</p> <p>20 is that we are very well aware that</p> <p>21 HIV-associated stigma remains one of the key</p> <p>22 blocks to long-term good health because of some</p> <p>23 of the things we've discussed. We know that</p> <p>24 people are still not taking HIV tests early</p> <p>25 enough and so we're seeing ill health from late</p>
<p style="text-align: right;">195</p> <p>1 diagnosis, which is associated with stigma. We</p> <p>2 know that people are avoiding medical care, so</p> <p>3 stigma is having a direct impact on well-being.</p> <p>4 PROFESSOR COOKE: One of the things we were</p> <p>5 discussing outside was that I think as HIV is</p> <p>6 seen less as a public health crisis and more as</p> <p>7 a chronic infection increasingly in elderly</p> <p>8 populations, it's getting harder to motivate for</p> <p>9 resource for the kind of studies that will</p> <p>10 answer those questions. We have seen a recent</p> <p>11 example of where funding has ceased for the big</p> <p>12 UK cohort study which gave us a lot of this</p> <p>13 information.</p> <p>14 MS SCOTT: Those I think are the end of the</p> <p>15 questions that I have but can I just turn behind</p> <p>16 me to see if anything arose after the break.</p> <p>17 No, I don't think so. No further</p> <p>18 questions.</p> <p>19 SIR BRIAN LANGSTAFF: There's no question from me.</p> <p>20 When you were talking about advice, advising</p> <p>21 someone about their infection, you described the</p> <p>22 advice and learning as a process.</p> <p>23 I have to say that you collectively have</p> <p>24 contributed very much to what has been a process</p> <p>25 of learning for me. It's part of a process, in</p>	<p style="text-align: right;">196</p> <p>1 no way have I finished, but I must thank you</p> <p>2 very much for that. You might like to know that</p> <p>3 in the breaks a number of people have been</p> <p>4 speaking very highly, and entirely correctly so</p> <p>5 in my view, of the expertise and the empathy</p> <p>6 that you have shown.</p> <p>7 One of them described you as a fantastic</p> <p>8 group of experts. Another said that they were</p> <p>9 very grateful that they felt that you had, in</p> <p>10 what you had to say, acknowledged the symptoms</p> <p>11 which they have lived with for years, something</p> <p>12 which plainly is of real value.</p> <p>13 Can I thank you for that besides thanking</p> <p>14 you for having made the time to come here and</p> <p>15 share your expertise and indeed to give</p> <p>16 yourselves the task of going on to answer the</p> <p>17 supplementary questions and be available for any</p> <p>18 further expert advice that we may wish from you.</p> <p>19 Thank you.</p> <p>20 It is 10.30 tomorrow and we will be</p> <p>21 examining bleeding disorders, will we?</p> <p>22 MS SCOTT: Yes.</p> <p>23 SIR BRIAN LANGSTAFF: So 10.30.</p> <p>24 <b>(4.45 pm)</b></p> <p>25 <b>(Adjourned until 10.30 am the following day)</b></p>



<b>DR EDWARDS: [5]</b> 12/12 25/16 35/5 35/12 43/17 <b>DR JOHNSTON: [9]</b> 4/4 74/15 78/4 80/4 85/5 155/8 157/13 157/20 157/23 <b>DR WILLIAMS: [98]</b> 4/15 7/15 8/17 9/11 10/6 10/19 13/13 16/12 17/15 19/23 20/15 21/9 22/13 23/21 24/5 26/6 27/6 27/12 28/23 29/14 30/13 33/17 41/10 44/13 53/13 54/20 59/14 64/7 68/14 71/2 72/15 73/10 73/22 76/10 92/6 98/17 99/13 100/25 102/16 103/9 104/1 104/9 106/16 109/17 113/14 116/7 117/17 119/6 119/14 120/16 120/24 121/9 123/2 123/24 127/9 129/23 131/15 132/16 132/23 138/6 138/15 138/22 139/12 139/16 139/23 140/2 140/7 140/13 141/8 141/16 141/24 142/14 143/1 143/4 143/10 144/9 144/16 144/18 144/24 145/15 148/9 149/3 149/19 166/6 166/12 170/19 171/24 172/20 175/25 176/2 178/5 179/18 180/13 181/8 183/10 184/2 185/10 187/19 <b>MS EDWARDS: [33]</b> 3/6 51/17 55/13 62/8 62/18 63/14 67/14 75/12 77/1 79/14 85/24 100/9 110/25 113/6 121/8 124/24 135/24 143/22 144/20 147/22 148/2 171/3 172/19 179/20 180/10 181/2 181/13 182/6 182/13 183/9 183/17 184/23 190/12 <b>MS SCOTT: [159]</b> 1/21 2/19 3/5 4/3 4/14 5/1 5/24 6/8 6/12 6/15 7/12 8/4 8/14 8/18 9/12 10/15 11/19 12/10 13/2 13/9 15/23 16/2 17/4 19/11 20/13 20/25 22/9 23/18 24/1 25/23 27/1 27/7 28/19 29/9 30/11 31/10 33/4	33/11 35/1 36/3 36/24 37/18 38/7 40/16 40/20 42/2 42/4 43/15 44/21 45/1 45/9 45/21 49/3 49/20 50/7 50/13 52/11 52/20 54/16 54/19 54/21 55/12 56/17 59/24 60/14 61/11 62/3 63/18 64/22 66/16 68/24 71/9 73/7 73/19 74/4 77/24 81/12 87/2 87/13 87/25 89/17 90/12 90/22 91/24 95/6 99/10 100/23 102/13 103/6 103/23 104/7 106/7 112/18 113/12 116/3 117/12 119/3 119/13 120/23 121/7 122/20 123/19 126/7 127/4 129/19 130/17 132/20 134/1 136/19 148/19 148/22 149/4 149/20 149/24 153/4 153/17 153/22 157/7 157/14 157/22 158/17 160/4 161/7 162/3 164/6 167/3 169/18 171/14 172/3 172/18 174/6 175/3 175/22 176/12 177/8 177/25 178/14 179/13 180/1 180/8 180/21 180/25 182/8 182/14 183/7 183/22 185/8 187/6 189/3 190/5 190/13 192/9 193/2 193/10 193/13 193/19 193/24 195/13 196/21 <b>PROFESSOR ANDERSON: [113]</b> 5/2 6/7 6/19 7/22 8/7 8/20 9/16 13/4 13/12 14/17 20/16 25/8 28/4 31/14 33/10 33/13 34/14 35/4 35/11 35/15 38/4 38/10 40/18 42/3 42/7 42/12 44/25 45/20 46/6 49/6 50/22 52/22 53/21 54/18 54/22 56/18 58/9 60/17 62/9 63/6 63/12 63/15 63/19 65/4 66/1 67/10 68/4 69/11 71/11 76/25 77/17 80/10 82/1 87/8 96/1 97/25 98/11 108/10 109/25 119/24 120/25 122/8 125/22 127/3 129/6 131/14 138/5 139/19 141/2 146/10 146/15 146/17 146/22 146/25 147/8	148/5 149/18 154/13 157/19 158/21 160/6 161/12 162/10 164/9 166/11 167/8 169/5 169/23 170/23 171/20 171/25 176/1 177/17 178/12 178/24 179/9 179/19 179/25 180/24 182/12 183/20 184/13 189/7 189/22 190/10 190/19 191/15 191/23 193/6 193/11 193/18 193/21 194/5 <b>PROFESSOR COOKE: [59]</b> 2/20 9/18 10/20 11/25 15/3 15/25 16/6 23/7 28/12 33/22 40/21 49/22 50/12 52/4 52/13 56/11 56/21 59/25 61/12 66/23 69/24 81/1 87/24 88/6 89/22 96/20 98/8 98/12 100/3 121/16 123/11 124/16 128/18 140/17 146/8 146/11 146/16 147/9 147/23 172/10 173/16 174/11 175/8 175/15 176/7 177/1 177/12 177/21 178/3 179/3 180/5 182/19 186/20 188/23 189/18 189/23 190/15 191/17 195/3 <b>PROFESSOR TUDOR-WILLIAMS: [40]</b> 2/5 11/10 12/19 14/1 21/23 35/18 36/5 37/2 41/24 42/10 53/9 54/4 56/9 58/21 65/19 70/9 76/4 84/1 97/6 99/8 110/7 111/25 112/23 121/6 126/9 134/8 143/18 147/11 149/23 150/7 153/8 153/21 168/17 175/13 178/10 181/25 186/4 191/14 192/3 192/13 <b>SIR BRIAN LANGSTAFF: [48]</b> 1/2 1/19 45/3 63/1 63/7 63/17 90/16 90/21 91/21 131/4 132/14 136/23 137/4 138/10 138/16 139/6 139/13 139/17 139/20 139/24 140/4 140/11 140/21 141/3 141/14 141/17 142/7 142/23 143/2 143/5 143/20 144/7 144/15 144/17 144/19 144/21 145/6 146/6 146/20 146/23	147/1 147/10 147/20 148/4 148/16 148/20 195/18 196/22  <b>'80s [2]</b> 74/8 90/4 <b>'88 [1]</b> 191/14 <b>'88/89 [1]</b> 191/14 <b>'90s [5]</b> 14/4 71/2 74/9 90/4 110/13 <b>'96 [1]</b> 104/18 <b>'96/97 [1]</b> 104/18 <b>'97 [1]</b> 113/16 <b>'97/98 [1]</b> 113/16  ... [1] 190/18 <b>0</b> <b>0.03 per cent [1]</b> 191/20 <b>0.3 per cent [1]</b> 10/10 <b>1</b> <b>1 per cent [2]</b> 11/3 93/15 <b>1,000 [3]</b> 121/7 190/24 191/1 <b>1,200 [2]</b> 37/12 37/16 <b>1.00 pm [1]</b> 90/19 <b>1.6 per cent [1]</b> 115/10 <b>10 [3]</b> 11/25 127/21 153/15 <b>10 per cent [1]</b> 150/16 <b>10.30 [4]</b> 1/2 196/20 196/23 196/25 <b>100 [1]</b> 120/9 <b>100 per cent [1]</b> 176/9 <b>100,000 [2]</b> 36/13 122/5 <b>11 [1]</b> 153/15 <b>11.30 [1]</b> 45/7 <b>12 o'clock [2]</b> 45/5 45/6 <b>12.04 pm [1]</b> 45/9 <b>14 per cent [1]</b> 93/15 <b>15 [2]</b> 134/6 153/15 <b>15 per cent [3]</b> 12/19 192/19 192/22 <b>15 years [1]</b> 127/21 <b>16 [1]</b> 83/13 <b>16/15-year [1]</b> 76/4 <b>19 [1]</b> 93/16 <b>1970 [1]</b> 190/9 <b>1980s [3]</b> 40/17 40/19 91/9 <b>1984 [1]</b> 5/7 <b>1986 [1]</b> 3/10 <b>1987 [4]</b> 4/18 93/6 184/2 184/3 <b>1988 [2]</b> 2/10 97/18 <b>1989 [1]</b> 2/13	<b>1990 [1]</b> 5/11 <b>1990s [4]</b> 40/21 94/12 105/21 122/7 <b>1994 [3]</b> 2/13 35/24 186/8 <b>1996 [2]</b> 106/1 192/18 <b>1997 [1]</b> 109/20 <b>1998 [2]</b> 105/21 107/10 <b>2</b> <b>2.00 [1]</b> 90/21 <b>20 [2]</b> 120/11 125/7 <b>20 years [6]</b> 116/11 117/9 117/10 123/11 126/12 134/24 <b>20/25 years [1]</b> 133/7 <b>200 [3]</b> 29/4 32/19 34/17 <b>2000s [5]</b> 106/18 107/10 108/8 113/20 114/6 <b>2004 [3]</b> 5/14 81/14 83/10 <b>2007 [2]</b> 35/25 113/22 <b>2008 [1]</b> 83/10 <b>2015 [2]</b> 115/2 115/12 <b>2015/16 [1]</b> 83/13 <b>2020 [1]</b> 1/1 <b>20s [1]</b> 134/23 <b>21 [1]</b> 19/12 <b>24 [1]</b> 135/5 <b>24 hours [3]</b> 97/20 159/14 160/3 <b>25 [2]</b> 69/3 69/11 <b>25 years [3]</b> 2/23 4/7 14/16 <b>27 February 2020 [1]</b> 1/1 <b>28 [1]</b> 111/19 <b>3</b> <b>3,000 [1]</b> 37/16 <b>3.01 pm [1]</b> 137/2 <b>3.30 [1]</b> 137/1 <b>3.30 pm [1]</b> 137/4 <b>30 [2]</b> 94/22 101/21 <b>30 per cent [1]</b> 76/21 <b>300 [3]</b> 10/8 11/17 29/5 <b>30s [1]</b> 2/18 <b>33 [1]</b> 49/6 <b>4</b> <b>4 per cent [1]</b> 115/9 <b>4.45 pm [1]</b> 196/24 <b>40 [1]</b> 121/25 <b>40 per cent [1]</b> 101/21 <b>40 years [1]</b> 131/10 <b>400 [1]</b> 120/7 <b>5</b> <b>50 [2]</b> 120/10 121/25	<b>500 [1]</b> 32/17 <b>51 [1]</b> 95/7 <b>52 [1]</b> 95/8 <b>6</b> <b>60 [1]</b> 131/24 <b>61 [2]</b> 149/9 154/7 <b>7</b> <b>70 [1]</b> 131/24 <b>8</b> <b>80s [2]</b> 50/4 95/13 <b>85 per cent [1]</b> 193/1 <b>89 [1]</b> 191/14 <b>9</b> <b>90 per cent [1]</b> 118/11 <b>90s [1]</b> 50/4 <b>95/96 per cent [1]</b> 120/23 <b>97 [1]</b> 104/18 <b>97 per [1]</b> 115/19 <b>97 per cent [3]</b> 115/18 117/13 122/22 <b>98 [1]</b> 113/16 <b>A</b> <b>a 13 per cent [1]</b> 135/4 <b>a 20 per cent [1]</b> 135/7 <b>a background [1]</b> 103/1 <b>a balance [4]</b> 24/7 48/17 90/7 172/12 <b>a barrier [2]</b> 88/21 122/14 <b>a batch [1]</b> 10/17 <b>a benefit [1]</b> 110/6 <b>a big [6]</b> 28/18 58/15 81/11 84/13 164/20 166/25 <b>a bigger [1]</b> 125/22 <b>a biological [1]</b> 115/22 <b>a bit [17]</b> 1/23 3/9 12/9 27/12 40/8 51/7 57/24 97/3 108/8 111/3 140/5 142/25 144/3 156/7 160/17 169/3 170/13 <b>a blood [3]</b> 34/2 164/18 176/7 <b>a bloodstream [1]</b> 181/7 <b>a bone [1]</b> 174/2 <b>a booster [1]</b> 107/2 <b>a box [1]</b> 82/15 <b>a brain [1]</b> 47/21 <b>a branded [1]</b> 183/12 <b>a brief [1]</b> 92/5 <b>a cancer [1]</b> 63/21
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<b>A</b>	<b>a damaged [1]</b> 133/9 <b>a day [7]</b> 102/20 106/20 108/16 108/22 111/19 114/3 117/4 <b>a definition [1]</b> 34/18 <b>a desire [1]</b> 155/11 <b>a desperate [1]</b> 94/4 <b>a diagnosis [4]</b> 32/12 45/18 49/9 49/15 <b>a difference [2]</b> 34/21 184/21 <b>a different [8]</b> 8/3 15/25 104/19 111/3 111/24 141/16 165/15 170/15 <b>a differential [1]</b> 21/14 <b>a direct [3]</b> 41/20 87/7 195/3 <b>a disaster [1]</b> 151/21 <b>a disease [1]</b> 121/21 <b>a distance [1]</b> 156/23 <b>a DNA [2]</b> 140/10 142/16 <b>a doctor [3]</b> 2/7 5/5 160/10 <b>a dose [1]</b> 96/17 <b>a double-stranded [1]</b> 92/11 <b>a dreadful [1]</b> 56/5 <b>a drug [4]</b> 66/16 94/7 107/6 125/3 <b>a dual [1]</b> 139/22 <b>a factor [2]</b> 26/14 29/13 <b>a family [3]</b> 2/16 80/3 150/19 <b>a fantastic [2]</b> 106/2 196/7 <b>a far [1]</b> 114/18 <b>a fast [1]</b> 188/11 <b>a feature [1]</b> 187/22 <b>a feeling [1]</b> 51/5 <b>a few [3]</b> 87/15 89/12 93/10 <b>a few per cent [2]</b> 76/23 116/17 <b>a figure [2]</b> 13/9 190/16 <b>a financial [1]</b> 170/18 <b>a fingerprick [1]</b> 159/23 <b>a focus [1]</b> 70/2 <b>a fungus [1]</b> 52/24 <b>a further [1]</b> 15/18 <b>a future [1]</b> 136/7 <b>a general [2]</b> 4/6 4/7 <b>a generalist [1]</b> 4/12 <b>a genetic [1]</b> 30/5 <b>a good [4]</b> 57/12 71/10 151/6 166/12 <b>a GP [1]</b> 170/21 <b>a great [4]</b> 84/6 109/9	133/23 152/6 <b>a greater [2]</b> 82/23 177/11 <b>a group [1]</b> 175/10 <b>a healthy [1]</b> 75/19 <b>a high [10]</b> 31/5 76/3 93/25 94/21 101/11 106/21 106/22 115/6 118/10 148/11 <b>a higher [4]</b> 37/7 118/23 118/24 178/22 <b>a holistic [1]</b> 171/13 <b>a homely [1]</b> 142/24 <b>a horrid [1]</b> 55/12 <b>a hospital [1]</b> 165/14 <b>a huge [7]</b> 55/7 74/19 74/23 78/13 78/18 109/24 150/10 <b>a kitchen [1]</b> 181/5 <b>a lack [2]</b> 78/24 78/24 <b>a large [1]</b> 101/19 <b>a larger [2]</b> 128/15 176/3 <b>a late [1]</b> 187/1 <b>a lesion [1]</b> 70/18 <b>a less [1]</b> 36/10 <b>a lifestyle [1]</b> 109/3 <b>a limitation [1]</b> 50/6 <b>a link [2]</b> 72/2 183/8 <b>a list [1]</b> 156/13 <b>a little [8]</b> 15/10 45/22 69/10 110/10 134/19 156/11 160/1 192/17 <b>a London [2]</b> 54/8 54/12 <b>a long [6]</b> 65/11 76/17 123/22 127/8 129/10 132/6 <b>a look [1]</b> 137/9 <b>a loss [1]</b> 136/18 <b>a lot [33]</b> 12/18 23/9 36/25 37/21 46/25 49/17 49/17 53/24 54/1 68/11 81/16 84/23 95/11 96/6 96/19 96/21 98/8 99/14 107/7 107/23 108/15 108/16 110/20 136/18 147/14 148/12 165/20 169/8 184/25 185/12 189/10 194/18 195/12 <b>a loved [1]</b> 78/17 <b>a low [1]</b> 148/16 <b>a lower [1]</b> 118/25 <b>A lung [1]</b> 65/9 <b>a mammogram [1]</b> 87/11 <b>a marked [1]</b> 64/21 <b>a marker [1]</b> 40/15 <b>a mature [1]</b> 145/5 <b>a medical [2]</b> 56/6 159/12	<b>a medication [2]</b> 96/1 106/14 <b>a memory [1]</b> 20/22 <b>a microinfusion [1]</b> 9/8 <b>a milder [1]</b> 60/12 <b>a minor [1]</b> 169/10 <b>a minority [1]</b> 98/13 <b>a misnomer [1]</b> 3/9 <b>a molecule [1]</b> 29/19 <b>a moment [1]</b> 154/25 <b>a month [1]</b> 169/1 <b>a more [1]</b> 128/18 <b>a mortgage [1]</b> 85/13 <b>a mother [1]</b> 9/4 <b>a much [8]</b> 7/20 26/11 64/11 73/14 122/3 123/10 153/2 169/12 <b>a multidisciplinary [1]</b> 162/14 <b>a neurotropic [1]</b> 22/2 <b>a new [6]</b> 141/13 154/17 159/6 161/3 163/21 169/7 <b>a newer [1]</b> 128/8 <b>a non-specialist [1]</b> 4/13 <b>a non-specific [2]</b> 39/17 39/22 <b>a nucleus [1]</b> 144/11 <b>a number [13]</b> 4/9 9/23 10/17 24/3 31/23 33/8 59/10 86/8 138/2 162/16 167/13 194/7 196/3 <b>a nurse [2]</b> 55/16 167/22 <b>a nursing [1]</b> 56/1 <b>a paediatric [1]</b> 70/11 <b>a paediatrician [2]</b> 2/7 11/12 <b>a panel [1]</b> 1/4 <b>a parent [2]</b> 149/22 181/21 <b>a particular [5]</b> 6/22 48/10 63/4 79/5 82/10 <b>a particularly [2]</b> 46/22 53/1 <b>a pathway [1]</b> 159/1 <b>a patient [2]</b> 173/13 180/20 <b>a perceived [1]</b> 79/13 <b>a period [3]</b> 22/20 163/19 189/16 <b>a person [6]</b> 8/19 9/14 12/21 35/13 46/24 58/12 <b>a person's [2]</b> 26/21 45/24 <b>a pharmacist [1]</b> 167/23 <b>a phone [2]</b> 167/22 169/14	<b>a pill [1]</b> 122/13 <b>A pivotal [1]</b> 114/5 <b>a place [1]</b> 32/18 <b>a point [1]</b> 32/7 <b>a poison [1]</b> 186/13 <b>a pond [1]</b> 75/3 <b>a population [3]</b> 73/13 73/21 81/15 <b>a port-a-cath [1]</b> 58/18 <b>a position [1]</b> 174/6 <b>a positive [1]</b> 160/18 <b>a potential [1]</b> 22/23 <b>a powerful [1]</b> 39/1 <b>a practical [1]</b> 35/8 <b>a prescription [2]</b> 165/9 165/23 <b>a previous [2]</b> 47/15 93/1 <b>a primary [1]</b> 155/7 <b>a problem [3]</b> 48/14 49/2 133/13 <b>a process [6]</b> 154/25 155/4 162/13 195/22 195/24 195/25 <b>a prominent [1]</b> 184/15 <b>a proportion [1]</b> 175/10 <b>a protease [1]</b> 106/18 <b>a public [2]</b> 32/24 195/6 <b>a question [8]</b> 82/17 91/16 126/8 129/20 131/5 132/25 176/15 187/9 <b>a questionnaire-based [2]</b> 82/4 82/14 <b>a raised [1]</b> 19/15 <b>a randomised [1]</b> 103/12 <b>a rash [1]</b> 66/15 <b>a real [1]</b> 189/17 <b>a really [11]</b> 54/5 58/23 59/24 71/1 85/4 107/1 110/4 110/6 110/25 132/24 186/2 <b>a recent [1]</b> 195/10 <b>a regimen [1]</b> 116/13 <b>a relapse [1]</b> 102/13 <b>a relatively [1]</b> 11/9 <b>a reluctance [1]</b> 87/6 <b>a report [3]</b> 6/3 6/10 14/23 <b>a response [1]</b> 17/24 <b>a resting [2]</b> 142/4 187/12 <b>a result [1]</b> 83/7 <b>a role [1]</b> 99/7 <b>a routine [1]</b> 39/10 <b>a rural [1]</b> 191/7 <b>a ruthless [2]</b> 55/24	55/25 <b>a same [1]</b> 86/6 <b>a second [1]</b> 3/24 <b>a section [2]</b> 148/23 173/17 <b>a seizure [1]</b> 69/12 <b>a sense [4]</b> 30/23 33/2 73/7 114/8 <b>A sensitivity [1]</b> 186/5 <b>a series [1]</b> 68/21 <b>a set [1]</b> 20/20 <b>a severe [1]</b> 186/2 <b>a sexual [4]</b> 10/13 86/24 159/19 179/13 <b>a sexually [1]</b> 151/18 <b>a shock [1]</b> 156/7 <b>a short [1]</b> 91/5 <b>A short-lived [1]</b> 19/17 <b>a side [2]</b> 109/2 183/14 <b>a sign [1]</b> 169/12 <b>a signal [1]</b> 39/13 <b>a significant [1]</b> 60/4 <b>a similar [3]</b> 10/5 56/23 80/9 <b>A similar-ish [1]</b> 177/9 <b>a single [3]</b> 14/15 15/7 114/3 <b>a single strand [1]</b> 139/24 <b>a single-stranded [1]</b> 92/12 <b>a small [3]</b> 12/3 30/11 97/4 <b>a smaller [1]</b> 85/19 <b>a smoker [2]</b> 73/25 74/1 <b>a sore [1]</b> 19/15 <b>a sort [6]</b> 51/10 73/24 109/8 136/17 157/5 169/6 <b>a specialist [11]</b> 154/18 154/19 159/7 159/24 160/5 160/7 160/9 160/14 161/14 168/5 169/18 <b>a specific [1]</b> 63/9 <b>a standard [1]</b> 40/24 <b>a state [3]</b> 30/21 31/4 31/9 <b>a steady [1]</b> 25/11 <b>a Stevens-Johnson [1]</b> 186/1 <b>a stone [1]</b> 75/3 <b>a strand [1]</b> 139/11 <b>a study [1]</b> 81/14 <b>a subjective [1]</b> 82/17 <b>a subtype [2]</b> 13/21 13/24 <b>a summary [1]</b> 6/2 <b>a super-infection [1]</b>
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(51) a capsule - a super-infection



<b>A</b>	153/13 174/13	111/19 116/2 116/11	<b>accomplished [1]</b> 2/15	<b>acute [10]</b> 17/8 24/2 24/4 25/2 25/6 28/14 32/2 36/4 60/2 67/13	134/17 135/20 148/1
<b>a super-infection... [1]</b> 15/17	<b>a way [4]</b> 8/13 32/25 48/25 152/3	118/9 119/15 119/17 120/1 120/5 120/11 120/17 121/8 121/9 122/8 125/10 125/13 125/21 126/8 126/11 127/2 128/25 129/4 129/13 129/19 130/5 130/8 130/19 131/6 131/7 132/11 132/23 133/15 133/15 134/8 135/15 136/2 136/2 136/3 136/16 136/17 142/9 142/10 143/8 145/7 149/8 149/9 150/3 150/4 150/7 150/15 150/22 151/5 152/8 153/5 153/7 153/14 153/16 153/18 153/24 154/24 160/8 161/19 162/1 162/13 163/5 163/6 163/12 163/15 163/21 164/23 166/23 167/1 167/2 168/11 168/19 169/11 173/18 174/9 175/14 176/2 176/15 177/3 177/4 179/12 179/14 179/24 181/4 181/8 181/23 182/11 182/15 182/17 183/23 184/13 184/22 185/7 185/10 185/13 187/10 188/23 189/17 190/2 190/6 191/14 192/18 193/1 195/20 195/21	<b>account [2]</b> 33/25 127/1	<b>Acyclovir [1]</b> 40/23	<b>advantages [1]</b> 84/21
<b>a supplemental [2]</b> 6/6 36/23	<b>a website [1]</b> 192/7	125/21 126/8 126/11 127/2 128/25 129/4 129/13 129/19 130/5 130/8 130/19 131/6 131/7 132/11 132/23 133/15 133/15 134/8 135/15 136/2 136/2 136/3 136/16 136/17 142/9 142/10 143/8 145/7 149/8 149/9 150/3 150/4 150/7 150/15 150/22 151/5 152/8 153/5 153/7 153/14 153/16 153/18 153/24 154/24 160/8 161/19 162/1 162/13 163/5 163/6 163/12 163/15 163/21 164/23 166/23 167/1 167/2 168/11 168/19 169/11 173/18 174/9 175/14 176/2 176/15 177/3 177/4 179/12 179/14 179/24 181/4 181/8 181/23 182/11 182/15 182/17 183/23 184/13 184/22 185/7 185/10 185/13 187/10 188/23 189/17 190/2 190/6 191/14 192/18 193/1 195/20 195/21	<b>accounting [1]</b> 28/9	<b>adaptive [1]</b> 24/16	<b>advent [5]</b> 56/14 60/9 89/10 90/11 109/22
<b>a surgical [1]</b> 58/17	<b>a whole [3]</b> 36/19 82/7 83/19	125/21 126/8 126/11 127/2 128/25 129/4 129/13 129/19 130/5 130/8 130/19 131/6 131/7 132/11 132/23 133/15 133/15 134/8 135/15 136/2 136/2 136/3 136/16 136/17 142/9 142/10 143/8 145/7 149/8 149/9 150/3 150/4 150/7 150/15 150/22 151/5 152/8 153/5 153/7 153/14 153/16 153/18 153/24 154/24 160/8 161/19 162/1 162/13 163/5 163/6 163/12 163/15 163/21 164/23 166/23 167/1 167/2 168/11 168/19 169/11 173/18 174/9 175/14 176/2 176/15 177/3 177/4 179/12 179/14 179/24 181/4 181/8 181/23 182/11 182/15 182/17 183/23 184/13 184/22 185/7 185/10 185/13 187/10 188/23 189/17 190/2 190/6 191/14 192/18 193/1 195/20 195/21	<b>accusing [1]</b> 186/16	<b>adverse [2]</b> 114/9 135/17	<b>adverts [1]</b> 74/18
<b>a surprise [1]</b> 155/14	<b>a window [2]</b> 22/10 22/21	127/2 128/25 129/4 129/13 129/19 130/5 130/8 130/19 131/6 131/7 132/11 132/23 133/15 133/15 134/8 135/15 136/2 136/2 136/3 136/16 136/17 142/9 142/10 143/8 145/7 149/8 149/9 150/3 150/4 150/7 150/15 150/22 151/5 152/8 153/5 153/7 153/14 153/16 153/18 153/24 154/24 160/8 161/19 162/1 162/13 163/5 163/6 163/12 163/15 163/21 164/23 166/23 167/1 167/2 168/11 168/19 169/11 173/18 174/9 175/14 176/2 176/15 177/3 177/4 179/12 179/14 179/24 181/4 181/8 181/23 182/11 182/15 182/17 183/23 184/13 184/22 185/7 185/10 185/13 187/10 188/23 189/17 190/2 190/6 191/14 192/18 193/1 195/20 195/21	<b>achieving [1]</b> 94/9	<b>add [26]</b> 7/24 11/11 12/11 15/4 21/24 28/5 51/17 51/19 53/22 55/13 60/17 66/9 66/11 75/2 75/13 86/5 90/15 91/22 95/19 96/2 108/11 109/11 113/7 124/25 171/4 182/1	<b>advice [6]</b> 153/24 156/5 161/19 195/20 195/22 196/18
<b>a survey [1]</b> 76/19	<b>a woman [3]</b> 43/9 87/10 121/4	127/2 128/25 129/4 129/13 129/19 130/5 130/8 130/19 131/6 131/7 132/11 132/23 133/15 133/15 134/8 135/15 136/2 136/2 136/3 136/16 136/17 142/9 142/10 143/8 145/7 149/8 149/9 150/3 150/4 150/7 150/15 150/22 151/5 152/8 153/5 153/7 153/14 153/16 153/18 153/24 154/24 160/8 161/19 162/1 162/13 163/5 163/6 163/12 163/15 163/21 164/23 166/23 167/1 167/2 168/11 168/19 169/11 173/18 174/9 175/14 176/2 176/15 177/3 177/4 179/12 179/14 179/24 181/4 181/8 181/23 182/11 182/15 182/17 183/23 184/13 184/22 185/7 185/10 185/13 187/10 188/23 189/17 190/2 190/6 191/14 192/18 193/1 195/20 195/21	<b>acid [2]</b> 143/16 148/15	<b>addition [2]</b> 32/13 183/4	<b>advised [1]</b> 108/23
<b>a suspension [1]</b> 112/6	<b>a work-up [1]</b> 159/11	127/2 128/25 129/4 129/13 129/19 130/5 130/8 130/19 131/6 131/7 132/11 132/23 133/15 133/15 134/8 135/15 136/2 136/2 136/3 136/16 136/17 142/9 142/10 143/8 145/7 149/8 149/9 150/3 150/4 150/7 150/15 150/22 151/5 152/8 153/5 153/7 153/14 153/16 153/18 153/24 154/24 160/8 161/19 162/1 162/13 163/5 163/6 163/12 163/15 163/21 164/23 166/23 167/1 167/2 168/11 168/19 169/11 173/18 174/9 175/14 176/2 176/15 177/3 177/4 179/12 179/14 179/24 181/4 181/8 181/23 182/11 182/15 182/17 183/23 184/13 184/22 185/7 185/10 185/13 187/10 188/23 189/17 190/2 190/6 191/14 192/18 193/1 195/20 195/21	<b>acids [2]</b> 142/17 142/19	<b>added [2]</b> 12/13 43/2	<b>advisers [1]</b> 163/9
<b>a table [2]</b> 95/9 95/13	<b>a year [1]</b> 168/25	127/2 128/25 129/4 129/13 129/19 130/5 130/8 130/19 131/6 131/7 132/11 132/23 133/15 133/15 134/8 135/15 136/2 136/2 136/3 136/16 136/17 142/9 142/10 143/8 145/7 149/8 149/9 150/3 150/4 150/7 150/15 150/22 151/5 152/8 153/5 153/7 153/14 153/16 153/18 153/24 154/24 160/8 161/19 162/1 162/13 163/5 163/6 163/12 163/15 163/21 164/23 166/23 167/1 167/2 168/11 168/19 169/11 173/18 174/9 175/14 176/2 176/15 177/3 177/4 179/12 179/14 179/24 181/4 181/8 181/23 182/11 182/15 182/17 183/23 184/13 184/22 185/7 185/10 185/13 187/10 188/23 189/17 190/2 190/6 191/14 192/18 193/1 195/20 195/21	<b>acknowledge [2]</b> 79/16 115/24	<b>adding [1]</b> 61/10	<b>advising [1]</b> 195/20
<b>a team [2]</b> 160/22 162/17	<b>a younger [1]</b> 134/13	127/2 128/25 129/4 129/13 129/19 130/5 130/8 130/19 131/6 131/7 132/11 132/23 133/15 133/15 134/8 135/15 136/2 136/2 136/3 136/16 136/17 142/9 142/10 143/8 145/7 149/8 149/9 150/3 150/4 150/7 150/15 150/22 151/5 152/8 153/5 153/7 153/14 153/16 153/18 153/24 154/24 160/8 161/19 162/1 162/13 163/5 163/6 163/12 163/15 163/21 164/23 166/23 167/1 167/2 168/11 168/19 169/11 173/18 174/9 175/14 176/2 176/15 177/3 177/4 179/12 179/14 179/24 181/4 181/8 181/23 182/11 182/15 182/17 183/23 184/13 184/22 185/7 185/10 185/13 187/10 188/23 189/17 190/2 190/6 191/14 192/18 193/1 195/20 195/21	<b>acknowledged [1]</b> 196/10	<b>address [1]</b> 84/1	<b>aetiology [1]</b> 63/22
<b>a terminal [1]</b> 78/9	<b>a zip [2]</b> 143/23 143/24	127/2 128/25 129/4 129/13 129/19 130/5 130/8 130/19 131/6 131/7 132/11 132/23 133/15 133/15 134/8 135/15 136/2 136/2 136/3 136/16 136/17 142/9 142/10 143/8 145/7 149/8 149/9 150/3 150/4 150/7 150/15 150/22 151/5 152/8 153/5 153/7 153/14 153/16 153/18 153/24 154/24 160/8 161/19 162/1 162/13 163/5 163/6 163/12 163/15 163/21 164/23 166/23 167/1 167/2 168/11 168/19 169/11 173/18 174/9 175/14 176/2 176/15 177/3 177/4 179/12 179/14 179/24 181/4 181/8 181/23 182/11 182/15 182/17 183/23 184/13 184/22 185/7 185/10 185/13 187/10 188/23 189/17 190/2 190/6 191/14 192/18 193/1 195/20 195/21	<b>acquire [1]</b> 21/25	<b>addressed [3]</b> 172/9 172/11 180/6	<b>AF [1]</b> 128/5
<b>a terrible [1]</b> 59/14	<b>ABC [1]</b> 36/3	127/2 128/25 129/4 129/13 129/19 130/5 130/8 130/19 131/6 131/7 132/11 132/23 133/15 133/15 134/8 135/15 136/2 136/2 136/3 136/16 136/17 142/9 142/10 143/8 145/7 149/8 149/9 150/3 150/4 150/7 150/15 150/22 151/5 152/8 153/5 153/7 153/14 153/16 153/18 153/24 154/24 160/8 161/19 162/1 162/13 163/5 163/6 163/12 163/15 163/21 164/23 166/23 167/1 167/2 168/11 168/19 169/11 173/18 174/9 175/14 176/2 176/15 177/3 177/4 179/12 179/14 179/24 181/4 181/8 181/23 182/11 182/15 182/17 183/23 184/13 184/22 185/7 185/10 185/13 187/10 188/23 189/17 190/2 190/6 191/14 192/18 193/1 195/20 195/21	<b>acquired [2]</b> 134/22 180/19	<b>addition [2]</b> 32/13 183/4	<b>affect [11]</b> 21/17 40/1 57/11 60/2 61/17 65/10 67/3 99/24 169/22 183/14 188/22
<b>a test [6]</b> 22/21 23/1 25/22 91/13 155/7 159/22	<b>abdominal [2]</b> 55/6 95/16	127/2 128/25 129/4 129/13 129/19 130/5 130/8 130/19 131/6 131/7 132/11 132/23 133/15 133/15 134/8 135/15 136/2 136/2 136/3 136/16 136/17 142/9 142/10 143/8 145/7 149/8 149/9 150/3 150/4 150/7 150/15 150/22 151/5 152/8 153/5 153/7 153/14 153/16 153/18 153/24 154/24 160/8 161/19 162/1 162/13 163/5 163/6 163/12 163/15 163/21 164/23 166/23 167/1 167/2 168/11 168/19 169/11 173/18 174/9 175/14 176/2 176/15 177/3 177/4 179/12 179/14 179/24 181/4 181/8 181/23 182/11 182/15 182/17 183/23 184/13 184/22 185/7 185/10 185/13 187/10 188/23 189/17 190/2 190/6 191/14 192/18 193/1 195/20 195/21	<b>acquires [1]</b> 17/16	<b>advised [1]</b> 108/23	<b>affected [8]</b> 2/12 43/3 64/2 65/1 76/9 87/5 178/8 188/4
<b>a therapeutic [1]</b> 108/14	<b>ability [6]</b> 10/4 32/14 51/14 123/4 136/7 171/1	127/2 128/25 129/4 129/13 129/19 130/5 130/8 130/19 131/6 131/7 132/11 132/23 133/15 133/15 134/8 135/15 136/2 136/2 136/3 136/16 136/17 142/9 142/10 143/8 145/7 149/8 149/9 150/3 150/4 150/7 150/15 150/22 151/5 152/8 153/5 153/7 153/14 153/16 153/18 153/24 154/24 160/8 161/19 162/1 162/13 163/5 163/6 163/12 163/15 163/21 164/23 166/23 167/1 167/2 168/11 168/19 169/11 173/18 174/9 175/14 176/2 176/15 177/3 177/4 179/12 179/14 179/24 181/4 181/8 181/23 182/11 182/15 182/17 183/23 184/13 184/22 185/7 185/10 185/13 187/10 188/23 189/17 190/2 190/6 191/14 192/18 193/1 195/20 195/21	<b>acquiring [1]</b> 16/22	<b>addition [2]</b> 32/13 183/4	<b>affecting [3]</b> 7/3 65/20 65/21
<b>a thing [2]</b> 55/1 58/18	<b>able [48]</b> 29/16 29/20 46/20 49/1 62/24 63/11 65/16 68/16 77/8 77/16 79/19 92/14 93/4 93/19 94/8 94/16 98/3 103/22 105/4 105/10 106/2 106/4 113/10 114/2 114/24 118/7 123/14 125/17 128/2 134/8 135/19 141/13 145/16 152/21 155/4 157/15 159/20 159/22 160/15 160/21 161/5 167/20 169/15 170/3 172/23 190/9 193/9 194/13	127/2 128/25 129/4 129/13 129/19 130/5 130/8 130/19 131/6 131/7 132/11 132/23 133/15 133/15 134/8 135/15 136/2 136/2 136/3 136/16 136/17 142/9 142/10 143/8 145/7 149/8 149/9 150/3 150/4 150/7 150/15 150/22 151/5 152/8 153/5 153/7 153/14 153/16 153/18 153/24 154/24 160/8 161/19 162/1 162/13 163/5 163/6 163/12 163/15 163/21 164/23 166/23 167/1 167/2 168/11 168/19 169/11 173/18 174/9 175/14 176/2 176/15 177/3 177/4 179/12 179/14 179/24 181/4 181/8 181/23 182/11 182/15 182/17 183/23 184/13 184/22 185/7 185/10 185/13 187/10 188/23 189/17 190/2 190/6 191/14 192/18 193/1 195/20 195/21	<b>across [11]</b> 5/14 7/12 7/17 11/2 47/12 86/20 181/12 188/16 190/22 191/2 192/9	<b>addition [2]</b> 32/13 183/4	<b>affects [3]</b> 6/23 18/23 188/20
<b>a third [1]</b> 139/3	63/11 65/16 68/16 77/8 77/16 79/19 92/14 93/4 93/19 94/8 94/16 98/3 103/22 105/4 105/10 106/2 106/4 113/10 114/2 114/24 118/7 123/14 125/17 128/2 134/8 135/19 141/13 145/16 152/21 155/4 157/15 159/20 159/22 160/15 160/21 161/5 167/20 169/15 170/3 172/23 190/9 193/9 194/13	127/2 128/25 129/4 129/13 129/19 130/5 130/8 130/19 131/6 131/7 132/11 132/23 133/15 133/15 134/8 135/15 136/2 136/2 136/3 136/16 136/17 142/9 142/10 143/8 145/7 149/8 149/9 150/3 150/4 150/7 150/15 150/22 151/5 152/8 153/5 153/7 153/14 153/16 153/18 153/24 154/24 160/8 161/19 162/1 162/13 163/5 163/6 163/12 163/15 163/21 164/23 166/23 167/1 167/2 168/11 168/19 169/11 173/18 174/9 175/14 176/2 176/15 177/3 177/4 179/12 179/14 179/24 181/4 181/8 181/23 182/11 182/15 182/17 183/23 184/13 184/22 185/7 185/10 185/13 187/10 188/23 189/17 190/2 190/6 191/14 192/18 193/1 195/20 195/21	<b>acted [1]</b> 104/19	<b>addition [2]</b> 32/13 183/4	<b>affirmed [4]</b> 1/14 1/16 1/18 1/19
<b>a tick [1]</b> 82/17	63/11 65/16 68/16 77/8 77/16 79/19 92/14 93/4 93/19 94/8 94/16 98/3 103/22 105/4 105/10 106/2 106/4 113/10 114/2 114/24 118/7 123/14 125/17 128/2 134/8 135/19 141/13 145/16 152/21 155/4 157/15 159/20 159/22 160/15 160/21 161/5 167/20 169/15 170/3 172/23 190/9 193/9 194/13	127/2 128/25 129/4 129/13 129/19 130/5 130/8 130/19 131/6 131/7 132/11 132/23 133/15 133/15 134/8 135/15 136/2 136/2 136/3 136/16 136/17 142/9 142/10 143/8 145/7 149/8 149/9 150/3 150/4 150/7 150/15 150/22 151/5 152/8 153/5 153/7 153/14 153/16 153/18 153/24 154/24 160/8 161/19 162/1 162/13 163/5 163/6 163/12 163/15 163/21 164/23 166/23 167/1 167/2 168/11 168/19 169/11 173/18 174/9 175/14 176/2 176/15 177/3 177/4 179/12 179/14 179/24 181/4 181/8 181/23 182/11 182/15 182/17 183/23 184/13 184/22 185/7 185/10 185/13 187/10 188/23 189/17 190/2 190/6 191/14 192/18 193/1 195/20 195/21	<b>acting [1]</b> 146/2	<b>addition [2]</b> 32/13 183/4	<b>after [25]</b> 2/8 14/16 17/22 18/8 18/9 19/6 22/25 23/6 24/6 36/13 58/19 65/23 81/3 90/14 93/10 97/18 109/18 110/2 127/16 154/12 155/3 156/15 159/2 180/15 195/16
<b>a time [2]</b> 103/20 189/9	63/11 65/16 68/16 77/8 77/16				



<b>A</b>	142/10 144/5 144/5 145/16 145/16 147/22 147/22 148/21 151/9 152/14 155/4 157/5 164/10 164/12 166/4 171/6 172/1 179/11 183/19 189/5 190/7 192/8 192/23 <b>allergies [1]</b> 66/13 <b>alleviate [1]</b> 99/20 <b>allocated [1]</b> 163/21 <b>allow [7]</b> 23/14 112/7 144/10 144/11 145/20 150/23 162/3 <b>allowing [1]</b> 32/25 <b>allows [11]</b> 32/11 34/18 118/14 118/15 118/19 118/19 118/20 139/4 140/10 141/20 141/21 <b>alluded [15]</b> 8/9 24/11 26/15 43/11 50/23 80/13 83/11 117/22 118/16 120/17 122/10 129/8 132/11 161/15 173/8 <b>alluding [2]</b> 82/2 82/10 <b>almost [4]</b> 75/7 79/11 146/22 155/10 <b>alone [1]</b> 80/3 <b>along [10]</b> 2/9 31/20 41/20 63/14 101/2 101/15 102/25 104/10 104/18 128/24 <b>alongside [1]</b> 88/18 <b>already [15]</b> 1/5 31/22 32/18 37/17 45/12 50/23 61/23 93/8 103/3 111/9 134/15 134/23 152/21 159/10 183/2 <b>also [62]</b> 3/16 4/1 4/9 5/12 5/21 9/2 9/3 9/5 10/2 10/16 21/9 24/11 27/8 28/6 30/9 33/1 34/15 53/22 54/5 57/17 58/22 68/23 68/25 69/8 69/20 72/8 75/23 78/6 80/11 84/2 86/16 96/15 103/1 104/25 107/23 107/23 113/8 113/24 118/12 119/23 119/25 120/17 128/7 129/7 135/6 136/17 156/6 156/22 158/12 160/25 161/1 164/3 164/17 165/16 169/6 169/16 170/11 171/1 174/14 177/12 177/18 182/20 <b>alter [2]</b> 87/21 87/22 <b>alteration [1]</b> 87/24	<b>alternative [1]</b> 164/16 <b>alters [1]</b> 176/17 <b>although [17]</b> 12/15 20/17 33/14 34/22 35/6 43/25 65/9 73/16 99/6 107/6 111/5 124/3 164/2 165/2 175/9 189/1 190/4 <b>always [8]</b> 59/12 60/10 76/7 85/21 90/3 146/22 158/16 168/14 <b>am [31]</b> 1/2 1/22 2/6 2/21 2/24 4/18 36/23 38/8 44/6 45/7 47/22 63/8 69/3 71/3 90/13 90/21 90/23 91/7 91/10 127/2 132/14 148/21 149/7 160/8 160/8 161/2 162/24 165/6 187/8 193/25 196/25 <b>amazing [1]</b> 135/18 <b>ambition [1]</b> 189/21 <b>ambitious [1]</b> 169/20 <b>America [2]</b> 2/13 31/18 <b>American [1]</b> 34/21 <b>Amitryptolene [1]</b> 43/13 <b>amongst [3]</b> 52/1 80/15 82/21 <b>amount [10]</b> 9/15 18/10 55/8 68/2 71/23 105/7 106/11 111/20 152/13 176/6 <b>amounts [2]</b> 117/16 168/3 <b>anaemia [5]</b> 95/15 96/5 96/11 184/12 184/13 <b>anaemic [1]</b> 185/21 <b>anal [1]</b> 8/24 <b>analogues [5]</b> 104/23 104/24 107/11 113/24 142/18 <b>analogy [2]</b> 76/5 142/24 <b>analysis [2]</b> 14/1 192/21 <b>ANDERSON [5]</b> 1/19 5/2 81/13 87/3 158/20 <b>another [20]</b> 28/14 29/22 29/24 54/23 59/20 61/10 61/13 66/8 107/24 125/23 136/19 141/7 165/18 173/1 173/24 180/12 182/9 183/23 185/9 196/8 <b>answer [16]</b> 6/4 6/18 6/19 83/8 123/25 127/10 131/16 132/25 146/9 173/20 176/1	176/18 179/25 190/9 195/10 196/16 <b>answered [2]</b> 156/14 156/21 <b>anti [32]</b> 5/1 11/15 17/23 35/17 39/19 41/19 43/8 43/10 49/11 54/3 61/23 66/4 67/22 71/13 74/10 80/24 90/11 104/11 109/22 115/4 116/9 117/14 118/5 122/21 127/6 165/19 166/11 166/14 166/19 167/18 168/20 185/24 <b>anti-fungal [2]</b> 54/3 61/23 <b>anti-nausea [1]</b> 67/22 <b>anti-retroviral [27]</b> 5/1 11/15 35/17 39/19 41/19 43/8 43/10 49/11 66/4 74/10 80/24 90/11 104/11 109/22 115/4 116/9 117/14 118/5 122/21 127/6 165/19 166/11 166/14 166/19 167/18 168/20 185/24 <b>anti-retrovirals [1]</b> 71/13 <b>antibiotic [1]</b> 66/6 <b>antibiotics [1]</b> 41/1 <b>antibodies [4]</b> 14/10 17/25 22/18 23/24 <b>antibody [6]</b> 18/6 23/22 23/23 25/22 175/11 184/6 <b>anvitosis [2]</b> 42/14 42/14 <b>anxiety [2]</b> 124/6 125/12 <b>anxious [1]</b> 158/10 <b>any [43]</b> 17/18 18/21 24/14 26/22 27/18 28/4 50/12 50/20 54/14 55/2 57/3 62/23 64/24 91/19 101/12 112/7 112/8 115/11 116/20 116/21 127/25 128/1 134/7 136/22 148/1 148/18 151/24 153/6 155/21 155/22 156/9 157/25 158/23 159/8 169/21 170/22 178/14 182/16 183/25 184/7 193/3 193/15 196/17 <b>anybody [4]</b> 51/19 85/2 152/24 170/18 <b>anyone [7]</b> 6/18 12/11 70/5 90/15 95/19 95/19 130/18 <b>anything [13]</b> 9/11	12/11 43/16 48/12 51/18 55/13 60/16 90/15 95/20 96/12 130/18 135/22 195/16 <b>anyway [1]</b> 144/2 <b>anywhere [1]</b> 144/5 <b>apart [1]</b> 144/23 <b>apology [1]</b> 2/1 <b>apparent [5]</b> 71/7 93/24 101/4 101/7 105/9 <b>appear [3]</b> 47/6 62/16 175/11 <b>appearances [1]</b> 100/3 <b>appearing [1]</b> 50/12 <b>appears [1]</b> 7/10 <b>appetite [1]</b> 51/14 <b>Applause [1]</b> 131/14 <b>appointed [1]</b> 5/10 <b>appointment [5]</b> 155/18 156/4 158/8 163/18 169/16 <b>appointments [4]</b> 84/23 164/23 168/11 169/2 <b>approach [2]</b> 91/6 174/18 <b>approaches [3]</b> 64/5 64/6 154/15 <b>appropriate [6]</b> 8/13 98/16 155/12 156/11 159/2 170/19 <b>arbitrary [2]</b> 25/11 25/25 <b>archived [2]</b> 126/20 126/25 <b>are [376]</b> <b>area [5]</b> 18/11 46/25 60/14 79/5 191/5 <b>areas [7]</b> 6/13 8/4 79/23 129/18 174/3 190/22 190/24 <b>aren't [5]</b> 27/18 50/11 158/16 179/5 194/15 <b>argument [1]</b> 97/8 <b>arise [4]</b> 81/25 148/19 162/4 178/21 <b>arises [2]</b> 131/6 157/8 <b>arm [2]</b> 58/16 93/16 <b>aromas [1]</b> 59/9 <b>arose [1]</b> 195/16 <b>around [20]</b> 3/4 15/10 33/17 42/19 47/3 55/2 55/10 69/19 89/16 113/9 115/25 116/25 132/2 132/13 179/15 181/20 184/2 185/1 185/7 192/24 <b>arrange [1]</b> 155/17 <b>array [3]</b> 82/7 82/21 83/19 <b>arrow [1]</b> 139/8	<b>ART [1]</b> 74/10 <b>as [207]</b> <b>aside [1]</b> 142/25 <b>ask [22]</b> 6/17 31/11 37/24 61/8 85/3 88/1 91/7 91/10 95/7 99/11 126/8 129/21 131/5 134/2 148/21 149/8 149/16 154/9 180/23 182/9 189/12 189/13 <b>asked [13]</b> 82/10 83/6 85/15 91/4 126/11 127/23 129/21 143/8 175/14 176/15 176/19 180/9 192/1 <b>asking [7]</b> 1/22 6/16 20/19 82/6 87/13 90/13 112/12 <b>aspect [2]</b> 125/22 125/23 <b>aspects [2]</b> 3/4 72/5 <b>assay [2]</b> 119/8 120/10 <b>assays [8]</b> 22/18 23/2 23/3 23/7 24/1 105/6 120/3 120/4 <b>assembled [1]</b> 141/7 <b>assess [1]</b> 160/15 <b>assessment [1]</b> 167/15 <b>assimilate [1]</b> 151/23 <b>associated [26]</b> 50/17 70/13 71/6 73/17 74/11 74/19 75/12 99/3 101/22 107/3 107/5 111/14 117/6 119/3 128/6 128/7 128/17 130/11 130/12 130/14 131/23 132/9 151/14 185/23 194/21 195/1 <b>association [4]</b> 4/24 5/22 159/16 187/2 <b>assume [1]</b> 167/10 <b>assuming [1]</b> 168/16 <b>assuring [1]</b> 156/1 <b>asymptomatic [1]</b> 33/22 <b>atazanavir [1]</b> 107/16 <b>attach [1]</b> 29/20 <b>attached [3]</b> 74/23 78/12 79/17 <b>attacks [1]</b> 24/11 <b>attempted [1]</b> 135/8 <b>attempts [1]</b> 83/20 <b>attend [1]</b> 170/22 <b>attending [2]</b> 82/5 115/17 <b>attention [6]</b> 27/9 65/14 69/1 69/7 69/15 69/23 <b>attenuated [1]</b> 130/13 <b>attenuation [1]</b>
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<b>A</b>	47/22 49/8 49/21 54/10 55/16 57/11 64/4 68/10 68/11 68/13 71/2 74/17 83/22 84/3 84/11 87/18 102/7 106/9 110/9 110/13 119/12 124/1 126/9 126/19 129/3 136/25 150/8 155/14 156/8 156/13 162/12 165/1 165/16 167/7 175/2 186/8 188/14 191/12 191/13 191/24 192/23 <b>background [7]</b> 39/8 40/6 40/13 40/16 72/18 75/7 103/1 <b>bacteria [1]</b> 48/10 <b>bad [5]</b> 44/8 61/21 184/25 187/25 187/25 <b>bags [1]</b> 111/16 <b>balance [5]</b> 24/7 48/17 89/24 90/7 172/12 <b>balanced [3]</b> 10/2 90/8 172/14 <b>Barr [1]</b> 40/3 <b>barrier [7]</b> 88/21 122/14 148/11 148/16 170/2 170/18 188/17 <b>barriers [1]</b> 170/10 <b>Bartholomew's [1]</b> 5/11 <b>Barts [1]</b> 5/12 <b>based [10]</b> 2/24 4/2 14/9 18/2 62/20 82/4 82/14 93/17 103/11 119/8 <b>baseline [3]</b> 101/7 163/25 167/15 <b>basic [2]</b> 155/20 157/2 <b>basically [8]</b> 13/22 31/4 92/7 92/17 94/15 104/14 119/8 138/9 <b>basis [2]</b> 167/7 170/4 <b>batch [4]</b> 9/25 10/17 13/4 16/4 <b>be [309]</b> <b>be different [1]</b> 17/1 <b>be on [1]</b> 58/14 <b>be only [1]</b> 116/17 <b>be two [1]</b> 146/13 <b>bearing [1]</b> 30/2 <b>became [24]</b> 43/3 43/3 43/4 66/5 71/7 71/8 93/24 94/11 94/25 95/1 101/3 101/7 104/11 105/8 105/9 107/15 108/9 113/21 113/24 114/4 114/23 114/25 182/3 187/4	<b>because [107]</b> 3/20 5/17 8/2 8/9 13/15 18/7 18/22 19/24 20/9 23/6 25/7 26/9 29/2 30/18 31/5 34/24 37/1 37/4 37/20 38/12 38/19 39/1 41/15 43/22 49/10 54/8 55/19 55/20 58/10 58/15 59/9 59/9 63/9 65/3 65/6 71/18 72/13 77/11 78/7 78/23 79/4 83/2 83/16 83/21 84/23 85/11 85/16 86/24 87/12 91/15 93/10 97/20 97/24 99/12 99/21 100/17 101/9 102/2 102/23 107/5 111/15 111/23 112/16 113/11 114/6 114/17 116/25 118/25 121/15 122/15 124/13 126/5 126/11 129/25 130/5 133/1 133/14 135/16 140/14 146/2 151/12 153/6 156/16 156/19 158/5 158/13 160/9 160/16 161/14 166/13 166/23 168/21 173/11 174/21 176/24 178/23 181/10 184/13 184/16 185/7 185/18 186/12 186/23 191/5 191/21 193/25 194/22 <b>become [16]</b> 8/19 9/14 10/19 11/24 14/21 18/7 30/8 44/15 46/9 49/1 85/22 101/8 114/21 142/4 169/11 185/17 <b>becomes [4]</b> 7/1 47/1 47/19 60/24 <b>becoming [3]</b> 77/23 181/21 185/21 <b>bed [1]</b> 165/14 <b>been [98]</b> 2/2 2/4 2/11 2/22 3/19 3/24 4/7 4/8 4/10 4/17 4/18 4/20 4/24 4/25 5/16 5/19 5/21 5/25 9/18 10/1 11/7 11/22 32/2 33/20 40/23 41/3 46/12 46/15 46/20 47/2 54/4 62/11 64/21 65/1 66/22 67/13 69/16 71/15 75/18 78/8 79/7 81/18 82/25 83/3 83/9 85/7 86/11 87/11 90/10 91/6 91/8 93/1 93/2 98/15 99/6 116/24 117/9 119/5 119/14 119/15 121/25 123/1 123/21 125/6	125/9 126/12 126/24 128/8 128/12 132/6 132/21 133/6 134/21 136/8 136/14 136/15 136/17 140/25 147/7 147/24 148/1 149/17 149/21 154/2 168/15 169/11 171/15 173/2 174/23 180/18 183/24 185/23 191/11 192/1 192/12 193/5 195/24 196/3 <b>before [36]</b> 1/3 22/14 33/6 35/14 39/18 44/10 47/8 48/19 59/22 66/4 78/5 87/14 90/10 90/23 90/24 95/1 95/10 102/21 107/14 109/7 111/18 116/24 124/24 130/19 133/2 137/5 151/24 153/19 161/15 162/20 165/22 167/25 169/9 177/4 180/3 181/22 <b>begin [1]</b> 191/13 <b>begin [2]</b> 71/17 155/8 <b>beginning [4]</b> 96/4 97/17 162/12 162/22 <b>behaviour [1]</b> 81/20 <b>behavioural [1]</b> 72/5 <b>behind [4]</b> 23/18 112/24 193/25 195/15 <b>being [48]</b> 8/11 9/22 11/21 14/12 23/25 33/21 37/9 51/5 58/8 63/11 67/18 67/18 74/22 77/16 84/7 85/15 86/18 89/17 91/11 93/3 94/8 97/25 100/22 101/5 106/4 114/24 118/16 120/18 125/21 128/2 129/12 147/6 147/19 154/11 156/23 158/24 165/2 165/4 174/8 184/1 184/22 185/4 185/10 186/11 186/18 187/13 194/17 195/3 <b>beings [1]</b> 7/4 <b>believe [2]</b> 4/11 150/3 <b>believed [1]</b> 14/12 <b>bells [1]</b> 40/12 <b>belongs [2]</b> 6/22 56/23 <b>below [3]</b> 32/19 34/17 36/17 <b>benefit [10]</b> 93/24 104/22 110/6 110/7 115/7 129/4 184/8 185/16 189/24 189/25 <b>benefits [2]</b> 98/7 105/15 <b>Berlin [1]</b> 173/13	<b>besides [1]</b> 196/13 <b>best [13]</b> 56/12 84/4 98/6 126/15 149/2 152/5 161/16 161/21 162/2 162/3 164/25 165/1 186/18 <b>better [37]</b> 24/24 26/12 35/10 45/5 58/3 60/1 61/19 72/17 75/14 81/6 100/23 103/13 103/18 103/22 104/23 106/15 107/16 108/10 114/10 114/18 114/18 114/24 116/15 116/22 118/24 123/9 123/10 124/4 124/11 124/14 124/15 134/4 134/13 151/23 152/6 153/2 189/14 <b>between [17]</b> 6/14 7/15 24/8 26/2 34/21 72/2 107/9 134/5 172/13 174/10 174/14 176/13 176/16 183/8 187/2 188/25 194/14 <b>beyond [1]</b> 36/15 <b>BHIVA [1]</b> 163/10 <b>big [18]</b> 15/22 28/18 58/15 58/23 71/1 79/17 81/11 84/13 85/5 95/1 137/18 152/10 153/12 156/17 163/16 164/20 166/25 195/11 <b>bigger [2]</b> 125/22 169/12 <b>bind [2]</b> 138/24 139/1 <b>binding [2]</b> 92/18 138/20 <b>binds [1]</b> 143/12 <b>biological [2]</b> 115/22 117/18 <b>biopsies [1]</b> 70/25 <b>bit [30]</b> 1/23 3/9 12/9 15/10 25/10 27/12 33/25 40/8 51/7 57/24 65/12 69/10 97/3 100/23 108/8 111/3 129/4 134/19 140/5 140/6 140/24 142/25 144/3 156/7 160/1 160/17 168/13 169/3 170/13 192/17 <b>bits [2]</b> 142/9 148/9 <b>black [1]</b> 5/19 <b>bleeding [9]</b> 65/2 113/9 113/11 148/25 178/16 181/17 181/18 181/18 196/21 <b>blind [2]</b> 59/3 59/23 <b>blinded [1]</b> 59/10 <b>blindness [1]</b> 53/15 <b>block [2]</b> 148/7 148/8	<b>blocked [1]</b> 147/18 <b>blocks [2]</b> 142/17 194/22 <b>blood [69]</b> 9/2 9/2 9/8 9/8 9/9 9/9 10/11 10/18 10/18 13/4 16/24 34/2 39/7 39/10 62/7 62/7 65/2 95/15 96/7 96/10 105/8 105/17 105/18 105/20 108/14 114/15 119/9 120/6 120/19 121/8 121/13 133/20 148/24 150/13 150/15 156/25 164/18 166/1 172/8 172/9 175/23 175/24 176/4 176/7 178/2 178/2 178/19 178/19 178/20 178/24 179/6 179/15 179/16 179/18 180/11 180/13 180/13 181/5 181/13 181/24 182/11 188/17 190/8 190/12 190/13 190/14 193/4 193/5 193/5 <b>blood-to-blood [1]</b> 180/13 <b>bloodstream [2]</b> 52/8 181/7 <b>blotches [1]</b> 62/21 <b>board [2]</b> 86/15 86/21 <b>bodies [1]</b> 62/22 <b>body [10]</b> 11/19 13/16 18/13 24/12 40/15 57/17 174/16 175/20 180/24 181/13 <b>body's [1]</b> 71/22 <b>bone [6]</b> 72/1 72/11 124/8 128/7 173/4 174/2 <b>boost [2]</b> 108/17 108/20 <b>booster [1]</b> 107/2 <b>born [1]</b> 58/25 <b>both [21]</b> 13/7 13/10 14/13 14/16 15/3 15/6 23/12 60/2 61/10 63/17 67/2 88/6 88/22 89/11 91/1 113/19 135/1 152/16 174/1 177/8 194/10 <b>bottom [1]</b> 160/2 <b>bought [1]</b> 110/22 <b>bound [1]</b> 143/16 <b>box [3]</b> 82/15 82/18 109/9 <b>brain [17]</b> 21/3 22/3 47/21 48/1 53/6 60/2 64/1 64/2 67/3 70/2 70/3 70/7 70/18 188/15 188/17 188/19 188/20 <b>branded [1]</b> 183/12
<b>B</b>	<b>babies [3]</b> 12/23 97/11 192/11 <b>baby [1]</b> 12/16 <b>back [52]</b> 1/7 14/4 14/23 28/16 30/17 33/13 33/21 37/19 44/3 46/20 47/20				



<b>B</b>	138/11 138/20 141/7 142/14 145/12 147/17 149/12 160/7 168/6 173/11 174/8 183/3 185/10 188/4 188/5 192/17 192/20 194/17	120/12 122/14 123/16 123/25 124/6 124/25 125/1 126/4 126/9 127/14 128/5 128/7 131/18 133/3 134/2 135/25 135/25 137/7 137/9 137/14 138/7 138/11 138/21 140/13 143/16 143/24 145/18 148/8 149/5 149/16 150/7 152/25 153/23 154/7 154/9 155/13 156/18 156/20 156/20 156/24 157/2 166/15 166/16 169/2 170/22 171/3 171/4 171/22 173/20 173/23 174/14 178/9 178/11 178/13 180/12 182/1 183/3 183/14 183/14 184/13 185/25 186/1 188/3 188/7 188/7 188/22 191/10 192/14 193/11 195/15 196/13	157/7 157/12 157/24 158/19 159/2 159/3 159/21 160/24 162/9 164/1 164/9 167/5 167/8 168/15 168/16 169/20 169/23 169/25 170/3 170/15 170/16 170/19 171/18 195/2 <b>carer</b> [1] 180/18 <b>caring</b> [2] 171/13 182/5 <b>carried</b> [3] 82/3 82/3 158/24 <b>carry</b> [5] 10/24 11/3 11/15 29/19 152/12 <b>case</b> [11] 9/21 10/16 23/4 53/3 61/14 89/3 90/3 123/18 131/8 173/14 174/24 <b>cases</b> [9] 21/2 21/7 78/21 79/2 172/20 173/2 173/25 174/1 174/19 <b>castigated</b> [1] 186/18 <b>categories</b> [1] 36/3 <b>category</b> [9] 32/4 33/10 33/19 35/4 35/8 37/20 38/9 38/25 45/16 <b>cath</b> [1] 58/18 <b>caths</b> [1] 58/12 <b>Caucasian</b> [1] 11/2 <b>cause</b> [15] 18/13 48/14 52/25 54/14 60/23 62/18 70/13 70/19 73/1 79/10 124/23 183/15 184/13 185/25 189/21 <b>caused</b> [7] 32/9 50/17 67/3 69/9 96/5 110/20 185/10 <b>causes</b> [4] 54/16 69/13 94/22 178/1 <b>causing</b> [8] 40/2 49/14 55/4 109/1 109/2 127/17 185/13 187/5 <b>caveat</b> [1] 98/1 <b>caveats</b> [1] 12/2 <b>CCR4</b> [2] 29/24 30/3 <b>CCR5</b> [10] 29/24 30/2 30/2 30/4 30/5 30/6 138/19 139/2 173/7 173/12 <b>CCR5-bearing</b> [1] 30/2 <b>CD4</b> [27] 26/18 28/10 29/19 29/20 32/14 32/17 32/19 33/24 34/1 34/3 34/5 34/13 34/17 36/16 36/17 36/18 37/7 37/12 37/16 105/14 114/14	115/6 115/11 118/24 118/25 138/25 163/24 <b>CD8</b> [1] 27/3 <b>CDC</b> [4] 31/17 35/23 45/14 63/10 <b>ceased</b> [1] 195/11 <b>cell</b> [33] 18/1 18/2 29/21 92/13 94/16 94/18 99/24 137/19 137/21 137/21 138/14 138/15 138/22 139/4 139/5 139/9 139/12 140/11 140/14 140/15 140/17 140/17 141/1 141/22 141/23 142/1 142/1 142/3 142/3 142/4 144/25 173/4 173/4 <b>cell-based</b> [1] 18/2 <b>cell-free</b> [1] 18/1 <b>cells</b> [30] 6/24 7/1 18/4 24/12 24/14 26/18 29/12 29/16 29/17 29/19 32/14 33/25 34/3 37/5 99/22 99/25 99/25 100/1 119/11 126/22 138/20 173/7 173/9 174/14 181/10 181/10 181/11 181/12 188/6 188/21 <b>cellular</b> [3] 24/17 29/11 139/12 <b>cent</b> [26] 10/10 11/3 12/19 76/21 76/23 93/15 93/15 94/22 101/21 115/9 115/10 115/18 115/19 116/17 117/13 118/11 120/23 122/22 135/4 135/7 150/16 176/9 191/20 192/19 192/22 193/1 <b>Centers</b> [1] 31/18 <b>central</b> [1] 188/3 <b>centre</b> [12] 137/20 138/22 141/1 141/22 141/23 156/18 168/5 170/8 170/22 189/19 189/20 191/6 <b>centred</b> [1] 171/23 <b>centres</b> [7] 79/17 79/21 80/16 80/21 86/22 167/6 171/6 <b>cerebral</b> [1] 47/10 <b>certain</b> [7] 15/2 25/1 26/16 26/18 44/6 117/19 132/25 <b>certainly</b> [26] 13/5 13/6 14/3 14/18 39/18 42/24 50/3 56/1 65/22 70/10 76/6 78/16 85/14 88/7 89/3 89/15 109/2 113/16 116/8 120/18 136/3 177/3	178/10 178/11 183/4 188/12 <b>cervical</b> [4] 43/2 43/5 43/7 43/9 <b>cetera</b> [3] 154/4 161/12 162/6 <b>chain</b> [3] 92/19 142/21 148/15 <b>chair</b> [3] 4/24 5/21 5/22 <b>chalky</b> [2] 95/1 109/7 <b>challenge</b> [3] 71/2 112/2 112/18 <b>challenges</b> [6] 60/6 81/25 82/8 88/12 88/21 89/15 <b>challenging</b> [2] 28/6 111/12 <b>chamber</b> [1] 186/14 <b>chances</b> [1] 103/8 <b>change</b> [9] 16/6 16/10 16/14 35/7 53/3 117/25 122/8 122/18 191/5 <b>changed</b> [7] 89/2 89/23 114/20 115/12 120/4 150/5 155/12 <b>changes</b> [4] 12/17 43/9 75/17 105/13 <b>changing</b> [4] 101/8 129/11 129/12 168/21 <b>characterised</b> [1] 17/12 <b>charity</b> [1] 5/23 <b>charting</b> [1] 32/25 <b>chat</b> [2] 155/18 156/8 <b>check</b> [5] 46/20 137/6 158/2 191/20 191/22 <b>checking</b> [1] 167/21 <b>cheeks</b> [3] 42/19 42/20 100/13 <b>chemokine</b> [1] 29/23 <b>chemotherapeutic</b> [1] 64/5 <b>chemotherapy</b> [4] 98/24 102/12 173/23 174/2 <b>cherry</b> [1] 109/11 <b>chest</b> [1] 58/14 <b>chicken</b> [3] 38/20 60/20 60/22 <b>child</b> [14] 2/8 9/4 12/23 14/15 76/8 84/22 85/3 97/18 111/11 149/22 152/8 171/12 181/21 186/10 <b>child's</b> [1] 168/22 <b>childhood</b> [3] 22/6 36/15 192/6 <b>children</b> [39] 2/11 2/17 21/25 22/5 22/9 35/21 36/7 36/12 36/17 36/21 36/25
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(55) break - children



<b>C</b>	20/3 20/23 32/11 33/9 34/20 35/2 37/19 37/25 38/8 45/15 46/9 60/4 69/16 82/5 93/24 103/12 103/12 104/22 105/1 105/9 105/15 105/19 105/19 105/22 114/19 115/7 115/18 122/16 126/2 128/12 128/13 129/16 129/17 183/25 <b>clinically</b> [3] 7/14 8/16 31/7 <b>clinicians</b> [1] 91/8 <b>clinics</b> [7] 5/18 20/3 60/11 122/5 163/10 163/20 165/3 <b>clock</b> [1] 97/20 <b>clogs</b> [1] 53/6 <b>clones</b> [1] 138/3 <b>close</b> [5] 80/18 166/1 174/20 187/2 194/13 <b>closer</b> [1] 20/24 <b>CMV</b> [7] 56/18 56/19 56/22 59/3 59/16 59/22 67/11 <b>CNS</b> [1] 64/1 <b>co</b> [34] 3/1 3/1 3/4 24/13 24/15 27/14 27/19 28/18 29/22 29/25 41/21 41/23 58/24 59/4 59/14 64/3 71/6 87/15 87/16 87/20 116/2 123/6 134/16 138/19 139/1 148/7 171/24 176/13 176/16 176/19 182/19 183/6 192/24 194/17 <b>co-infected</b> [1] 182/19 <b>co-infection</b> [15] 3/1 3/1 3/4 41/21 41/23 59/4 59/14 64/3 87/15 87/16 87/20 176/13 176/16 176/19 183/6 <b>co-infections</b> [3] 27/14 27/19 28/18 <b>co-morbidities</b> [5] 71/6 116/2 123/6 134/16 192/24 <b>co-ordinated</b> [2] 171/24 194/17 <b>co-ordinating</b> [1] 24/13 <b>co-ordination</b> [1] 24/15 <b>co-receptor</b> [3] 29/22 138/19 139/1 <b>co-receptors</b> [2] 29/25 148/7 <b>code</b> [1] 140/8 <b>cognition</b> [1] 188/22 <b>cognitive</b> [3] 60/6 187/16 188/14	<b>cohort</b> [10] 2/19 27/21 62/8 131/11 132/14 134/20 135/3 135/8 192/15 195/12 <b>cohorts</b> [2] 134/10 192/16 <b>colleagues</b> [5] 2/15 37/9 37/12 80/19 186/11 <b>collecting</b> [2] 86/1 136/21 <b>collectively</b> [1] 195/23 <b>College</b> [1] 4/22 <b>colon</b> [1] 57/19 <b>column</b> [1] 32/4 <b>combination</b> [5] 101/18 105/25 106/3 110/24 145/25 <b>combinations</b> [2] 164/15 182/21 <b>combined</b> [2] 88/18 146/14 <b>combining</b> [1] 139/22 <b>come</b> [33] 13/23 15/22 28/16 29/7 38/25 39/12 40/7 41/4 46/20 47/20 47/23 47/24 61/1 66/15 68/8 96/9 106/17 112/21 118/17 120/2 124/1 125/24 126/9 138/3 152/1 155/18 156/8 156/13 158/16 169/8 169/15 175/2 196/14 <b>comes</b> [13] 7/4 38/15 54/10 82/24 119/12 121/15 128/24 138/2 155/14 158/18 173/6 188/14 192/23 <b>comfortably</b> [1] 30/25 <b>coming</b> [16] 14/5 38/12 44/3 44/24 55/16 68/11 68/13 102/7 137/24 139/8 149/1 161/11 165/1 186/8 194/12 194/16 <b>comment</b> [4] 14/25 90/25 157/22 178/10 <b>comments</b> [2] 150/4 185/7 <b>common</b> [41] 7/9 8/22 9/1 11/9 19/20 19/22 20/7 36/7 38/17 38/18 38/21 39/2 39/23 39/25 41/2 46/24 49/25 51/3 51/21 52/24 56/9 57/9 62/14 63/25 65/7 70/6 72/12 72/25 95/14 96/22 100/8 104/5 124/17 124/20 128/14 130/16 155/10 173/18 178/7	182/24 190/5 <b>commonest</b> [1] 65/19 <b>commonest</b> <b>opportunistic</b> [1] 65/19 <b>commonly</b> [10] 28/17 31/19 31/25 36/13 47/21 52/24 58/25 63/23 65/9 104/1 <b>communication</b> [1] 156/1 <b>communities</b> [3] 5/20 77/13 78/11 <b>community</b> [10] 4/9 54/7 74/16 77/8 79/15 85/19 86/8 86/10 159/4 193/4 <b>compare</b> [1] 33/1 <b>compared</b> [10] 73/15 76/22 93/12 117/8 117/11 124/10 131/3 135/5 170/23 176/7 <b>comparing</b> [1] 147/25 <b>compartmentalised</b> [1] 79/11 <b>compete</b> [1] 142/19 <b>competes</b> [1] 142/15 <b>competing</b> [1] 142/14 <b>competitive</b> [1] 142/22 <b>complaint</b> [1] 51/3 <b>complete</b> [4] 14/8 21/6 140/6 140/7 <b>completely</b> [5] 59/23 168/10 174/16 175/20 188/24 <b>complex</b> [5] 53/8 116/25 117/3 133/22 146/4 <b>complicated</b> [3] 13/13 58/21 170/13 <b>complications</b> [6] 69/2 69/6 72/10 73/9 167/11 189/3 <b>component</b> [1] 125/2 <b>comprehensive</b> [1] 36/1 <b>compromised</b> [1] 35/15 <b>computer</b> [3] 138/8 138/10 152/22 <b>concentration</b> [1] 34/3 <b>concern</b> [8] 85/8 85/22 132/10 179/14 181/20 181/22 181/24 182/2 <b>concerned</b> [7] 127/25 131/11 166/23 167/1 167/2 180/3 187/4 <b>concord</b> [1] 184/16 <b>condition</b> [11] 52/6 52/15 56/9 61/20 62/1	65/4 97/3 116/21 119/24 150/19 155/22 <b>conditions</b> [34] 32/11 37/10 37/25 38/12 38/15 40/10 40/11 40/18 41/4 42/5 49/25 50/2 50/11 50/15 52/9 52/11 65/7 66/19 66/21 66/25 67/3 67/8 71/5 72/13 73/3 74/11 83/23 92/3 133/18 133/19 133/25 170/23 180/8 190/1 <b>conference</b> [2] 106/1 110/3 <b>confidentiality</b> [2] 85/20 111/14 <b>confines</b> [1] 159/17 <b>confirm</b> [1] 68/10 <b>confirmatory</b> [1] 163/23 <b>confusion</b> [1] 60/5 <b>conscious</b> [1] 53/4 <b>consent</b> [1] 168/17 <b>consequence</b> [6] 7/22 22/7 46/11 53/14 70/2 87/4 <b>consequences</b> [10] 22/4 44/20 81/25 177/6 187/17 187/19 193/17 193/20 194/5 194/6 <b>consequent</b> [1] 82/11 <b>consider</b> [1] 114/7 <b>considerably</b> [1] 192/9 <b>considered</b> [2] 167/16 190/23 <b>consistent</b> [1] 91/5 <b>consists</b> [1] 139/11 <b>constant</b> [4] 31/6 68/2 71/22 122/15 <b>constantly</b> [3] 24/19 30/19 126/15 <b>constitutional</b> [4] 39/15 41/11 41/24 43/19 <b>consultant</b> [3] 4/18 5/10 160/10 <b>consultation</b> [1] 171/12 <b>contact</b> [2] 80/8 180/13 <b>contain</b> [1] 107/19 <b>contained</b> [1] 116/14 <b>contains</b> [2] 180/12 183/12 <b>contemporaneous</b> [2] 91/18 91/24 <b>contend</b> [1] 60/11 <b>contents</b> [1] 6/11 <b>context</b> [3] 52/25 55/3 58/24	<b>continually</b> [1] 173/19 <b>continue</b> [9] 4/23 21/9 66/22 96/15 99/18 116/18 118/4 122/11 128/1 <b>continued</b> [2] 5/8 101/24 <b>continues</b> [1] 78/16 <b>continuing</b> [2] 72/15 155/2 <b>continuous</b> [1] 97/19 <b>continuum</b> [1] 157/5 <b>contract</b> [1] 172/7 <b>contraindications</b> [1] 184/1 <b>contrast</b> [1] 49/23 <b>contrasts</b> [1] 23/16 <b>contribute</b> [1] 26/3 <b>contributed</b> [1] 195/24 <b>contribution</b> [1] 27/11 <b>control</b> [13] 18/21 31/18 56/2 56/7 61/9 93/16 103/17 103/21 114/17 120/21 172/23 175/7 177/6 <b>controlled</b> [1] 24/24 <b>controllers</b> [3] 172/22 175/4 175/6 <b>convenient</b> [2] 136/21 136/25 <b>conversations</b> [5] 155/3 162/20 163/13 163/16 171/6 <b>COOKE</b> [3] 1/15 2/20 2/21 <b>cope</b> [1] 151/22 <b>copies</b> [2] 11/4 120/8 <b>coping</b> [1] 25/8 <b>copy</b> [3] 92/11 92/12 140/10 <b>core</b> [3] 76/14 136/22 172/5 <b>corner</b> [1] 132/2 <b>corners</b> [1] 14/5 <b>corona</b> [2] 74/20 74/21 <b>correct</b> [6] 2/4 10/20 45/21 87/25 157/20 185/3 <b>correctly</b> [2] 137/7 196/4 <b>correlation</b> [1] 125/16 <b>cost</b> [2] 94/1 110/5 <b>cotton</b> [1] 144/3 <b>cough</b> [2] 51/9 65/12 <b>could</b> [45] 9/15 10/16 12/25 13/21 15/12 30/14 30/25 33/7 33/8 33/12 40/8 47/9 50/6 51/7 57/13 58/15 60/8 60/8 65/10 66/7 69/18 79/5 86/12 89/8 98/7
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(56) children... - could

<b>C</b>	<b>cure</b> [6] 3/5 119/17 140/19 173/18 173/22 174/7 <b>cured</b> [4] 66/22 119/14 119/15 174/11 <b>curing</b> [1] 174/9 <b>curls</b> [1] 140/1 <b>current</b> [1] 127/24 <b>currently</b> [2] 4/2 5/22 <b>cut</b> [4] 25/12 141/7 144/16 144/19 <b>cut-off</b> [1] 25/12 <b>cycle</b> [3] 92/9 129/6 130/2 <b>cycles</b> [1] 128/20 <b>cytokines</b> [1] 187/24 <b>cytomegalovirus</b> [3] 27/21 58/22 68/17 <b>cytoplasm</b> [1] 139/6	<b>DDI</b> [6] 94/13 94/23 99/2 102/22 104/6 109/6 <b>dead</b> [1] 192/20 <b>deal</b> [21] 17/6 43/25 45/5 49/11 49/19 66/9 84/6 96/10 108/5 108/6 124/18 133/23 136/19 143/9 149/6 152/6 152/10 153/12 153/23 154/5 154/7 <b>dealing</b> [11] 74/25 75/2 75/4 75/4 75/15 128/22 150/20 150/21 151/2 157/9 157/16 <b>dealt</b> [4] 66/25 67/14 73/11 76/2 <b>death</b> [7] 73/1 93/14 93/17 187/3 189/22 192/11 192/15 <b>deaths</b> [6] 93/16 185/10 185/14 185/23 187/5 189/5 <b>debilitating</b> [10] 43/20 44/12 44/18 52/2 53/4 54/16 55/4 57/7 59/24 68/24 <b>decision</b> [4] 90/6 126/19 171/9 184/22 <b>decision-making</b> [3] 126/19 171/9 184/22 <b>decline</b> [2] 28/10 74/3 <b>declining</b> [1] 34/5 <b>decrease</b> [3] 53/11 97/9 186/10 <b>deemed</b> [1] 131/25 <b>deeply</b> [1] 62/22 <b>defend</b> [1] 24/9 <b>defer</b> [1] 170/7 <b>deferred</b> [1] 115/3 <b>defining</b> [12] 21/19 35/3 37/10 45/16 45/19 65/7 69/4 71/5 72/24 73/3 74/7 92/3 <b>definitely</b> [12] 15/3 15/23 17/1 26/13 104/7 104/7 113/15 188/2 188/18 188/20 189/2 190/3 <b>definition</b> [1] 34/18 <b>degree</b> [2] 46/2 60/12 <b>delayed</b> [1] 151/3 <b>deletion</b> [1] 30/6 <b>deliver</b> [1] 94/9 <b>delivered</b> [2] 160/24 167/6 <b>delivering</b> [1] 157/12 <b>delivery</b> [1] 12/22 <b>demonstrate</b> [1] 83/18 <b>demonstrating</b> [1] 186/15 <b>demoted</b> [1] 2/2	<b>density</b> [1] 124/8 <b>dentists</b> [1] 81/21 <b>depend</b> [5] 13/6 38/13 40/19 47/7 168/12 <b>dependency</b> [1] 75/23 <b>dependent</b> [5] 17/3 19/2 96/8 165/24 168/22 <b>depending</b> [3] 46/14 88/9 114/14 <b>depends</b> [9] 9/22 12/24 16/14 62/9 62/10 123/4 154/14 160/1 177/13 <b>depos</b> [1] 122/18 <b>depressed</b> [1] 79/7 <b>depression</b> [2] 78/21 125/11 <b>depth</b> [1] 77/5 <b>dermatitis</b> [1] 39/24 <b>dermatological</b> [1] 99/19 <b>describe</b> [1] 31/20 <b>described</b> [11] 19/14 20/9 24/2 75/21 98/5 109/6 111/9 137/12 139/10 195/21 196/7 <b>describes</b> [1] 173/25 <b>describing</b> [3] 82/16 153/8 157/11 <b>description</b> [1] 188/15 <b>designed</b> [1] 8/12 <b>desire</b> [2] 151/1 155/11 <b>desperate</b> [2] 94/4 98/19 <b>desperately</b> [1] 56/17 <b>despite</b> [4] 66/22 100/21 109/23 185/4 <b>destroy</b> [1] 182/10 <b>detail</b> [5] 12/10 153/24 154/5 172/3 193/9 <b>detect</b> [2] 23/7 23/12 <b>detected</b> [3] 17/22 22/18 23/25 <b>detection</b> [4] 23/15 23/23 105/6 120/9 <b>determine</b> [7] 13/24 16/20 16/21 18/6 25/6 26/20 28/1 <b>determined</b> [4] 27/25 28/1 29/22 186/7 <b>determines</b> [3] 25/3 25/7 123/3 <b>determining</b> [1] 105/2 <b>devastating</b> [1] 57/17 <b>develop</b> [10] 19/4 21/18 22/15 22/17 24/10 41/6 69/8 94/5 118/1 132/3 <b>developed</b> [11] 59/22	92/24 95/3 99/18 100/1 101/2 117/1 118/2 147/7 147/7 147/20 <b>developing</b> [4] 22/3 102/6 123/21 127/20 <b>development</b> [4] 23/10 23/17 91/13 123/5 <b>develops</b> [2] 16/17 87/23 <b>DF</b> [2] 128/4 128/5 <b>diabetes</b> [2] 122/4 133/20 <b>diagnose</b> [2] 22/19 45/13 <b>diagnosed</b> [18] 2/8 20/5 20/7 20/19 33/6 115/6 115/17 117/8 123/8 123/10 123/11 123/14 133/5 133/9 136/4 154/2 154/11 166/9 <b>diagnoses</b> [2] 71/1 149/13 <b>diagnosing</b> [1] 37/10 <b>diagnosis</b> [33] 3/5 12/22 20/13 20/24 21/14 23/10 32/12 45/18 49/9 49/15 50/5 76/16 78/9 84/8 115/14 149/8 149/10 149/17 149/22 149/25 150/6 150/22 150/25 154/1 154/13 154/17 159/7 161/4 161/12 163/21 169/8 169/10 195/1 <b>diagnostic</b> [3] 23/25 48/6 71/1 <b>diagram</b> [1] 141/6 <b>diagrams</b> [1] 191/9 <b>diarrhea</b> [3] 55/5 95/16 107/24 <b>diarrhoea</b> [10] 19/18 41/13 44/9 55/5 55/9 55/11 56/3 57/19 104/5 107/22 <b>did</b> [25] 43/16 61/7 61/17 70/13 78/23 79/18 80/10 83/9 85/11 93/2 93/13 94/23 100/21 106/5 108/11 110/22 112/21 112/21 126/8 147/2 175/4 181/5 185/5 185/21 186/25 <b>didanosine</b> [4] 94/13 97/5 101/16 127/18 <b>didn't</b> [28] 14/23 35/19 50/3 53/14 54/7 56/10 70/19 70/22 71/18 79/3 79/4 84/17	84/18 85/10 86/14 89/7 93/11 93/12 93/22 100/18 106/17 107/13 114/22 123/17 150/9 151/14 184/20 187/6 <b>die</b> [7] 78/10 101/24 136/9 136/10 136/11 181/12 189/11 <b>died</b> [8] 4/1 59/22 71/18 78/12 86/19 185/3 186/3 188/19 <b>dietary</b> [1] 106/21 <b>differ</b> [2] 26/2 27/1 <b>difference</b> [13] 21/25 26/4 28/3 28/4 34/21 34/24 109/14 109/24 152/18 174/10 175/15 179/2 184/21 <b>differences</b> [3] 7/13 7/14 194/14 <b>different</b> [47] 7/6 7/7 7/11 8/3 8/15 8/17 13/11 14/21 15/25 17/1 29/2 35/20 36/2 36/19 37/22 46/5 46/14 49/17 59/8 64/20 67/4 88/9 89/12 103/5 104/19 111/3 111/24 116/10 117/6 131/23 140/19 141/16 145/14 146/2 146/19 146/19 149/12 155/3 160/23 162/16 165/15 168/3 168/4 170/15 182/8 188/17 191/7 <b>differential</b> [1] 21/14 <b>differentiate</b> [2] 14/24 15/1 <b>differentiation</b> [1] 14/20 <b>differently</b> [2] 26/24 46/5 <b>differs</b> [1] 189/19 <b>difficult</b> [39] 13/14 26/19 41/24 42/22 43/20 43/24 44/13 44/17 51/4 56/6 58/1 61/5 61/25 64/16 67/21 75/10 88/16 94/24 95/25 100/9 100/17 102/22 102/22 106/19 107/1 109/16 110/19 110/25 112/17 121/17 135/11 135/13 140/19 142/6 146/5 150/23 151/22 174/17 177/2 <b>difficulties</b> [8] 44/23 78/4 84/3 88/2 88/5 95/24 109/5 124/6 <b>difficulty</b> [1] 51/13 <b>digest</b> [1] 165/21
----------	-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------	-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------	---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------	-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------	----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------



<b>D</b>	69/24 72/1 72/11 76/21 95/15 148/25 178/17 196/21 <b>dispensed</b> [1] 109/8 <b>disruptive</b> [1] 96/18 <b>distance</b> [1] 156/23 <b>distant</b> [1] 143/13 <b>distinguish</b> [1] 16/1 <b>distinguished</b> [1] 1/4 <b>distorts</b> [1] 143/14 <b>distributed</b> [1] 97/21 <b>diverse</b> [1] 128/18 <b>diversity</b> [1] 129/8 <b>divided</b> [1] 17/24 <b>dizziness</b> [2] 95/17 188/10 <b>DNA</b> [24] 13/25 92/11 92/19 139/12 139/15 140/4 140/5 140/6 140/10 140/11 140/15 140/17 140/20 140/21 140/24 142/2 142/2 142/16 142/21 144/10 144/12 144/14 174/14 175/18 <b>do</b> [76] 4/23 6/19 18/21 18/24 20/18 21/8 21/9 23/7 30/5 30/6 35/14 42/15 48/12 49/13 50/15 51/22 53/14 55/13 56/14 61/14 70/16 73/20 75/13 77/12 83/7 83/20 83/20 83/20 85/21 90/24 92/14 93/23 105/18 105/19 109/10 111/7 112/12 118/13 118/21 118/21 120/12 131/12 131/15 131/18 131/22 132/22 138/20 144/9 145/13 145/17 145/20 148/12 151/8 152/5 156/8 157/21 157/23 161/22 162/7 163/5 163/5 163/6 167/7 170/6 170/9 172/7 174/18 175/10 176/11 176/23 186/18 189/4 189/5 189/21 192/10 194/11 <b>doctor</b> [12] 2/7 5/5 40/10 77/7 112/8 112/8 112/9 125/4 125/15 156/17 156/17 160/10 <b>doctors</b> [3] 5/9 79/24 156/12 <b>document</b> [2] 42/1 95/9 <b>documents</b> [1] 192/7 <b>does</b> [30] 8/19 12/11 12/15 16/3 16/5 16/19	20/22 21/23 28/3 28/22 36/2 54/18 95/19 101/11 103/8 143/9 143/11 144/12 146/12 163/7 163/8 164/8 167/5 167/5 176/13 176/19 177/9 180/23 181/1 189/6 <b>doesn't</b> [24] 7/19 7/21 12/21 13/16 15/21 26/10 28/21 30/18 34/11 35/7 47/6 48/12 54/14 60/23 70/24 87/21 87/22 115/24 130/25 131/4 143/25 152/12 175/16 181/3 <b>doing</b> [15] 3/12 5/8 70/17 70/25 85/25 105/22 105/22 111/11 137/22 138/9 143/14 147/21 148/2 174/6 189/15 <b>domestic</b> [1] 181/15 <b>don't</b> [68] 8/1 8/6 9/17 11/8 14/15 16/8 16/13 19/4 26/17 26/25 27/15 37/13 37/13 38/6 38/22 39/2 46/17 49/11 51/16 51/18 56/13 60/20 61/18 71/8 73/22 84/10 87/11 87/18 98/14 101/2 106/12 112/1 118/1 124/2 125/1 127/10 129/12 129/24 131/15 131/16 132/1 133/1 135/25 138/18 138/20 145/19 148/18 148/20 153/7 153/14 157/1 158/9 163/17 165/6 166/5 170/21 173/7 173/12 174/21 175/3 175/6 175/12 179/8 179/10 179/20 188/16 190/11 195/17 <b>done</b> [16] 49/13 86/6 91/11 114/5 114/12 119/2 134/13 147/25 154/12 156/24 157/2 157/2 160/2 164/1 167/15 174/5 <b>donor</b> [4] 15/8 190/8 190/12 190/14 <b>donors</b> [2] 190/13 191/19 <b>door</b> [2] 86/25 143/20 <b>dormant</b> [1] 126/22 <b>dosage</b> [1] 168/22 <b>dose</b> [5] 94/2 94/3 96/17 107/23 130/3 <b>doses</b> [3] 66/6 98/2 103/5 <b>dosing</b> [2] 96/5	102/19 <b>dot</b> [1] 137/16 <b>double</b> [4] 92/11 103/8 140/2 140/3 <b>doubt</b> [3] 109/20 116/11 188/23 <b>down</b> [27] 19/3 36/11 42/3 51/1 52/21 53/7 54/18 64/23 69/11 77/19 80/2 83/14 93/15 102/4 105/11 115/8 115/10 120/9 120/10 120/11 128/11 144/1 144/7 147/23 148/4 168/8 173/6 <b>DR</b> [8] 1/17 1/18 2/3 4/4 4/15 23/9 23/12 95/24 <b>DR DAVID</b> [1] 1/17 <b>DR IAN</b> [1] 1/18 <b>Dr Jefferey</b> [1] 23/9 <b>Dr Johnston</b> [1] 4/4 <b>Dr Williams</b> [2] 23/12 95/24 <b>dramatic</b> [2] 110/4 113/19 <b>dramatically</b> [1] 191/6 <b>draw</b> [3] 69/7 69/14 69/22 <b>drawn</b> [3] 27/8 68/25 135/15 <b>dreadful</b> [2] 56/5 56/5 <b>drenched</b> [1] 44/9 <b>drinking</b> [1] 11/13 <b>drive</b> [2] 72/19 166/25 <b>driven</b> [1] 84/9 <b>drives</b> [1] 166/21 <b>drug</b> [38] 58/14 63/9 63/11 66/8 66/16 92/17 92/22 93/1 93/12 94/7 94/21 96/11 96/15 97/21 97/23 98/4 101/15 102/23 106/24 107/1 107/6 108/24 110/25 121/12 125/2 125/3 125/4 127/16 128/15 128/17 145/22 145/24 148/10 148/13 148/13 148/16 168/21 185/6 <b>drugs</b> [89] 8/1 8/6 8/11 8/12 9/7 49/11 49/13 61/23 66/13 89/17 92/15 92/22 94/5 94/18 95/2 95/5 99/22 100/13 100/20 101/18 102/24 103/2 103/5 103/7 103/11 103/18 103/25 104/11 104/18 104/20 104/20 105/7 107/10 107/14 107/19 107/22 108/10 111/4 111/8 111/15	111/15 111/16 111/17 111/20 111/20 112/20 113/10 113/20 113/23 114/9 116/15 117/2 119/16 122/16 123/23 124/3 124/10 124/11 124/19 124/22 124/24 125/7 125/18 126/13 127/7 127/12 127/18 127/24 128/22 129/5 129/10 130/3 130/6 130/24 131/8 145/14 146/2 146/13 146/19 147/6 147/13 147/17 148/4 161/21 164/14 183/1 183/11 188/16 188/16 <b>dual</b> [6] 100/24 101/15 101/22 102/15 126/24 139/22 <b>due</b> [3] 6/4 37/18 116/16 <b>durability</b> [2] 174/22 175/1 <b>during</b> [18] 1/5 4/8 5/13 19/6 22/6 25/2 25/5 25/5 25/19 46/13 91/8 113/20 163/22 178/19 178/20 179/7 179/13 180/14 <b>dwelling</b> [1] 58/13 <b>dying</b> [1] 98/21 <b>dysplasia</b> [2] 43/2 43/7 <b>E</b>	113/5 <b>easier</b> [1] 125/6 <b>easily</b> [1] 89/9 <b>east</b> [2] 5/18 82/6 <b>easy</b> [3] 39/9 62/16 113/2 <b>eat</b> [3] 51/5 51/15 51/23 <b>eating</b> [3] 43/23 47/13 51/6 <b>echo</b> [2] 110/1 186/14 <b>editions</b> [1] 69/16 <b>educated</b> [1] 70/21 <b>education</b> [5] 3/13 80/1 85/1 85/2 152/14 <b>EDWARDS</b> [4] 1/16 3/6 3/8 43/16 <b>effect</b> [17] 28/8 41/15 41/18 41/20 93/2 96/11 99/13 99/22 105/20 107/13 108/4 109/2 109/3 125/12 136/12 183/14 185/15 <b>effective</b> [22] 36/8 36/10 42/25 50/9 52/13 52/16 57/13 60/9 66/4 71/8 72/16 88/24 90/8 122/23 122/23 147/14 174/15 179/5 179/12 179/23 180/4 185/18 <b>effectively</b> [3] 73/12 172/22 181/3 <b>effectiveness</b> [2] 129/23 129/25 <b>effects</b> [51] 6/24 19/22 38/3 57/18 67/23 68/2 94/1 94/19 95/6 95/14 95/23 96/13 96/14 96/21 97/1 98/14 99/4 102/10 103/8 103/9 103/14 104/2 104/4 104/5 105/7 107/21 108/7 109/24 113/8 114/23 116/5 116/16 116/19 116/20 117/5 123/7 124/18 127/5 128/14 129/2 131/7 132/15 182/16 182/21 182/25 183/7 183/19 184/24 185/8 185/22 187/14 <b>efficiently</b> [1] 37/6 <b>either</b> [8] 14/10 108/15 122/17 130/12 139/2 162/16 171/1 177/22 <b>elbowing</b> [1] 142/25 <b>elderly</b> [1] 195/7 <b>element</b> [2] 69/19 84/20 <b>elements</b> [1] 75/8
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---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------	----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------



<b>E</b>					
<b>eleven</b> [1] 152/9	<b>ensuring</b> [1] 157/4	86/22 87/9 109/1	<b>excitement</b> [2] 128/20	56/24 56/25 62/11	<b>family</b> [18] 2/16 3/25
<b>eliminate</b> [1] 142/7	<b>enter</b> [1] 29/12	115/5 124/9 124/19	128/21	126/24 172/8	6/22 52/3 56/23 75/4
<b>elite</b> [3] 172/22 175/4	<b>entering</b> [1] 137/21	124/21 127/6 127/16	<b>excluding</b> [1] 167/10	<b>exposure</b> [29] 9/13	75/24 76/10 78/6
175/5	<b>enthusiasm</b> [1] 186/9	129/5 158/4 164/22	<b>exclusively</b> [1] 82/22	10/5 10/14 10/15 11/7	78/13 78/15 78/23
<b>elongating</b> [1] 92/19	<b>entirely</b> [2] 156/10	168/11 171/7 174/15	<b>Excuse</b> [1] 187/7	11/20 11/23 11/24	80/3 85/3 150/19
<b>else</b> [14] 12/11 40/12	196/4	174/19 175/5 181/5	<b>exercise</b> [1] 50/16	11/25 12/5 12/6 12/13	163/14 179/17 189/25
54/18 55/23 66/16	<b>entry</b> [8] 29/21 138/24	<b>event</b> [2] 50/12 55/2	<b>exertion</b> [1] 65/17	15/18 16/4 16/17	<b>family's</b> [1] 151/2
68/10 70/15 77/21	140/23 141/18 147/3	<b>events</b> [2] 91/5 114/9	<b>exhausted</b> [1] 31/2	16/25 22/20 22/23	<b>fantastic</b> [2] 106/2
95/19 96/12 116/3	147/7 148/6 148/9	<b>ever</b> [4] 20/20 54/12	<b>exhaustion</b> [1] 177/5	23/1 23/6 45/25 47/17	196/7
130/18 152/25 161/4	<b>enzyme</b> [12] 92/8	149/16 149/21	<b>exhibit</b> [1] 66/23	48/3 123/5 172/13	<b>far</b> [18] 8/10 20/6 26/7
<b>emerge</b> [8] 46/21 47/3	92/13 92/17 92/18	<b>every</b> [10] 83/22	<b>exist</b> [3] 50/3 79/18	172/14 175/25 177/14	32/2 46/8 59/2 72/23
47/4 48/14 71/17	93/4 94/15 141/10	102/17 119/21 122/12	124/24	177/25	73/1 94/7 108/2
101/15 106/5 169/16	141/19 142/20 143/14	122/20 123/15 125/4	<b>existing</b> [1] 147/17	<b>exposures</b> [1] 46/14	112/22 114/18 114/18
<b>emerged</b> [3] 107/11	143/18 147/5	125/19 126/14 126/18	<b>expand</b> [2] 111/6	<b>express</b> [6] 26/17	123/16 129/14 131/10
107/11 182/2	<b>enzymes</b> [2] 141/10	<b>everybody</b> [10] 84/16	111/23	27/4 30/5 30/6 173/7	137/25 182/4
<b>emerges</b> [1] 175/12	141/13	90/2 97/22 115/1	<b>expanding</b> [1] 142/21	173/12	<b>fast</b> [2] 176/24 188/11
<b>emerging</b> [1] 190/1	<b>epidemic</b> [1] 189/9	159/6 163/11 166/4	<b>expect</b> [4] 20/21	<b>expression</b> [2] 43/18	<b>faster</b> [1] 74/3
<b>emotional</b> [3] 78/1	<b>epidemiological</b> [3]	169/25 177/22 179/22	42/22 123/13 159/16	100/12	<b>fat</b> [3] 99/12 99/25
78/4 87/6	32/24 131/19 133/2	<b>everybody's</b> [1]	<b>expectancy</b> [16]	<b>exquisitely</b> [1] 61/4	100/1
<b>empathy</b> [1] 196/5	<b>epidemiology</b> [1]	135/19	73/17 123/13 123/16	<b>extensive</b> [1] 173/22	<b>fatal</b> [5] 52/7 52/8
<b>emphasise</b> [3] 117/20	131/20	<b>everyone</b> [2] 10/14	123/24 126/9 130/20	<b>extent</b> [7] 9/13 25/2	52/11 62/3 64/7
118/13 190/21	<b>epilepsy</b> [6] 69/7	128/25	130/22 130/24 131/3	51/25 79/20 117/19	<b>fear</b> [1] 77/19
<b>emphasising</b> [2]	69/12 69/20 69/21	<b>everything</b> [8] 28/24	131/13 131/20 131/25	133/1 172/12	<b>feature</b> [3] 53/5 55/7
186/21 186/22	70/1 70/5	66/16 85/20 101/3	132/23 133/23 134/7	<b>extra</b> [1] 169/16	187/22
<b>employed</b> [1] 23/3	<b>episode</b> [3] 59/17	116/3 154/23 155/1	184/17	<b>extraordinarily</b> [4]	<b>features</b> [2] 188/9
<b>emtricitabine</b> [1]	67/13 68/9	162/21	<b>expectation</b> [1] 158/7	2/15 52/3 97/11	188/12
183/12	<b>episodes</b> [2] 83/3	<b>everything's</b> [1] 46/18	<b>experience</b> [29] 1/24	150/23	<b>February</b> [2] 1/1
<b>Encephalitis</b> [1] 59/25	84/24	<b>evidence</b> [23] 6/2	38/16 38/18 82/18	<b>extraordinary</b> [1] 68/4	173/16
<b>encephalopathy</b> [1]	<b>Epstein</b> [1] 40/3	11/5 16/8 16/9 16/11	84/9 84/18 84/19	<b>extrapolate</b> [1] 131/24	<b>feed</b> [1] 51/22
70/12	<b>equally</b> [4] 73/4 94/19	37/21 45/13 50/14	85/15 103/24 106/2	<b>extreme</b> [2] 52/2 79/2	<b>feel</b> [10] 6/19 35/10
<b>encode</b> [1] 140/8	135/18 179/14	66/17 91/23 95/11	106/8 116/10 116/19	<b>eye</b> [2] 57/11 59/16	35/14 65/24 78/23
<b>encoded</b> [4] 92/13	<b>equals</b> [3] 152/19	103/21 121/11 121/14	116/20 123/9 123/11	<b>F</b>	80/10 83/7 86/25 87/1
140/11 140/12 144/15	152/19 158/6	121/14 149/1 149/11	126/3 126/14 127/11	<b>face</b> [2] 39/24 147/14	109/16
<b>encodes</b> [1] 141/9	<b>equation</b> [1] 152/19	174/8 176/14 178/18	128/10 128/17 131/20	<b>facilitating</b> [1] 157/3	<b>feeling</b> [10] 39/16
<b>encoding</b> [1] 138/9	<b>equivalent</b> [1] 73/5	184/7 187/5 187/10	132/5 150/11 153/3	<b>facing</b> [2] 82/8 192/25	39/21 41/13 51/5
<b>encountered</b> [1] 46/2	<b>era</b> [1] 80/24	<b>evolve</b> [1] 163/18	156/20 161/7 178/18	<b>fact</b> [25] 14/9 14/14	55/18 65/11 65/15
<b>encouraged</b> [1]	<b>eradicate</b> [1] 175/19	<b>evolves</b> [2] 13/15	193/22	34/22 37/6 41/3 64/1	79/2 96/18 158/10
171/13	<b>eradicated</b> [1] 173/3	18/8	<b>experienced</b> [6] 38/1	69/1 77/11 79/16	<b>feelings</b> [1] 150/22
<b>end</b> [16] 24/4 25/15	<b>eradication</b> [1] 119/18	<b>exacerbated</b> [1] 183/3	66/18 82/11 83/1	91/16 93/15 99/6	<b>feet</b> [1] 41/18
57/21 85/9 85/9 102/3	<b>errors</b> [2] 13/17	<b>exact</b> [1] 191/10	103/4 116/7	100/21 124/1 129/20	<b>fell</b> [2] 93/14 102/9
105/1 105/9 105/22	101/12	<b>exactly</b> [10] 35/5	<b>experiences</b> [3] 3/23	132/12 143/23 148/3	<b>felt</b> [6] 36/24 79/1
105/23 137/23 137/25	<b>escape</b> [1] 146/5	35/12 118/18 139/20	37/22 46/6	151/17 175/22 176/13	82/18 185/5 185/12
172/4 173/1 173/1	<b>especially</b> [1] 1/9	146/11 146/16 147/1	<b>experiencing</b> [2]	176/16 176/19 176/21	196/9
195/14	<b>essentially</b> [4] 7/2	161/3 161/13 161/13	48/22 83/2	177/9	<b>fever</b> [3] 19/9 20/11
<b>ended</b> [2] 112/13	121/21 146/7 158/23	<b>examination</b> [1]	<b>experiential</b> [1] 126/6	<b>factor</b> [4] 26/14 29/10	56/23
113/3	<b>established</b> [1] 20/7	160/20	<b>expert</b> [5] 91/7 136/23	29/13 121/2	<b>fevers</b> [1] 39/21
<b>energy</b> [1] 99/23	<b>estimate</b> [2] 34/2	<b>examinations</b> [1]	174/9 193/10 196/18	<b>factors</b> [12] 9/23 17/3	<b>few</b> [7] 76/23 87/15
<b>enfuvirtide</b> [3] 110/12	131/18	49/18	<b>expertise</b> [8] 1/24	17/4 19/2 26/3 26/20	89/12 93/10 116/17
110/15 110/17	<b>et</b> [3] 154/4 161/12	<b>Examined</b> [1] 1/21	126/6 161/6 161/7	27/6 30/20 45/25 46/4	164/23 165/24
<b>engage</b> [1] 161/5	162/6	<b>examining</b> [1] 196/21	162/19 163/12 196/5	88/18 172/17	<b>fewer</b> [2] 8/10 189/16
<b>England</b> [4] 76/19	<b>et cetera</b> [3] 154/4	<b>example</b> [27] 9/6	196/15	<b>fail</b> [1] 118/6	<b>field</b> [1] 122/19
115/15 191/10 191/16	161/12 162/6	10/23 15/8 20/3 43/21	<b>experts</b> [2] 1/5 196/8	<b>failed</b> [1] 102/2	<b>fifth</b> [1] 23/3
<b>enhanced</b> [1] 75/11	<b>eternal</b> [1] 135/21	46/22 47/9 48/9 54/6	<b>explain</b> [7] 23/20	<b>fails</b> [2] 18/24 18/24	<b>fight</b> [1] 17/25
<b>enjoy</b> [1] 51/15	<b>ethnic</b> [1] 5/19	56/12 57/12 58/2	37/24 45/23 46/4	<b>fair</b> [3] 14/25 128/19	<b>fighting</b> [1] 176/20
<b>enlarged</b> [1] 19/16	<b>Europe</b> [2] 192/16	73/23 94/20 97/2	117/16 144/2 154/9	185/7	<b>fighths</b> [1] 18/3
<b>enormous</b> [4] 106/11	192/25	105/16 121/12 128/3	<b>explained</b> [3] 45/17	<b>fairly</b> [2] 19/22 154/8	<b>figure</b> [7] 13/9 18/16
111/20 152/12 152/18	<b>even</b> [42] 15/19 20/22	147/12 159/19 166/17	109/13 137/13	<b>fairness</b> [1] 112/3	190/16 190/17 190/18
<b>enough</b> [8] 8/3 32/9	25/21 36/7 50/4 50/25	175/18 184/11 185/20	<b>explaining</b> [1] 156/6	<b>fall</b> [4] 36/2 36/16	191/18 191/23
47/2 71/14 96/24	52/5 52/6 54/2 57/13	185/24 191/8 195/11	<b>explanation</b> [3] 90/25	113/18 114/1	<b>fill</b> [2] 100/6 100/6
108/14 110/23 194/25	60/5 60/9 66/3 67/10	<b>excellence</b> [1] 155/24	137/13 144/9	<b>families</b> [8] 5/17 56/7	<b>fillers</b> [1] 99/19
<b>ensure</b> [2] 71/13	67/13 76/18 76/19	<b>excessive</b> [1] 71/23	<b>explore</b> [1] 178/13	75/19 78/17 84/11	<b>films</b> [1] 62/20
80/17	77/22 77/23 78/11	<b>exchange</b> [1] 65/22	<b>exposed</b> [9] 9/24	86/4 111/24 171/8	<b>financial</b> [2] 170/4
	79/2 80/21 80/23 81/8	<b>excited</b> [1] 128/25	10/17 46/12 54/13		170/18

<b>F</b>	102/23	186/8 187/23 188/19	<b>genes [1]</b> 140/9	54/7 54/20 56/4 58/15	148/23 154/15 160/23
<b>find [17]</b> 16/7 39/3	<b>formulations [2]</b>	189/19 191/7 191/9	<b>genetic [8]</b> 10/25 19/2	58/16 68/9 70/22 71/3	165/12 167/4 167/12
49/18 51/8 69/23 95/4	102/21 112/5	192/5 192/15 193/9	26/20 27/5 30/5	77/21 78/5 86/12	176/24 176/25 181/6
98/20 100/8 120/7	<b>forth [3]</b> 60/7 68/11	194/12 194/16 194/25	148/11 148/16 172/16	87/12 87/13 87/18	181/7
126/15 129/18 158/10	68/13	195/19 196/18	<b>genetically [4]</b> 26/16	100/20 106/9 106/12	<b>GP [2]</b> 158/2 170/21
159/21 160/18 178/14	<b>fortunate [1]</b> 96/24	<b>front [1]</b> 154/6	26/25 27/25 186/6	111/18 116/18 120/21	<b>GP's [1]</b> 157/25
189/18 190/19	<b>fortunately [2]</b> 61/19	<b>full [5]</b> 59/8 111/15	<b>genital [2]</b> 120/19	126/19 144/1 144/5	<b>gp120 [1]</b> 138/25
<b>finding [1]</b> 186/11	62/1	111/16 160/19 186/9	176/6	145/14 151/5 151/19	<b>GPs [3]</b> 81/21 84/5
<b>findings [1]</b> 81/16	<b>forward [5]</b> 83/21	<b>function [1]</b> 74/3	<b>genitals [1]</b> 60/19	153/14 154/12 156/16	86/9
<b>fine [2]</b> 46/18 132/18	108/2 152/15 160/22	<b>functioned [1]</b> 99/25	<b>genotypes [1]</b> 15/11	158/2 158/14 159/22	<b>grade [1]</b> 72/14
<b>fingerprick [1]</b> 159/23	167/17	<b>functioning [1]</b>	<b>geographic [1]</b>	165/22 167/24 170/25	<b>gradually [1]</b> 129/1
<b>finished [1]</b> 196/1	<b>found [4]</b> 16/9 108/20	183/15	177/15	171/1 172/3 185/6	<b>GRAHAM [9]</b> 1/15
<b>first [54]</b> 1/25 2/7 4/21	129/2 150/22	<b>funding [4]</b> 81/8	<b>germs [1]</b> 46/13	<b>goal [1]</b> 119/18	2/21 9/17 26/15 30/10
5/4 5/6 5/12 17/13	<b>four [14]</b> 1/7 17/22	169/21 170/12 195/11	<b>get [60]</b> 10/15 12/4	<b>goes [7]</b> 20/13 30/17	34/24 56/21 104/3
17/16 18/12 19/7 22/5	18/8 18/9 19/6 19/7	<b>fungal [3]</b> 53/23 54/3	13/18 13/20 20/1 20/5	74/2 122/24 130/23	179/2
25/12 32/4 47/1 63/5	23/6 102/17 122/1	61/23	20/7 23/1 25/11 26/9	152/14 162/12	<b>Graham's [1]</b> 188/15
83/17 91/9 92/5 92/7	147/3 147/22 147/22	<b>fungus [2]</b> 51/10	31/2 32/7 32/10 42/9	<b>going [85]</b> 1/22 6/17	<b>grapefruit [2]</b> 108/19
92/15 92/22 94/24	147/25 167/19	52/24	44/1 53/8 59/16 59/19	10/22 23/22 25/13	108/23
95/21 97/18 99/16	<b>fourth [1]</b> 23/3	<b>further [10]</b> 15/18	60/21 62/17 64/11	30/24 31/21 34/13	<b>grateful [1]</b> 196/9
102/18 104/12 109/19	<b>frailty [4]</b> 73/18	54/7 67/21 77/23 98/9	70/5 74/21 77/10	39/8 39/13 40/5 40/16	<b>great [14]</b> 39/17 43/14
112/10 113/1 116/13	133/13 133/16 133/25	102/4 123/21 137/11	84/11 86/18 94/5	45/2 45/10 46/6 47/4	57/3 75/23 77/5 84/6
117/7 128/5 138/13	<b>France [1]</b> 47/12	195/17 196/18	96/16 98/14 108/13	47/20 48/2 50/1 50/11	109/9 133/23 150/9
140/22 141/18 142/10	<b>free [3]</b> 6/19 18/1	<b>fusion [2]</b> 139/4	108/14 108/24 111/7	51/10 52/21 55/18	152/6 152/23 153/24
147/4 153/16 154/1	169/25	147/18	124/2 129/9 130/3	64/4 70/15 71/3 71/24	154/5 186/15
154/12 154/16 160/13	<b>frequently [3]</b> 19/14	<b>future [2]</b> 135/16	130/7 138/14 138/21	75/16 75/22 76/9 77/2	<b>greater [6]</b> 16/23
163/17 164/10 164/12	50/13 168/23	136/7	142/20 143/1 143/1	77/14 77/17 77/25	82/23 175/24 177/11
164/22 164/23 165/8	<b>fridges [1]</b> 111/15	<b>futures [1]</b> 75/20	143/4 145/24 148/12	83/20 83/22 87/12	190/24 190/25
165/24 166/19 168/13	<b>friends [1]</b> 136/10	<b>G</b>	148/15 156/4 158/8	90/13 90/23 91/7	<b>greatest [1]</b> 121/2
190/7 191/19	<b>from [125]</b> 2/13 2/18	<b>gain [1]</b> 29/21	158/9 158/14 162/7	91/10 105/25 120/6	<b>greatly [1]</b> 70/9
<b>fit [2]</b> 79/4 94/8	6/1 8/23 8/24 8/24 9/4	<b>gambit [1]</b> 78/20	166/15 174/16 181/6	127/7 129/5 129/13	<b>grief [1]</b> 136/18
<b>fitted [1]</b> 58/19	11/11 11/14 11/17	<b>game [1]</b> 111/3	181/11 182/12 188/7	129/22 131/13 135/19	<b>ground [1]</b> 97/12
<b>five [4]</b> 2/12 3/16 63/4	13/25 13/25 15/7 16/2	<b>garden [1]</b> 59/7	188/11 188/16 193/9	136/9 144/5 144/7	<b>ground-breaking [1]</b>
137/24	22/9 22/20 23/22	<b>GARETH [7]</b> 1/14	<b>gets [4]</b> 6/25 52/12	148/4 148/21 149/7	97/12
<b>flaky [1]</b> 39/24	27/21 29/7 31/17	14/19 55/1 75/13	153/9 153/9	152/5 153/3 154/20	<b>group [5]</b> 2/15 52/1
<b>flat [1]</b> 48/13	33/12 45/10 46/8	75/24 111/2 121/6	<b>getting [18]</b> 44/4 44/5	157/16 158/5 158/8	82/3 175/10 196/8
<b>flavour [1]</b> 109/11	48/22 50/8 53/9 56/1	<b>gas [1]</b> 65/22	51/8 53/18 58/1 58/20	160/21 160/21 161/2	<b>groups [3]</b> 11/7 80/8
<b>flavoursome [1]</b>	56/6 56/24 66/18 67/8	<b>gastrointestinal [3]</b>	70/25 72/17 85/12	161/22 162/24 163/5	91/7
109/10	68/5 69/3 70/10 75/6	107/21 109/3 124/7	100/22 103/2 130/10	163/18 164/18 164/19	<b>grow [1]</b> 152/20
<b>flow [1]</b> 53/7	75/19 81/16 81/20	<b>gastrostomy [1]</b>	133/14 147/23 161/10	164/25 165/6 165/15	<b>growing [2]</b> 126/17
<b>flows [1]</b> 170/13	81/21 81/21 82/24	112/14	188/8 194/15 195/8	166/7 167/14 167/21	150/24
<b>flu [4]</b> 19/10 19/25	84/11 85/1 86/2 86/14	<b>gathering [1]</b> 164/4	<b>give [18]</b> 11/18 30/23	168/8 171/18 171/21	<b>growth [2]</b> 168/23
20/1 20/10	87/19 87/20 92/11	<b>gave [4]</b> 63/5 134/16	33/2 34/10 42/17 46/5	176/6 182/7 183/18	169/1
<b>flu-like [3]</b> 19/10	93/14 95/12 97/10	187/10 195/12	73/7 91/23 92/4	191/24 194/19 196/16	<b>grumbling [1]</b> 33/16
19/25 20/10	101/4 102/5 103/2	<b>general [12]</b> 4/6 4/7	100/25 111/11 112/15	<b>gone [10]</b> 30/16 33/18	<b>guarantee [1]</b> 132/20
<b>Fluconazole [1]</b> 42/11	103/21 104/3 104/6	12/1 12/6 25/9 70/6	145/24 149/1 156/5	49/16 59/3 83/14	<b>guardians [1]</b> 191/17
<b>fluff [1]</b> 144/4	104/6 105/8 105/21	80/22 84/7 85/18	162/9 177/10 196/15	119/10 120/15 137/8	<b>guess [4]</b> 51/7 70/21
<b>fluid [3]</b> 53/6 55/8	111/6 113/16 115/12	87/25 177/13 183/5	<b>given [12]</b> 6/2 45/12	151/9 191/12	154/21 163/2
106/21	115/16 116/20 117/7	<b>generalist [1]</b> 4/12	96/12 149/7 149/10	<b>good [16]</b> 4/5 5/3 19/4	<b>guesswork [1]</b> 135/15
<b>fluids [4]</b> 8/25 11/19	117/10 117/11 119/1	<b>generality [1]</b> 191/2	153/25 161/24 174/8	23/11 53/21 54/5 57/4	<b>guided [1]</b> 48/6
120/20 176/6	120/2 120/20 121/1	<b>generally [22]</b> 6/18	176/4 176/15 183/25	57/12 71/10 151/6	<b>guidelines [2]</b> 115/12
<b>focus [2]</b> 2/25 70/2	121/15 122/11 124/19	13/24 13/25 17/21	184/3	155/21 157/7 158/13	164/16
<b>follow [2]</b> 23/8 156/25	126/14 127/12 131/9	27/18 27/23 30/1	<b>gives [3]</b> 34/2 139/12	166/12 179/9 194/22	<b>gut [4]</b> 57/18 57/19
<b>follow-up [1]</b> 156/25	131/24 134/3 134/10	39/21 52/18 70/3	168/17	<b>Goodness [1]</b> 56/20	57/21 62/17
<b>following [4]</b> 17/22	135/21 136/22 140/19	107/18 116/22 130/14	<b>giving [6]</b> 10/7 93/8	<b>got [41]</b> 7/7 14/13	<b>gym [1]</b> 30/24
68/5 93/6 196/25	144/13 145/17 147/2	132/8 147/24 148/1	155/20 184/7 184/12	15/9 18/17 25/20	<b>H</b>
<b>food [3]</b> 51/2 51/5	149/12 149/18 149/22	148/14 179/6 179/15	184/13	31/21 31/24 32/16	<b>Hackney [1]</b> 5/15
51/15	151/10 152/15 159/3	187/5 187/25 189/20	<b>glandular [2]</b> 20/11	37/4 37/7 43/5 46/14	<b>had [109]</b> 3/23 4/1 4/2
<b>forefront [1]</b> 38/15	159/23 164/9 164/13	<b>generate [2]</b> 24/19	56/23	46/17 47/14 54/12	9/18 10/17 14/16 16/2
<b>Forgive [1]</b> 35/19	168/3 170/15 172/5	156/20	<b>globe [1]</b> 7/17	58/3 61/3 63/22 96/6	20/20 20/25 22/3
<b>form [2]</b> 91/20 139/22	173/15 174/16 175/5	<b>generation [6]</b> 23/3	<b>go [53]</b> 12/9 17/4 17/5	98/6 100/18 105/3	22/23 22/23 36/19
<b>forming [1]</b> 77/22	175/20 178/21 179/18	92/5 92/15 95/21	20/2 21/1 25/21 36/15	108/9 120/4 120/16	37/8 37/22 38/20 41/9
<b>forms [1]</b> 128/3	180/20 181/21 182/21	99/16 107/25	37/24 40/10 42/3 43/9	138/13 138/14 140/5	41/21 43/5 43/6 45/5
<b>formulation [1]</b>	183/7 184/22 185/22		49/21 50/2 50/16 52/9	141/4 141/22 147/16	



<b>H</b>	118/18 154/18	<b>he</b> [1] 149/2	69/15 69/16 69/23	14/10 15/7 29/5	180/23 187/4 188/16
<b>had...</b> [88] 46/15 48/4	<b>happy</b> [2] 36/23 168/7	<b>headache</b> [4] 19/10	91/3 97/3 136/25	<b>HIV-associated</b> [2]	188/18
51/20 51/20 53/19	<b>hard</b> [8] 11/5 43/11	19/20 53/3 95/17	137/23 139/7 141/2	50/17 194/21	<b>how's</b> [1] 161/22
53/25 55/21 55/22	56/8 57/20 64/4	<b>health</b> [38] 32/24 69/2	141/6 152/22 170/7	<b>HIV-negative</b> [1]	<b>However</b> [2] 154/22
55/23 56/8 58/6 59/2	108/13 112/4 153/17	69/5 73/8 74/11 76/19	180/7 182/17 190/17	73/10	170/6
59/10 60/20 61/21	<b>harder</b> [2] 179/5 195/8	76/20 76/25 77/7 78/3	190/21 196/14	<b>HLAB57</b> [1] 27/4	<b>HTLV</b> [1] 91/10
61/22 62/25 64/10	<b>harm</b> [1] 78/21	79/8 79/14 80/14	<b>herpes</b> [11] 38/17	<b>hold</b> [2] 136/8 146/6	<b>HTLV-3</b> [1] 91/10
65/23 66/4 66/12	<b>has</b> [96] 2/4 4/25 7/7	80/19 80/22 80/25	40/24 41/22 44/14	<b>holds</b> [1] 135/16	<b>huge</b> [15] 55/7 74/19
67/13 78/7 78/14 80/1	8/9 10/14 12/22 13/1	81/4 81/18 86/24 87/8	44/19 60/15 60/16	<b>holidays</b> [1] 169/5	74/23 75/17 77/17
80/3 81/17 82/19	14/21 22/17 23/9	115/14 116/3 135/2	60/18 60/22 61/4	<b>holistic</b> [1] 171/13	78/13 78/18 86/13
82/25 83/3 83/4 84/3	24/11 26/15 27/17	135/7 136/1 136/3	62/12	<b>hollow</b> [1] 51/11	88/21 106/4 107/21
86/11 86/19 93/1 93/2	27/22 31/21 32/9	136/13 159/19 161/17	<b>herpes simplex</b> [3]	<b>home</b> [4] 68/9 78/10	109/24 113/16 150/10
93/8 94/19 94/21 95/5	33/15 33/20 37/20	163/9 163/10 167/1	60/16 60/18 60/22	86/12 153/14	151/13
96/15 96/19 97/5	46/24 49/1 49/12	167/2 191/9 191/16	<b>herpes zoster</b> [1]	<b>homely</b> [1] 142/24	<b>human</b> [4] 7/4 129/11
99/18 99/22 100/20	50/14 55/1 62/11	194/22 194/25 195/6	60/15	<b>Homerton</b> [2] 5/13	140/21 152/11
101/16 103/1 103/3	63/22 64/21 66/17	<b>healthcare</b> [7] 78/25	<b>HHV8</b> [1] 178/1	5/14	<b>humane</b> [1] 59/13
103/14 104/25 106/20	73/17 75/1 78/18 79/9	82/21 83/16 83/24	<b>hid</b> [1] 77/6	<b>honest</b> [1] 127/11	<b>hump</b> [1] 100/14
106/20 107/20 107/20	80/7 83/11 83/14	86/7 87/7 125/14	<b>hideous</b> [1] 111/9	<b>hope</b> [5] 46/8 98/22	<b>hurdle</b> [1] 153/10
108/5 108/6 108/15	83/19 87/21 89/2 91/6	<b>healthy</b> [2] 61/15	<b>high</b> [34] 13/1 18/13	135/22 161/1 161/1	<b>hydrocephalus</b> [1]
108/16 110/14 111/15	95/11 95/24 97/24	75/19	18/15 19/5 22/16	<b>hopefully</b> [1] 156/4	53/8
111/16 111/25 113/8	99/6 99/7 104/4	<b>hear</b> [1] 122/11	25/23 31/2 31/5 31/9	<b>horrible</b> [4] 43/24	<b>hyper</b> [2] 187/11
113/9 114/9 114/18	109/12 117/9 119/10	<b>heard</b> [8] 22/10 28/17	31/9 53/18 66/13 76/3	55/12 59/24 65/25	187/15
121/19 123/1 126/11	119/14 119/22 120/2	37/21 50/14 96/22	82/19 93/25 94/21	<b>horrid</b> [1] 55/12	<b>hyper-activated</b> [1]
127/19 129/2 133/7	120/4 120/16 121/4	149/11 164/13 178/18	95/6 96/5 101/11	<b>hospital</b> [2] 2/9 3/2	187/11
134/15 136/4 169/9	121/19 122/2 122/10	<b>hearing</b> [1] 153/16	105/17 105/18 106/21	3/18 4/22 5/4 5/6 5/11	<b>hyper-activation</b> [1]
172/5 174/2 181/16	123/1 123/21 123/23	<b>heart</b> [2] 72/22 85/20	106/22 107/23 108/13	5/15 21/12 21/21 68/7	187/15
184/4 184/9 184/11	125/12 125/16 125/21	<b>helix</b> [1] 140/2	109/22 114/9 115/6	68/8 81/21 84/23	<b>hyperactivation</b> [1]
184/16 184/25 185/16	128/12 129/8 130/8	<b>Hello</b> [1] 3/7	118/10 148/11 161/25	107/5 121/25 159/10	30/21
189/17 196/9 196/10	131/9 133/6 133/7	<b>help</b> [11] 1/11 20/12	165/17 190/23 190/25	159/13 165/14 167/11	<b>hypertension</b> [1]
<b>hadn't</b> [3] 11/22 98/6	133/10 133/23 138/14	66/7 79/20 80/8 99/19	<b>higher</b> [16] 25/1 27/3	186/16	122/4
112/10	140/24 140/25 141/10	108/17 152/17 168/1	36/18 37/7 47/14	<b>hospitals</b> [4] 4/23	<b>hysteria</b> [1] 75/8
<b>haematological</b> [1]	148/10 149/11 149/21	184/19 186/18	64/11 71/25 80/14	68/12 86/11 121/23	
180/8	150/5 152/18 155/11	<b>helped</b> [3] 5/11 108/2	89/2 93/11 118/23	<b>host</b> [7] 17/4 26/5	
<b>haemophilia</b> [8] 3/17	159/16 159/18 161/3	108/5	118/24 133/17 133/19	26/8 27/10 46/3	
3/21 4/2 86/2 113/9	164/8 165/12 165/16	<b>helper</b> [3] 24/14 29/16	135/4 178/22	174/14 175/18	
131/10 136/4 181/17	167/11 168/15 169/7	33/25	<b>highest</b> [1] 83/1	<b>host-related</b> [3] 26/5	
<b>haemophiliac</b> [1]	174/23 177/6 178/18	<b>helpful</b> [3] 15/15	<b>highly</b> [7] 25/18 25/20	26/8 27/10	
134/10	180/18 195/11 195/24	16/12 126/7	53/12 90/11 113/4	<b>hour</b> [3] 163/17	
<b>hairy</b> [1] 40/2	<b>hasn't</b> [3] 8/8 148/1	<b>helping</b> [1] 126/7	122/23 196/4	163/22 170/8	
<b>half</b> [4] 91/9 150/16	175/13	<b>Henry</b> [5] 31/11 49/5	<b>hindered</b> [1] 145/12	<b>hours</b> [7] 97/20	
163/17 170/9	<b>hate</b> [1] 156/12	49/21 95/7 137/9	<b>Histoplasmosis</b> [1]	102/18 159/14 159/25	
<b>half-hour</b> [1] 163/17	<b>have</b> [372]	<b>hep</b> [1] 175/15	61/12	160/3 181/2 181/6	
<b>halved</b> [1] 115/9	<b>haven't</b> [7] 20/23	<b>hep C</b> [1] 175/15	<b>historic</b> [1] 180/3	<b>household</b> [4] 178/20	
<b>handful</b> [1] 172/5	36/22 66/25 110/11	<b>hepatitis</b> [38] 15/11	<b>historical</b> [1] 146/13	179/16 179/18 180/15	
<b>handfuls</b> [1] 108/22	127/24 143/8 180/6	22/11 23/17 27/14	<b>histories</b> [1] 3/23	<b>how</b> [62] 8/19 16/14	
<b>handle</b> [1] 57/3	<b>having</b> [40] 20/25	27/15 81/7 87/17	<b>history</b> [15] 7/10 7/20	16/16 17/1 17/12	
<b>handled</b> [1] 130/6	22/21 27/4 27/10 31/1	87/19 87/21 87/24	16/20 22/8 25/3 25/6	18/22 19/1 21/16 25/4	
<b>handling</b> [1] 180/19	34/17 47/14 62/25	88/3 88/4 88/10 88/11	27/20 30/3 30/9 86/7	25/7 26/22 27/16	
<b>hands</b> [1] 171/3	66/22 74/13 78/9	88/15 88/23 88/25	113/14 125/20 160/19	27/16 29/14 37/25	
<b>happen</b> [13] 46/10	88/22 100/16 100/19	89/5 89/8 89/14 89/19	186/13 192/17	45/13 47/4 48/4 48/25	
48/24 68/8 154/2	103/9 112/11 115/22	90/6 174/8 174/10	<b>hit</b> [1] 44/11	50/18 50/24 51/4	
155/16 162/25 163/8	116/23 119/1 120/24	174/11 175/16 175/21	<b>hits</b> [1] 22/2	55/20 83/20 90/7	
163/17 168/14 168/17	126/6 131/8 133/9	176/16 176/24 177/8	<b>HIV</b> [310]	95/25 96/20 97/21	
169/17 174/22 180/20	134/23 141/4 141/22	177/8 177/19 177/19	<b>HIV treatment</b> [1]	98/6 99/24 100/25	
<b>happened</b> [3] 83/5	153/6 159/11 159/22	182/6 182/7 182/19	170/14	101/1 103/23 111/7	
122/2 189/18	159/23 161/19 163/4	183/6 186/2	<b>HIV-1</b> [18] 7/5 7/7 7/12	111/9 116/6 119/22	
<b>happening</b> [11] 17/13	169/14 170/18 176/23	<b>hepatitis B</b> [3] 27/14	7/15 7/16 7/22 8/1 8/7	120/6 121/3 121/8	
30/13 32/25 48/7 48/8	176/23 182/21 185/21	89/14 182/6	8/12 8/16 14/8 14/10	125/17 126/12 129/13	
83/18 144/13 154/25	195/3 196/14	<b>here</b> [33] 1/6 1/11	14/21 15/6 15/21 29/1	131/12 139/10 144/2	
155/6 168/5 172/2	<b>HBV</b> [2] 176/14	2/22 31/14 32/3 38/5	29/1 29/9	146/7 150/4 153/19	
<b>happens</b> [10] 7/10	177/10	38/8 38/11 39/5 39/5	<b>HIV-1/2</b> [1] 14/11	154/11 161/3 163/7	
24/7 24/17 25/2 31/3	<b>HCV</b> [3] 176/14	48/23 49/10 50/17	<b>HIV-2</b> [10] 7/5 7/17	163/12 167/21 170/7	
62/14 91/20 100/11	177/10 191/22	55/15 60/16 65/6	7/19 8/2 8/6 8/10 8/12	170/9 172/13 174/4	

(61) had... - I don't



<b>I</b>	<b>I right [1]</b> 63/8	194/3	67/13 70/22 71/3	30/13 30/14 30/16	34/15 49/16 53/19
<b>I don't... [22]</b> 16/8	<b>I said [9]</b> 22/14 24/10	<b>I trained [1]</b> 5/6	71/13 71/22 73/4	30/18 30/20 31/3 31/4	71/4 76/1 80/5 98/18
16/13 71/8 84/10	55/20 101/10 101/23	<b>I understand [5]</b> 33/6	73/25 73/25 74/5	31/9 32/16 33/20	103/10 103/13 115/11
87/18 106/12 112/1	133/2 133/13 135/11	50/8 134/6 137/7	74/17 74/20 76/7	34/25 35/15 36/8	117/20 118/9 118/13
124/2 125/1 129/24	162/12	180/2	81/23 89/21 98/14	36/10 36/20 37/1	119/25 120/15 125/2
131/15 132/1 133/1	<b>I should [1]</b> 91/22	<b>I undertook [1]</b> 3/20	101/4 105/10 105/16	38/13 40/5 41/16 44/2	130/8 131/17 132/19
135/25 138/18 148/18	<b>I simply [1]</b> 14/15	<b>I was [4]</b> 45/2 55/16	106/9 109/1 110/9	45/24 46/11 46/18	148/10 160/6 161/25
148/20 165/6 166/5	<b>I specialise [1]</b> 5/5	77/2 182/7	112/9 113/9 115/5	47/1 47/19 48/13	162/18 169/17 170/6
179/8 179/10 190/11	<b>I spent [1]</b> 2/12	<b>I will [1]</b> 46/7	117/23 118/17 119/11	48/20 48/24 48/25	170/11 170/24 177/21
<b>I explain [1]</b> 144/2	<b>I start [1]</b> 163/1	<b>I won't [1]</b> 87/13	120/18 120/21 123/13	50/25 54/13 54/15	189/10 191/4
<b>I feel [1]</b> 86/25	<b>I started [1]</b> 3/8	<b>I work [2]</b> 86/23	123/17 126/11 127/23	57/3 57/5 57/14 60/24	<b>imported [1]</b> 7/18
<b>I finished [1]</b> 196/1	<b>I still [3]</b> 99/17 109/18	173/15	134/17 136/14 136/20	61/16 71/14 72/17	<b>impossible [3]</b> 13/5
<b>I first [2]</b> 2/7 5/4	111/16	<b>I would [16]</b> 7/23 28/5	138/7 140/12 141/25	73/13 102/8 105/12	13/9 75/23
<b>I guess [3]</b> 51/7	<b>I suppose [4]</b> 15/14	32/23 41/8 53/22 66/3	142/1 143/24 145/22	118/15 118/19 118/20	<b>impression [1]</b> 97/23
154/21 163/2	24/6 26/7 32/20	78/6 88/1 108/7	145/23 145/23 148/11	119/1 132/7 133/8	<b>improve [4]</b> 93/19
<b>I had [1]</b> 172/5	<b>I suspect [1]</b> 138/1	121/10 124/11 154/22	148/18 151/17 151/23	133/10 137/22 166/15	118/15 124/16 133/24
<b>I have [18]</b> 2/10 2/22	<b>I talked [1]</b> 86/8	165/16 170/7 190/20	152/6 152/6 153/15	166/16 172/23 173/9	<b>improved [7]</b> 73/17
3/19 4/6 4/8 4/16 4/18	<b>I think [144]</b> 3/19 8/9	194/19	153/15 154/16 155/5	176/20 176/22 177/6	89/9 102/3 102/24
4/24 5/16 39/4 79/16	10/22 12/1 12/8 13/8	<b>I wouldn't [4]</b> 13/8	156/8 156/9 158/7	187/10 187/21 187/23	103/19 105/4 133/23
86/3 162/23 172/5	13/14 14/2 14/14	97/22 112/7 164/11	159/8 159/10 159/18	<b>immunity [1]</b> 16/17	<b>improvement [7]</b>
187/7 188/13 195/15	14/24 15/5 15/7 16/7	<b>I'd [2]</b> 11/11 110/8	160/2 160/3 160/3	<b>immunocompromised [1]</b> 37/17	64/21 81/1 101/21
195/23	16/10 27/13 28/13	<b>I'll [1]</b> 123/25	165/12 165/16 166/5	<b>immunodeficiency [1]</b> 152/11	101/25 104/21 105/12
<b>I haven't [1]</b> 143/8	28/21 34/22 40/22	<b>I'm [13]</b> 2/7 4/2 4/6 5/4	166/8 167/17 167/20	<b>immunosuppressed [3]</b> 38/23 39/3 48/4	114/19
<b>I hear [1]</b> 122/11	41/2 41/11 43/18 45/5	6/17 55/15 55/18 59/5	168/15 168/24 169/7	<b>immunosuppression [9]</b> 46/3 47/6 52/6	<b>improvements [2]</b>
<b>I helped [1]</b> 5/11	49/7 51/13 55/9 55/14	79/22 167/13 178/4	169/16 170/8 170/12	54/25 64/10 67/5	106/3 184/17
<b>I hope [1]</b> 46/8	55/19 55/19 61/2 64/1	182/4 191/11	171/10 172/24 175/6	88/15 121/22 132/9	<b>improves [1]</b> 166/14
<b>I ill [1]</b> 162/24	66/24 69/14 69/21	<b>I've [6]</b> 5/19 85/14	176/4 177/24 178/25	<b>impact [40]</b> 7/21	<b>improving [2]</b> 44/2
<b>I imagine [1]</b> 121/24	69/25 70/11 71/20	86/6 87/10 87/20	179/21 180/13 180/16	27/10 27/15 27/17	73/12
<b>I just [11]</b> 59/1 81/23	74/16 74/20 75/9	137/8	180/17 181/4 181/5	27/19 27/22 28/19	<b>in infection [1]</b> 10/6
86/7 87/11 90/24 96/2	75/13 76/11 77/3	<b>i.e [1]</b> 32/19	184/5 184/11 184/19	28/22 28/23 44/16	<b>in-dwelling [1]</b> 58/13
131/5 137/6 137/14	78/15 78/21 78/22	<b>IAN [13]</b> 1/18 4/16	188/18 188/18 189/5	46/4 51/14 55/25	<b>in-patient [2]</b> 4/21
191/20 193/25	78/25 79/15 79/16	5/22 8/9 43/11 71/21	189/13 190/19 191/1	59/15 73/18 75/1	84/24
<b>I know [6]</b> 1/8 43/22	80/5 82/9 83/9 84/2	83/11 108/11 109/12	195/16	76/12 76/24 77/25	<b>in-patients [1]</b> 59/11
73/21 81/13 157/9	85/6 85/7 85/9 85/23	126/21 129/8 164/13	<b>ill [7]</b> 77/7 80/14 102/9	78/2 78/13 78/18 87/7	<b>incidence [7]</b> 76/20
181/17	86/5 87/1 87/13 88/8	173/25	162/24 184/4 185/17	101/20 106/4 113/16	89/1 106/22 113/17
<b>I lead [1]</b> 3/1	89/24 90/17 96/3 96/4	<b>Ian's [4]</b> 80/13 83/23	194/25	113/19 119/22 121/18	131/22 132/8 135/4
<b>I like [1]</b> 76/5	98/4 98/5 98/18 100/9	98/5 120/1	<b>ill-health [1]</b> 77/7	123/7 125/11 125/22	<b>include [2]</b> 19/20 65/5
<b>I look [1]</b> 109/18	103/10 103/13 110/2	<b>idea [5]</b> 11/18 54/11	<b>illness [8]</b> 21/11	129/22 129/24 130/8	<b>included [2]</b> 94/12
<b>I looked [1]</b> 65/23	111/6 111/13 113/11	84/6 164/18 181/23	21/19 24/7 28/15 66/1	131/3 133/25 148/24	110/11
<b>I mean [21]</b> 21/10	117/19 122/9 125/14	<b>ideal [2]</b> 108/3 113/20	75/1 78/15 78/20	193/23 195/3	<b>including [6]</b> 3/5 9/23
28/24 70/24 78/19	125/20 125/24 129/6	<b>identified [1]</b> 128/12	<b>illnesses [7]</b> 21/4	<b>impacted [6]</b> 50/19	14/6 51/24 111/21
82/22 97/11 97/13	130/22 131/17 131/18	<b>identifier [1]</b> 63/4	45/16 45/19 69/4 74/7	103/6 104/7 104/14	163/14
110/22 112/6 119/7	132/24 132/24 134/19	<b>identify [5]</b> 17/7 23/5	136/15 184/10	176/22 176/25	<b>increase [5]</b> 11/21
134/9 150/10 152/17	135/10 135/14 136/12	84/14 128/2 128/13	<b>image [1]</b> 187/13	<b>impactful [1]</b> 44/21	11/25 12/7 16/5 72/23
157/24 168/7 169/3	136/24 141/2 151/12	<b>idiopathic [2]</b> 39/5	<b>image [3]</b> 55/17	<b>impacts [7]</b> 74/13	<b>increased [15]</b> 12/5
177/2 185/22 186/7	152/2 154/22 155/19	69/20	121/24 179/7	117/21 125/10 127/19	12/6 70/9 73/8 73/9
186/19 192/14	155/25 156/5 157/6	<b>ie [1]</b> 23/23	<b>imagining [1]</b> 51/10	127/24 153/20 162/6	73/14 73/18 73/20
<b>I mentioned [1]</b>	158/5 158/12 161/14	<b>if [150]</b> 4/13 10/7	<b>immature [1]</b> 37/2	<b>impart [1]</b> 157/15	73/21 102/25 134/25
107/14	162/11 162/22 164/12	10/10 10/13 10/16	<b>immediate [1]</b> 163/2	<b>Imperial [1]</b> 194/17	135/1 135/2 177/17
<b>I missed [1]</b> 9/10	165/4 166/21 170/10	11/4 12/20 13/1 13/10	<b>immediately [3]</b>	<b>implant [1]</b> 58/11	179/17
<b>I moved [1]</b> 5/14	170/11 171/22 172/9	15/17 16/8 16/10	159/20 160/10 166/23	<b>implicated [2]</b> 11/19	<b>increases [1]</b> 72/20
<b>I must [1]</b> 196/1	174/23 176/9 176/14	17/11 20/18 20/19	<b>immensely [1]</b> 182/3	54/9	<b>increasing [1]</b> 125/25
<b>I never [1]</b> 87/1	179/4 180/6 180/8	20/23 21/25 22/2	<b>immune [94]</b> 7/1 7/3	<b>implications [3]</b> 15/22	<b>increasingly [5]</b> 56/25
<b>I note [1]</b> 136/20	181/14 182/17 185/11	22/12 22/22 25/16	16/15 17/2 17/14	85/11 96/19	156/22 159/21 165/2
<b>I often [1]</b> 74/17	186/3 186/7 187/3	26/22 30/14 30/23	17/17 17/20 17/21	<b>importance [4]</b>	195/7
<b>I pick [1]</b> 130/19	187/20 188/24 189/1	30/23 31/1 32/5 33/14	18/20 18/23 18/23	125/25 138/19 161/9	<b>incredibly [4]</b> 51/21
<b>I qualified [1]</b> 5/8	189/9 189/15 189/15	36/24 38/25 39/13	19/5 21/16 21/18	161/10	96/18 126/7 135/10
<b>I really [2]</b> 76/24	190/4 190/16 190/17	40/8 47/7 47/11 47/18	22/17 23/13 24/9	<b>important [35]</b> 24/13	<b>incremental [1]</b>
135/22	190/19 190/20 191/4	48/6 49/2 49/8 49/10	24/10 24/13 24/16	26/8 29/13 29/18 33/1	104/21
<b>I remain [1]</b> 135/21	191/13 191/18 191/21	49/19 50/19 50/25	24/17 24/18 24/20		<b>indeed [6]</b> 36/9 63/7
<b>I remember [5]</b> 37/8	191/23 193/8 194/8	51/16 53/14 54/2	25/4 25/7 26/9 26/12		91/25 97/16 148/17
96/16 97/17 105/25	194/19 195/5 195/14	54/13 54/15 57/3 61/3	26/21 26/24 27/16		196/15
186/7	<b>I think studies [1]</b>	62/3 62/23 62/25 63/2	27/24 28/1 28/7 29/18		<b>independently [1]</b>

(62) I don't... - independently

I	21/19 22/15 23/5 24/14 25/2 25/5 25/12 25/16 26/22 28/3 30/15 30/16 31/17 31/21 32/1 32/3 32/3 32/6 32/9 33/16 35/18 37/23 38/20 41/21 41/23 44/12 45/11 47/11 49/14 52/8 52/22 52/25 53/9 53/16 55/12 56/22 57/2 57/8 58/24 59/4 59/14 60/3 60/3 60/18 61/14 62/25 64/3 65/9 66/10 70/2 70/3 70/7 81/7 82/12 83/8 86/16 87/15 87/16 87/20 106/25 142/6 162/5 165/13 165/17 166/10 167/12 175/19 175/23 176/13 176/16 176/19 178/22 179/17 183/6 183/9 183/10 183/22 187/22 190/7 195/7 195/21	inhibitor [3] 104/13 106/19 143/7 inhibitors [14] 92/16 94/11 104/16 104/17 107/15 107/18 108/1 108/9 108/13 110/4 113/21 113/23 142/12 148/7 initial [7] 19/13 25/19 92/15 164/7 164/11 167/4 167/12 initiatives [1] 4/9 inject [1] 139/5 injectables [1] 122/18 injecting [1] 121/12 injection [2] 110/18 110/20 inoculum [7] 16/18 16/23 17/2 18/12 176/3 176/5 181/8 Inquiry [16] 1/25 3/20 4/11 6/1 6/3 6/4 37/20 50/14 66/17 91/16 91/17 95/11 124/2 124/12 149/11 178/17 inserted [1] 144/12 inserting [2] 92/19 112/13 inside [2] 70/18 126/22 insofar [1] 155/24 instance [2] 147/15 152/4 institution [1] 2/24 instruction [3] 6/1 6/6 157/10 insurance [1] 85/12 intake [1] 106/21 integrase [10] 104/15 104/16 108/8 113/21 113/22 141/12 141/21 143/7 144/8 144/10 integrate [2] 141/21 175/18 integrated [4] 140/20 140/25 171/25 172/1 integrates [1] 174/13 integration [4] 140/18 141/1 143/6 145/11 intercourse [4] 11/17 178/21 179/7 180/14 interest [3] 60/14 136/7 178/16 interested [2] 5/16 5/19 interesting [1] 169/7 interestingly [1] 20/17 interests [2] 84/5 110/11 interface [2] 130/9 130/10 interferon [2] 88/12	88/13 interlude [1] 110/13 international [2] 32/21 33/4 internationally [1] 31/16 internet [2] 80/6 158/15 interplay [1] 172/18 interrelate [1] 136/19 intervention [1] 50/7 interviewing [1] 82/15 intimate [1] 151/20 into [60] 6/25 7/18 10/22 12/9 17/4 17/24 24/4 29/21 30/17 33/15 33/18 35/20 36/2 36/22 38/12 51/1 51/8 58/15 58/16 62/17 62/17 68/8 77/21 79/5 82/20 85/1 86/23 92/19 108/24 127/1 128/15 130/21 134/20 135/13 135/15 138/9 138/14 138/22 139/5 140/3 140/11 140/21 140/25 141/22 141/22 142/21 143/1 151/8 151/24 159/2 172/3 173/9 173/9 174/13 175/18 176/4 178/13 181/6 184/18 192/3 intracellular [1] 48/11 intractable [2] 55/9 56/3 intravenous [1] 9/7 introduce [1] 1/23 introduced [3] 13/18 101/12 101/13 introduction [1] 110/3 invasive [2] 53/12 74/7 investigate [1] 63/11 investigating [1] 91/17 investigations [1] 163/25 invite [1] 64/23 involved [7] 4/8 4/10 4/17 4/20 59/7 81/14 146/4 ish [1] 177/9 isn't [6] 55/21 81/8 100/14 121/10 121/14 127/3 isolated [6] 4/10 77/13 77/24 78/10 80/10 101/4 issue [18] 11/10 25/18 58/23 60/11 76/10 77/3 81/2 81/11	84/25 85/5 90/10 97/6 100/8 111/14 129/7 130/8 136/5 181/8 issues [21] 55/10 72/8 88/9 113/9 115/25 116/25 124/5 124/15 124/23 132/12 135/2 135/7 136/1 136/3 151/25 153/23 157/8 162/4 162/10 169/21 180/7 it's [173] 7/2 7/20 8/12 9/2 9/5 11/9 13/5 13/23 15/5 20/6 21/15 22/7 22/14 24/21 24/23 24/25 26/7 27/23 29/3 29/5 29/19 30/4 32/22 32/23 33/1 33/17 38/22 39/1 43/22 43/24 44/3 46/8 48/17 52/24 54/5 54/23 54/25 55/1 55/14 55/24 56/12 57/2 57/6 57/12 58/5 60/25 61/13 62/12 62/14 62/15 63/21 63/22 64/10 67/16 67/16 67/24 69/11 70/5 70/8 70/11 71/20 73/21 73/25 74/22 76/1 76/10 81/5 81/10 81/11 83/21 83/25 87/23 88/3 88/9 92/13 95/10 97/15 98/18 100/13 102/2 102/11 102/13 103/13 105/16 110/12 117/6 117/19 119/3 119/5 119/6 119/9 119/15 119/17 120/11 120/11 120/14 120/15 120/15 121/9 121/17 122/15 128/19 129/24 130/13 132/24 132/24 133/14 133/15 134/24 135/10 136/12 137/10 138/13 138/18 138/20 139/24 140/6 140/8 140/8 140/9 140/9 140/18 141/6 141/9 142/25 143/23 145/22 146/1 146/4 146/10 149/16 152/5 152/10 152/11 152/23 153/12 153/13 154/20 157/3 161/1 164/20 167/21 170/6 171/7 172/12 173/18 174/12 175/19 176/3 176/5 176/10 176/23 176/24 176/25 177/2 177/20 178/6 178/7 179/5 179/21 181/14 183/16 183/21 184/8 186/21	186/22 187/3 189/8 190/2 190/25 190/25 195/8 195/25 items [4] 178/20 179/16 179/18 180/15 iteration [1] 83/12 its [15] 6/24 72/10 81/1 102/23 105/20 107/1 120/16 133/10 137/22 138/25 140/15 140/15 140/17 140/17 189/3 itself [25] 10/6 13/17 28/9 45/3 45/12 47/4 49/20 51/25 56/15 62/13 65/11 70/25 74/6 81/19 92/2 92/4 94/17 101/11 118/12 118/20 138/3 138/9 140/1 144/14 145/21
J					
	James [1] 121/24 JANE [7] 1/19 24/11 67/15 85/25 102/18 171/6 192/8 Jefferey [1] 23/9 jigsaw [1] 164/5 job [3] 18/24 86/9 137/22 jog [1] 20/22 Johnson [1] 186/1 JOHNSTON [3] 1/17 4/4 4/6 join [1] 2/14 joined [1] 171/24 joining [1] 112/8 joint [1] 181/18 joints [1] 19/17 journeys [1] 157/1 juice [2] 108/20 108/23 junior [2] 5/8 112/9 just [95] 10/7 11/11 11/18 12/13 15/4 17/11 21/24 23/8 23/22 28/5 29/23 33/23 34/17 37/7 39/16 39/21 44/19 45/22 46/3 47/25 49/8 59/1 64/23 67/15 67/24 71/15 75/13 75/21 77/2 78/5 78/14 79/25 81/23 84/2 85/17 85/21 86/5 86/7 87/11 87/14 90/24 91/3 95/22 96/2 97/7 100/10 104/24 106/10 106/13 108/11 108/24 109/16 110/8 111/1 113/7 116/4 120/1 120/15 121/19 123/20 123/25 126/10 131/5				



<b>J</b>	129/13 130/5 131/12 131/16 132/2 132/4 132/22 133/1 133/6 136/11 143/23 148/18 151/6 151/7 151/11 152/5 152/7 152/11 153/7 153/12 155/1 155/1 157/4 157/9 161/15 162/23 163/1 163/17 164/17 166/5 166/25 171/7 171/10 173/18 174/21 175/3 179/20 180/11 180/17 181/17 185/2 185/22 190/11 192/10 194/23 195/2 196/2	<b>layer</b> [2] 51/10 61/10 <b>lead</b> [2] 3/1 60/4 <b>leading</b> [1] 73/1 <b>leads</b> [1] 187/23 <b>learn</b> [2] 153/18 190/2 <b>learned</b> [2] 129/4 151/16 <b>learning</b> [5] 154/24 174/25 189/24 195/22 195/25 <b>learnt</b> [2] 98/2 110/2 <b>least</b> [4] 22/25 146/17 167/19 175/12 <b>leave</b> [4] 44/16 56/4 97/22 165/8 <b>led</b> [2] 83/19 186/14 <b>left</b> [4] 73/13 78/13 171/15 186/25 <b>lend</b> [1] 70/24 <b>length</b> [1] 88/11 <b>lesion</b> [1] 70/18 <b>lesions</b> [1] 62/16 <b>less</b> [22] 24/25 30/8 35/10 36/7 36/10 50/13 57/10 62/2 62/13 88/17 101/14 112/25 121/7 129/14 129/15 168/9 174/24 184/19 189/1 190/5 193/23 195/6 <b>let</b> [3] 2/6 80/2 136/25 <b>let's</b> [3] 109/21 151/8 167/10 <b>letter</b> [5] 6/6 112/22 157/10 157/18 171/20 <b>letters</b> [2] 6/1 168/16 <b>letting</b> [1] 151/10 <b>leukoencephalopathy</b> [1] 67/2 <b>leukoplakia</b> [1] 40/2 <b>level</b> [23] 31/2 31/5 31/6 35/8 53/4 61/2 73/21 94/21 95/6 101/11 108/14 108/15 108/24 114/14 115/11 118/11 118/24 119/2 120/3 120/8 121/11 171/17 176/12 <b>levels</b> [5] 18/14 19/3 47/14 65/24 66/13 <b>liaison</b> [1] 168/16 <b>licenced</b> [1] 93/18 <b>licensed</b> [1] 94/3 <b>lie</b> [1] 163/7 <b>life</b> [39] 3/23 22/4 46/1 47/15 56/25 73/16 73/19 74/25 76/14 82/7 85/12 86/6 96/20 96/20 103/6 115/25 123/12 123/15 123/24 125/5 126/8 130/20 130/21 130/24 131/3 131/13 131/20	131/25 132/23 133/16 133/22 134/1 134/6 136/18 164/20 171/9 179/15 184/9 184/17 <b>life-long</b> [1] 22/4 <b>life-shortening</b> [1] 74/25 <b>lifelong</b> [2] 119/20 119/21 <b>lifestyle</b> [4] 62/9 72/8 109/3 110/5 <b>light</b> [1] 19/21 <b>Lighthouse</b> [2] 59/6 59/11 <b>like</b> [85] 5/22 8/8 11/11 13/9 17/17 19/9 19/10 19/10 19/25 19/25 20/10 20/11 20/20 25/16 26/22 27/14 32/6 37/11 38/25 39/4 39/13 39/25 40/9 41/1 41/11 41/13 41/16 44/14 45/4 48/6 52/7 57/24 65/3 67/8 67/22 67/23 71/23 72/24 76/5 82/8 82/16 85/13 86/16 86/21 88/1 92/22 97/3 99/21 100/16 100/16 100/19 102/11 105/16 107/19 107/22 107/22 110/8 113/24 118/16 122/3 124/16 126/1 126/13 127/18 127/25 138/8 142/17 142/25 143/3 143/23 144/3 144/6 145/13 145/23 157/7 162/5 164/9 167/3 167/6 170/12 181/16 181/19 188/2 188/11 196/2 <b>likelihood</b> [2] 10/12 135/7 <b>likely</b> [8] 12/7 18/15 26/11 30/8 70/19 88/17 90/7 123/8 <b>limit</b> [1] 120/16 <b>limitation</b> [1] 50/6 <b>limited</b> [5] 7/18 40/22 81/4 102/10 134/9 <b>line</b> [9] 25/9 36/16 110/10 110/16 113/1 128/11 160/2 167/22 169/15 <b>lines</b> [1] 101/2 <b>lining</b> [1] 42/19 <b>link</b> [3] 72/2 183/8 188/25 <b>linked</b> [1] 69/9 <b>lipoatrophy</b> [6] 95/18 99/12 99/18 99/20 100/2 100/7 <b>list</b> [6] 41/8 48/22	52/21 64/24 156/11 156/13 <b>listed</b> [2] 33/9 97/3 <b>listening</b> [2] 55/15 91/2 <b>lists</b> [2] 97/16 156/12 <b>litre</b> [2] 11/13 11/18 <b>little</b> [18] 15/10 16/9 34/23 45/22 69/10 80/4 97/20 100/23 110/10 130/5 134/19 137/16 137/24 139/8 144/6 156/11 160/1 192/17 <b>live</b> [7] 74/17 116/18 132/8 142/4 171/3 180/23 181/5 <b>lived</b> [10] 19/17 46/24 48/3 77/13 93/25 96/20 134/23 150/17 161/6 196/11 <b>liver</b> [6] 72/22 89/1 127/19 127/20 182/24 183/1 <b>lives</b> [10] 38/20 46/13 46/16 47/11 49/2 75/18 75/20 109/20 116/18 127/9 <b>living</b> [36] 2/23 5/7 8/10 13/7 13/10 13/11 15/3 43/4 46/10 72/5 82/5 82/25 83/4 122/13 123/1 123/22 126/3 126/12 129/9 132/18 132/18 132/22 133/6 133/7 133/15 133/16 134/21 136/14 151/18 161/5 170/16 178/25 193/16 193/20 194/5 194/14 <b>load</b> [42] 12/25 13/2 16/5 16/10 16/13 18/10 18/14 19/6 22/16 24/24 25/1 25/14 28/11 28/15 28/19 33/24 34/8 34/10 36/11 102/4 102/7 105/14 105/23 117/15 119/4 119/9 119/12 121/3 121/5 121/9 123/24 152/23 163/24 165/17 167/8 168/9 172/25 175/7 179/1 179/21 179/23 180/4 <b>load's</b> [1] 168/8 <b>loads</b> [2] 36/12 115/20 <b>local</b> [2] 157/3 158/3 <b>location</b> [1] 177/15 <b>London</b> [13] 3/12 3/18 4/22 5/18 54/8 54/12 59/6 59/11 81/15 82/6	173/15 191/6 191/8 <b>long</b> [46] 22/4 31/1 57/13 61/8 65/11 67/9 67/12 72/12 76/17 83/23 89/4 96/23 98/15 110/23 112/2 117/21 118/3 118/7 123/2 123/19 123/22 125/21 126/23 127/5 127/8 127/15 127/19 127/23 128/1 129/10 129/25 131/7 132/6 133/10 133/11 135/11 136/16 151/24 180/23 193/16 193/19 193/20 193/24 194/4 194/6 194/22 <b>long-lasting</b> [1] 96/23 <b>long-standing</b> [1] 127/15 <b>long-term</b> [20] 57/13 61/8 67/9 67/12 72/12 83/23 127/5 127/19 127/23 128/1 131/7 133/11 135/11 136/16 193/16 193/19 193/20 194/4 194/6 194/22 <b>longer</b> [19] 30/10 37/1 83/4 99/4 103/15 103/16 113/2 122/17 131/9 132/10 132/18 132/19 133/8 133/15 133/17 143/16 147/17 173/10 189/8 <b>longest</b> [1] 83/1 <b>look</b> [23] 10/8 47/12 50/11 50/18 62/19 64/23 73/4 100/18 105/19 109/18 119/19 126/14 126/18 126/19 131/22 137/9 152/22 164/9 167/5 188/18 188/18 191/13 194/13 <b>looked</b> [15] 2/8 14/16 58/19 65/23 81/14 93/7 97/18 100/15 101/17 105/23 114/6 114/20 131/21 176/11 178/5 <b>looking</b> [19] 3/22 27/21 31/14 39/11 45/14 76/20 105/1 105/5 106/10 115/3 120/5 122/17 133/4 134/11 155/23 176/4 180/15 191/4 194/10 <b>looks</b> [3] 43/22 43/23 157/7 <b>loops</b> [1] 144/14 <b>lopinavir</b> [2] 107/20 107/24 <b>lose</b> [3] 53/15 100/1 175/7
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<b>L</b>	<b>maintaining [1]</b> 119/16 <b>major [4]</b> 7/8 35/1 50/17 99/4 <b>majority [2]</b> 3/2 160/23 <b>make [15]</b> 26/19 28/4 51/9 57/25 75/9 77/21 86/13 90/25 109/10 111/1 111/17 150/4 157/1 179/1 184/20 <b>makes [6]</b> 13/17 28/2 34/23 35/10 65/24 140/18 <b>making [9]</b> 49/9 49/15 49/23 109/13 109/16 126/2 126/19 171/9 184/22 <b>malaise [1]</b> 95/17 <b>Malawi [1]</b> 192/21 <b>mammogram [1]</b> 87/11 <b>manage [5]</b> 61/6 98/4 112/18 122/4 153/19 <b>managed [2]</b> 58/20 121/22 <b>management [1]</b> 121/20 <b>managing [3]</b> 88/2 96/13 109/6 <b>manifestation [1]</b> 60/5 <b>manufacturing [1]</b> 92/10 <b>many [37]</b> 3/22 20/10 37/5 40/7 43/20 46/13 46/16 50/1 50/3 51/24 51/24 52/9 56/24 57/2 59/2 59/21 60/22 62/20 67/17 69/15 76/2 77/12 80/16 86/12 86/14 97/6 111/4 127/15 127/15 127/17 132/22 142/5 150/18 155/3 177/15 184/12 184/25 <b>mapped [1]</b> 191/11 <b>marked [2]</b> 64/21 94/19 <b>marker [1]</b> 40/15 <b>markers [2]</b> 27/4 105/13 <b>market [3]</b> 96/13 106/15 164/14 <b>marks [2]</b> 24/4 25/15 <b>marrow [1]</b> 174/2 <b>Mary's [3]</b> 2/9 5/4 5/6 <b>material [3]</b> 91/18 141/5 180/19 <b>maternal [1]</b> 12/25 <b>matters [1]</b> 38/9 <b>mature [2]</b> 145/5 173/9	<b>may [78]</b> 1/13 9/25 10/5 10/6 10/25 11/3 12/2 12/15 13/20 14/23 15/20 15/22 19/20 20/12 20/24 21/2 25/21 28/7 28/9 28/10 28/15 29/12 34/4 34/5 35/9 35/13 39/11 39/12 46/16 47/2 59/6 67/15 68/17 69/20 69/23 72/6 72/7 79/1 80/1 80/20 83/16 85/18 98/10 98/16 104/2 117/1 117/3 117/3 117/5 117/11 121/24 122/18 125/7 128/11 131/5 131/11 135/16 135/18 143/4 143/4 146/24 156/14 158/3 158/24 159/19 159/21 162/19 164/17 164/21 165/10 166/10 169/8 169/11 172/24 181/15 188/17 193/25 196/18 <b>maybe [17]</b> 10/8 11/3 40/10 48/2 75/24 79/8 100/22 101/18 111/2 111/23 114/10 117/4 122/1 122/5 155/5 158/20 169/4 <b>me [13]</b> 2/6 35/19 75/14 127/23 129/21 137/8 149/2 157/21 187/7 193/25 195/16 195/19 195/25 <b>mean [36]</b> 6/24 11/23 12/16 19/23 21/10 28/24 35/3 51/19 64/12 70/24 78/19 82/22 97/11 97/13 99/1 101/14 110/22 112/6 119/7 134/9 150/10 152/17 157/24 164/13 165/15 168/7 168/24 169/3 176/14 176/21 177/2 181/1 185/22 186/7 186/19 192/14 <b>meaning [2]</b> 153/6 186/17 <b>means [15]</b> 7/25 23/21 30/6 37/7 44/24 80/23 117/17 117/23 119/6 138/2 146/18 148/16 152/24 164/24 176/20 <b>meant [3]</b> 14/13 56/3 79/13 <b>measure [1]</b> 105/4 <b>measured [1]</b> 34/9 <b>meat [2]</b> 47/11 47/13 <b>mechanism [2]</b> 28/12	158/25 <b>medical [7]</b> 20/12 56/2 56/6 65/14 91/11 159/12 195/2 <b>medication [23]</b> 35/9 52/13 58/20 61/10 74/9 74/10 88/6 89/19 89/21 96/1 102/14 106/14 116/21 123/4 126/4 128/1 130/1 130/19 161/11 164/8 167/20 183/4 183/7 <b>medications [20]</b> 52/14 52/18 58/8 66/11 72/9 88/20 88/20 89/10 89/13 95/22 98/22 106/9 106/11 106/14 113/14 114/13 114/21 117/4 118/11 129/23 <b>medicine [5]</b> 4/19 5/10 79/24 97/24 153/1 <b>medicines [1]</b> 112/16 <b>medics [2]</b> 157/17 185/6 <b>meet [7]</b> 20/18 45/17 159/20 160/10 160/14 161/4 162/16 <b>meeting [4]</b> 22/5 83/24 159/24 160/22 <b>MEI [1]</b> 68/21 <b>Melbourne [1]</b> 4/3 <b>members [4]</b> 4/1 52/3 78/6 163/14 <b>membrane [1]</b> 139/4 <b>membranes [1]</b> 60/20 <b>memories [4]</b> 44/6 44/7 55/16 55/20 <b>memory [5]</b> 20/22 60/7 96/3 108/19 110/17 <b>men [2]</b> 178/8 178/8 <b>meningitis [11]</b> 21/3 21/13 21/15 47/25 48/2 53/1 53/1 53/2 53/9 54/16 69/18 <b>menstruation [2]</b> 178/19 179/13 <b>mental [18]</b> 76/20 76/25 78/3 78/20 79/8 79/14 80/14 80/19 80/22 80/25 81/4 116/3 132/16 135/2 135/7 136/1 136/2 136/13 <b>mentally [1]</b> 125/17 <b>mention [2]</b> 27/2 29/10 <b>mentioned [11]</b> 9/15 26/2 44/22 61/24 77/5 86/3 107/14 126/21 127/13 138/17 175/4	<b>mentioning [3]</b> 33/23 100/10 181/14 <b>message [1]</b> 139/12 <b>messages [1]</b> 187/8 <b>messenger [2]</b> 145/2 145/3 <b>met [7]</b> 1/5 5/6 47/7 47/14 47/18 48/19 167/25 <b>metabolic [1]</b> 72/1 <b>microbacterium [1]</b> 48/11 <b>microbes [1]</b> 46/13 <b>microbial [2]</b> 45/25 47/17 <b>microinfusion [1]</b> 9/8 <b>mid [5]</b> 93/6 106/18 107/10 108/8 114/6 <b>mid-1987 [1]</b> 93/6 <b>mid-2000s [4]</b> 106/18 107/10 108/8 114/6 <b>middle [5]</b> 33/5 45/11 96/17 122/7 137/19 <b>Middlesex [1]</b> 4/21 <b>might [53]</b> 10/9 11/4 15/14 19/25 20/21 35/6 39/9 46/23 46/23 48/3 48/19 51/16 53/25 54/3 58/12 61/8 66/9 66/12 68/15 68/16 68/19 77/20 77/21 79/6 85/22 88/19 98/24 102/12 122/3 123/7 124/24 125/24 130/3 142/4 145/8 154/16 155/7 159/3 161/20 163/13 164/2 164/22 165/18 169/10 170/16 170/25 172/13 172/15 172/16 187/17 187/18 191/21 196/2 <b>migration [1]</b> 5/20 <b>mild [1]</b> 124/19 <b>milder [1]</b> 60/12 <b>milk [4]</b> 9/5 9/10 11/13 11/18 <b>millions [1]</b> 18/16 <b>minor [1]</b> 169/10 <b>minority [3]</b> 5/20 12/3 98/13 <b>minus [1]</b> 158/6 <b>minutes [1]</b> 181/1 <b>misconception [1]</b> 14/8 <b>misnomer [1]</b> 3/9 <b>missed [1]</b> 9/10 <b>missing [1]</b> 84/22 <b>mitochondria [1]</b> 99/23 <b>mitochondrial [1]</b> 99/22 <b>mixture [1]</b> 164/25	<b>ml [1]</b> 120/8 <b>model [3]</b> 133/3 135/14 171/17 <b>models [2]</b> 131/19 133/3 <b>modern [9]</b> 23/2 97/24 116/5 122/25 123/23 124/3 124/9 127/24 130/24 <b>modes [1]</b> 9/6 <b>modified [1]</b> 192/17 <b>modify [1]</b> 32/15 <b>modulate [1]</b> 187/18 <b>molecular [1]</b> 146/19 <b>molecule [2]</b> 29/19 138/25 <b>molecules [3]</b> 8/3 147/19 148/8 <b>moment [17]</b> 8/11 42/2 119/20 122/16 122/19 129/14 132/20 135/14 146/23 148/22 154/25 169/24 174/4 174/6 174/17 186/13 194/8 <b>money [1]</b> 170/14 <b>monitor [6]</b> 34/8 34/12 105/6 115/15 119/8 183/17 <b>monitored [3]</b> 34/6 124/9 183/19 <b>monitoring [1]</b> 174/20 <b>mono [4]</b> 81/7 92/6 93/21 126/24 <b>mono-infection [1]</b> 81/7 <b>mono-therapies [1]</b> 92/6 <b>mono-therapy [2]</b> 93/21 126/24 <b>month [4]</b> 25/24 25/25 37/15 169/1 <b>monthly [1]</b> 168/11 <b>months [8]</b> 19/6 22/22 22/25 24/3 25/10 25/13 93/10 101/5 <b>morbidities [6]</b> 71/6 116/2 123/6 134/16 134/25 192/24 <b>morbidity [1]</b> 119/3 <b>more [114]</b> 7/20 12/5 12/9 13/13 14/22 15/12 15/20 20/7 20/20 21/8 23/2 23/6 24/23 26/16 26/19 28/10 31/2 32/23 35/22 41/2 41/5 42/5 42/21 43/3 43/4 46/24 48/8 49/25 51/7 52/16 57/9 60/5 63/23 63/25 68/14 71/7 71/8 72/5 72/12 72/23 72/25 73/14 75/25 77/22
<b>M</b>	<b>machine [1]</b> 187/13 <b>made [15]</b> 1/9 32/12 34/18 41/20 83/9 93/5 94/6 100/1 103/5 103/11 106/4 109/24 113/15 152/18 196/14 <b>main [3]</b> 7/4 7/16 118/6 <b>mainly [1]</b> 116/13 <b>maintained [1]</b> 106/6				

<b>M</b>	167/17 moved [4] 5/14 33/15 105/21 122/2 moving [4] 24/4 25/24 92/1 92/3 Mr [1] 173/14 Mr Brown [1] 173/14 MRI [1] 70/17 Ms [6] 1/20 1/21 3/6 43/16 90/22 148/19 Ms Edwards [1] 43/16 Ms Scott [2] 90/22 148/19 much [77] 7/20 9/22 10/22 12/24 12/25 14/22 16/14 16/23 19/25 20/5 23/4 25/1 26/11 26/19 30/10 31/2 32/23 36/18 46/24 47/6 47/14 48/12 48/15 50/13 57/10 58/3 60/1 62/2 62/6 62/13 63/12 63/23 63/25 64/11 64/16 65/13 68/13 71/7 73/14 93/10 98/3 108/10 116/9 116/22 116/24 119/18 120/6 122/3 123/9 123/10 124/4 124/11 124/13 124/15 130/25 133/8 133/17 133/17 146/4 146/5 148/17 150/11 151/23 151/23 152/2 153/2 153/2 156/23 169/12 171/9 172/13 177/14 178/7 179/10 186/23 195/24 196/2 mucus [1] 60/19 multidisciplinary [2] 160/25 162/14 multifocal [1] 67/1 multiple [7] 68/1 68/1 69/13 103/4 162/20 164/14 164/15 murderers [1] 186/19 muscles [1] 19/16 must [2] 131/8 196/1 mustn't [1] 153/10 mutate [1] 117/25 mutating [3] 101/8 102/5 145/17 mutation [1] 11/4 mutations [1] 101/13 my [27] 2/25 3/2 3/7 4/5 4/16 4/25 5/8 37/9 37/12 44/6 63/2 67/6 71/11 79/22 87/13 96/3 110/17 121/23 126/14 132/14 137/14 150/11 169/3 186/11 187/7 187/8 196/5 myalgia [1] 95/17	<b>N</b> naive [1] 117/12 name [11] 2/2 3/7 4/5 4/16 151/11 152/9 152/12 153/5 153/11 153/13 183/12 narrowing [1] 23/15 nasty [1] 53/1 nation [1] 192/9 national [8] 5/23 5/24 75/7 158/22 159/5 159/15 164/15 192/5 nationally [1] 169/22 natural [12] 7/10 7/20 16/20 22/8 25/3 25/6 27/20 30/3 30/9 87/22 176/17 192/17 naturally [2] 12/4 12/16 nature [1] 16/6 nausea [4] 19/18 67/22 95/16 104/6 near [2] 75/22 171/2 nearest [1] 170/8 nearly [1] 168/14 neatly [2] 79/4 90/17 necessarily [13] 14/24 15/22 28/11 39/12 65/12 71/18 109/1 129/13 147/4 160/9 165/9 166/3 175/9 need [31] 45/18 47/6 56/17 68/18 80/23 81/1 94/4 99/19 103/20 118/9 118/10 122/10 122/19 124/9 125/4 128/1 130/3 141/24 142/16 145/4 145/16 145/25 155/5 159/2 162/15 162/19 168/13 170/17 170/21 190/2 191/20 needed [8] 41/6 58/19 88/15 94/16 114/25 117/11 174/1 187/1 needs [12] 29/15 29/22 115/24 117/20 141/12 148/12 148/14 158/25 161/24 165/25 171/23 177/23 negative [8] 23/23 25/22 64/20 73/10 73/16 74/4 76/23 125/12 negotiating [1] 151/20 nervous [4] 187/15 188/1 188/2 188/3 net [1] 137/20 neural [1] 124/18 Neurological [1] 19/19	neurones [2] 188/7 188/21 Neurons [1] 188/5 neuropathic [1] 44/17 neuropathy [10] 41/17 43/10 94/21 94/22 97/2 127/13 127/14 188/8 188/9 188/10 neurotropic [1] 22/2 neutropaenia [1] 95/15 never [1] 87/1 nevirapine [3] 104/12 107/3 107/6 new [23] 77/22 100/6 105/5 107/11 110/14 112/8 113/17 124/21 128/24 128/24 140/16 141/4 141/13 145/1 145/2 147/13 147/19 154/17 159/6 161/3 163/21 169/7 173/9 New-Fill [1] 100/6 newborn [1] 97/14 newer [3] 107/10 107/14 128/8 newest [1] 129/5 newly [4] 20/19 122/24 125/3 130/23 next [9] 42/3 54/17 63/13 71/11 77/18 131/2 137/5 163/3 164/20 NHS [1] 192/6 night [6] 39/20 41/14 44/7 44/8 44/9 96/17 nights [1] 153/14 no [60] 6/13 8/11 8/18 9/12 20/14 21/10 28/2 33/7 33/12 33/13 37/3 37/4 37/6 51/23 63/1 64/20 76/8 87/9 97/24 99/4 103/15 103/16 104/4 109/20 116/11 118/2 118/3 131/8 132/1 132/10 143/16 144/21 144/22 147/17 147/21 149/19 149/20 149/24 151/4 151/4 151/4 151/6 151/6 151/6 153/10 166/7 170/2 173/10 176/18 179/2 179/19 179/20 180/16 188/22 189/8 190/18 195/17 195/17 195/19 196/1 no-one [2] 51/23 131/8 nobody [1] 170/4 nodding [1] 138/4 nodes [1] 19/16 non [14] 4/13 39/17	39/22 40/4 72/24 104/13 135/6 142/11 142/13 143/8 143/11 144/1 144/3 185/25 non-AIDS [1] 72/24 non-HIV [1] 135/6 non-nucleoside [4] 104/13 142/11 143/8 143/11 non-nucleosides [3] 144/1 144/3 185/25 non-specific [1] 40/4 nonetheless [5] 60/13 62/2 124/20 162/18 179/11 norm [5] 189/4 189/4 189/6 189/7 189/8 normal [2] 116/18 165/7 normally [5] 48/11 99/21 163/19 165/5 168/4 north [2] 81/15 82/6 north-east [1] 82/6 nosebleeds [1] 181/19 not [161] 8/4 8/18 10/3 10/6 10/14 10/18 11/14 12/16 13/5 13/9 14/14 16/19 20/12 23/4 24/21 27/23 28/3 28/10 28/18 29/1 30/5 30/6 34/4 35/14 39/11 39/16 39/19 39/22 40/13 40/15 42/23 42/25 43/14 44/3 44/4 44/19 53/21 60/1 60/10 62/3 62/5 62/24 65/12 65/16 66/9 67/16 70/13 70/24 71/3 71/15 73/2 76/8 77/15 78/14 79/25 79/25 80/1 80/20 85/15 87/5 89/19 89/20 89/25 91/7 91/10 91/20 92/24 94/8 97/15 101/11 104/16 106/5 109/16 110/16 111/4 113/2 113/20 114/22 117/20 118/1 118/5 119/2 119/6 119/9 119/14 119/15 119/17 120/15 121/19 123/18 124/4 124/11 124/13 124/20 125/8 128/12 128/23 129/9 133/14 136/2 136/14 140/6 140/6 142/1 143/4 144/5 144/15 144/19 146/19 147/4 149/13 149/16 151/1 151/5 151/7 152/10 152/24 153/5	153/12 155/11 160/3 161/1 161/2 161/5 161/16 164/2 165/6 165/9 165/10 166/3 166/10 167/14 169/11 170/17 171/17 172/7 172/25 174/6 175/3 175/9 176/10 176/16 178/4 179/12 180/20 182/6 182/25 183/10 183/21 183/24 185/3 185/5 185/20 185/21 187/8 187/11 188/5 191/11 194/1 194/12 194/24 note [1] 136/20 notes [3] 84/12 84/12 85/11 nothing [1] 155/1 notice [2] 39/9 39/12 noticed [1] 39/4 noting [3] 25/17 35/6 67/15 now [79] 2/17 3/3 15/19 17/5 23/2 23/4 23/14 32/9 32/13 40/7 43/1 43/8 45/2 46/12 47/10 47/23 50/9 50/11 52/14 52/20 58/3 61/19 64/18 67/6 71/10 71/12 71/15 71/19 72/13 73/1 74/12 74/21 76/20 77/18 79/22 91/13 92/22 92/23 93/23 94/3 96/25 98/4 100/5 101/9 102/2 102/19 103/7 107/2 114/3 115/15 120/20 121/13 122/1 122/5 123/8 123/12 123/14 123/23 132/14 133/5 133/9 133/24 139/10 147/6 147/19 148/4 155/10 155/12 157/7 159/18 160/24 161/15 164/14 165/12 171/22 174/24 179/23 189/15 194/16 nowadays [1] 158/14 NRTIs [1] 104/19 nucleic [4] 142/17 142/18 143/16 148/15 nucleoside [12] 92/15 94/10 104/13 104/23 104/24 107/11 113/24 142/11 142/11 142/13 143/8 143/11 nucleosides [4] 144/1 144/3 144/6 185/25 nucleus [6] 137/19 138/7 140/11 140/16 142/3 144/11 number [16] 4/9 9/23
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<b>N</b>	66/12 67/20 73/17 74/17 75/15 77/13 78/7 78/10 79/18 80/3 81/7 83/2 103/4 112/6 112/13 116/24 124/19 128/13 129/1 150/19 150/20 151/1 156/9 156/11 156/16 160/13 160/16 164/21 166/22 166/24 167/22 168/9 181/9 181/18 182/24 187/1 190/3 <b>oh</b> [6] 37/13 39/10 87/10 143/7 153/10 169/12 <b>okay</b> [6] 54/14 76/8 83/13 112/1 142/17 162/23 <b>old</b> [1] 37/15 <b>older</b> [17] 26/9 26/13 124/10 127/12 129/9 129/16 130/3 130/7 130/10 131/12 133/14 134/14 134/15 182/18 194/11 194/11 194/15 <b>olds</b> [1] 76/4 <b>on in</b> [1] 48/15 <b>once</b> [20] 1/4 15/16 30/16 33/18 43/9 48/13 102/15 114/3 119/4 132/5 140/24 147/16 154/2 164/7 167/4 167/8 168/5 168/9 169/1 177/24 <b>oncology</b> [1] 64/6 <b>one</b> [105] 3/18 9/15 11/16 12/23 13/4 15/12 15/20 16/2 29/24 29/25 31/15 31/17 31/24 31/24 33/8 34/19 35/3 38/24 44/3 44/4 44/5 45/19 46/23 47/9 51/23 53/4 53/16 54/17 54/23 55/21 57/8 59/16 60/25 61/6 63/14 65/6 65/8 65/18 65/18 67/16 69/6 70/15 71/20 77/2 78/1 78/17 79/15 81/15 85/8 87/21 93/17 100/8 103/18 103/20 103/25 104/2 107/25 110/8 111/18 113/7 115/24 116/14 116/17 117/20 118/8 124/17 124/25 128/4 128/4 131/8 136/19 137/11 137/17 138/2 143/24 145/24 146/10 146/14 146/21 146/24 148/15 155/11 157/6 157/9 162/17 162/17 164/18 166/2	166/4 168/18 171/4 171/15 173/1 174/3 180/22 182/24 183/11 183/13 187/7 187/21 194/9 194/19 194/21 195/4 196/7 <b>one's</b> [2] 78/3 167/4 <b>one-to-one</b> [1] 162/17 <b>onerous</b> [1] 157/1 <b>ones</b> [11] 7/9 29/24 38/15 41/5 60/25 69/7 79/25 106/16 131/21 134/15 169/20 <b>ongoing</b> [11] 30/19 31/8 66/20 72/18 102/7 121/16 124/15 155/2 167/7 177/25 194/7 <b>only</b> [27] 21/24 29/4 29/4 29/6 41/19 48/23 58/7 63/10 76/22 87/5 93/17 107/2 115/9 116/17 122/16 129/10 136/14 141/25 145/23 148/14 161/6 161/16 164/2 169/1 173/2 173/23 185/16 <b>onto</b> [2] 97/18 124/25 <b>onward</b> [1] 166/24 <b>open</b> [1] 171/1 <b>operating</b> [1] 100/5 <b>opportunist</b> [2] 68/22 166/10 <b>opportunistic</b> [7] 44/10 53/17 65/19 103/3 111/22 113/17 167/12 <b>opposed</b> [1] 18/16 <b>opposite</b> [1] 37/4 <b>optimist</b> [1] 135/21 <b>optimum</b> [2] 171/17 171/17 <b>options</b> [4] 58/7 124/21 126/16 127/2 <b>or</b> [128] 9/25 10/5 10/18 11/25 13/25 14/10 14/21 16/20 17/8 17/8 17/25 18/1 18/18 19/6 19/11 20/1 20/11 20/11 21/3 21/9 21/11 27/14 27/20 27/25 28/3 28/22 29/4 29/6 32/17 33/7 33/8 39/9 41/22 43/3 44/5 44/5 46/15 49/14 51/15 60/5 64/25 65/2 65/2 65/4 69/21 70/16 70/20 73/1 77/9 77/16 77/20 78/11 79/6 79/9 83/3 84/13 87/21 88/3 88/11 89/18 89/20 91/8 91/10 91/10 91/10 91/12 92/23	93/8 93/19 94/13 94/13 97/9 106/14 108/16 110/15 111/18 116/14 119/18 121/25 122/1 122/4 122/18 125/18 126/24 127/18 127/21 130/13 131/24 136/7 141/6 142/8 144/4 145/13 147/7 149/11 149/16 150/17 152/9 153/15 155/1 155/14 160/10 162/17 165/13 166/3 167/7 167/11 167/22 167/22 169/23 169/23 171/2 171/8 172/8 175/12 175/21 176/14 176/15 176/15 177/10 180/15 185/13 186/10 187/17 188/11 191/2 193/23 193/23 <b>oral</b> [6] 8/24 40/2 41/22 43/21 61/3 66/17 <b>orange</b> [1] 139/7 <b>order</b> [6] 29/21 45/17 96/14 108/23 145/5 162/9 <b>ordinated</b> [2] 171/24 194/17 <b>ordinating</b> [1] 24/13 <b>ordination</b> [1] 24/15 <b>organisms</b> [1] 46/2 <b>origin</b> [1] 62/11 <b>originally</b> [3] 16/2 93/5 106/24 <b>Ormond</b> [1] 186/15 <b>other</b> [90] 1/7 1/10 5/20 7/19 7/23 8/5 9/6 11/19 12/13 15/14 16/11 17/3 19/4 20/10 24/21 26/20 26/23 27/9 27/13 27/18 27/19 28/6 29/10 30/20 34/16 44/1 47/15 49/7 57/16 59/20 62/12 64/8 64/9 65/10 69/1 69/4 69/5 71/4 71/6 71/16 72/7 72/20 73/8 73/15 74/11 77/3 79/15 80/8 84/19 85/9 86/3 91/6 94/10 100/13 102/24 104/3 104/10 107/10 110/5 113/22 116/1 117/21 118/22 122/9 123/6 133/25 134/7 139/16 140/19 146/15 146/24 147/5 148/6 148/9 152/17 158/18 163/6 163/25 166/21 169/8 170/16 170/23 172/2 172/17 175/25	177/7 178/8 180/22 192/4 192/23 <b>other reasons</b> [1] 172/17 <b>others</b> [5] 76/16 82/22 139/2 163/14 166/24 <b>otherwise</b> [1] 184/6 <b>ought</b> [1] 37/15 <b>our</b> [21] 2/18 5/18 12/8 15/5 46/13 46/15 55/20 57/3 80/19 90/18 108/1 110/21 112/8 134/20 135/3 135/4 135/8 151/1 163/19 178/13 191/25 <b>ours</b> [2] 34/22 83/17 <b>out</b> [49] 4/20 4/23 9/11 14/5 25/14 35/23 37/15 38/9 39/21 41/25 43/16 45/15 48/7 49/18 55/17 56/4 66/15 74/5 76/7 82/3 82/3 87/1 94/5 95/13 102/12 105/23 106/13 108/22 110/14 113/4 129/14 129/18 131/6 137/24 138/3 139/15 145/20 148/19 151/1 157/8 158/10 158/18 158/24 160/19 160/20 165/22 169/2 169/3 189/18 <b>out-patient</b> [2] 4/20 4/23 <b>outcome</b> [11] 53/20 61/21 64/15 64/19 103/13 105/17 114/19 115/2 115/15 115/23 117/19 <b>outcomes</b> [5] 79/14 134/4 135/12 161/16 162/3 <b>outline</b> [2] 92/5 100/25 <b>outpatient</b> [1] 122/5 <b>outside</b> [11] 22/6 79/20 80/20 81/22 86/10 154/17 180/23 181/13 186/15 191/7 195/5 <b>outwards</b> [1] 76/6 <b>over</b> [22] 2/23 3/10 12/7 14/16 30/12 62/21 87/18 89/23 98/9 102/24 110/23 117/10 128/21 151/16 152/1 153/10 155/2 163/18 164/5 167/4 171/16 174/23 <b>overall</b> [3] 150/16 177/16 192/25 <b>overestimate</b> [2] 76/11 76/24	<b>overrepresented</b> [1] 11/6 <b>overspeaking</b> [1] 143/10 <b>own</b> [10] 21/5 21/9 75/18 91/24 104/24 121/23 135/3 150/21 151/1 167/1 <b>oxygen</b> [2] 58/1 65/24	
<b>O</b>	<b>o'clock</b> [3] 45/5 45/6 90/18 <b>obliged</b> [1] 58/9 <b>observe</b> [1] 69/5 <b>observed</b> [2] 69/2 69/6 <b>obvious</b> [1] 131/2 <b>obviously</b> [12] 12/17 75/1 78/20 79/10 89/25 123/18 124/23 150/11 156/7 159/18 165/12 185/3 <b>occasionally</b> [1] 61/15 <b>occur</b> [6] 19/19 30/18 63/24 71/16 118/7 124/6 <b>occurred</b> [2] 99/21 121/16 <b>occurring</b> [2] 66/8 118/22 <b>occurs</b> [2] 29/14 181/9 <b>oesophagus</b> [3] 51/1 51/11 57/22 <b>off</b> [5] 25/12 46/7 92/25 93/17 118/17 <b>off-the-shelf</b> [1] 92/25 <b>offer</b> [2] 126/18 150/10 <b>offered</b> [2] 50/7 115/13 <b>offering</b> [2] 156/15 158/11 <b>often</b> [70] 14/23 19/10 20/2 20/5 20/18 22/3 33/16 34/8 40/13 42/17 43/8 43/11 47/13 50/6 53/2 53/7 55/3 57/8 58/11 58/14 58/17 60/12 61/5 61/18 61/24 62/2 62/15 63/17 64/6 64/6 64/15 65/10 65/14	<b>old</b> [1] 37/15 <b>older</b> [17] 26/9 26/13 124/10 127/12 129/9 129/16 130/3 130/7 130/10 131/12 133/14 134/14 134/15 182/18 194/11 194/11 194/15 <b>olds</b> [1] 76/4 <b>on in</b> [1] 48/15 <b>once</b> [20] 1/4 15/16 30/16 33/18 43/9 48/13 102/15 114/3 119/4 132/5 140/24 147/16 154/2 164/7 167/4 167/8 168/5 168/9 169/1 177/24 <b>oncology</b> [1] 64/6 <b>one</b> [105] 3/18 9/15 11/16 12/23 13/4 15/12 15/20 16/2 29/24 29/25 31/15 31/17 31/24 31/24 33/8 34/19 35/3 38/24 44/3 44/4 44/5 45/19 46/23 47/9 51/23 53/4 53/16 54/17 54/23 55/21 57/8 59/16 60/25 61/6 63/14 65/6 65/8 65/18 65/18 67/16 69/6 70/15 71/20 77/2 78/1 78/17 79/15 81/15 85/8 87/21 93/17 100/8 103/18 103/20 103/25 104/2 107/25 110/8 111/18 113/7 115/24 116/14 116/17 117/20 118/8 124/17 124/25 128/4 128/4 131/8 136/19 137/11 137/17 138/2 143/24 145/24 146/10 146/14 146/21 146/24 148/15 155/11 157/6 157/9 162/17 162/17 164/18 166/2	166/4 168/18 171/4 171/15 173/1 174/3 180/22 182/24 183/11 183/13 187/7 187/21 194/9 194/19 194/21 195/4 196/7 <b>one's</b> [2] 78/3 167/4 <b>one-to-one</b> [1] 162/17 <b>onerous</b> [1] 157/1 <b>ones</b> [11] 7/9 29/24 38/15 41/5 60/25 69/7 79/25 106/16 131/21 134/15 169/20 <b>ongoing</b> [11] 30/19 31/8 66/20 72/18 102/7 121/16 124/15 155/2 167/7 177/25 194/7 <b>only</b> [27] 21/24 29/4 29/4 29/6 41/19 48/23 58/7 63/10 76/22 87/5 93/17 107/2 115/9 116/17 122/16 129/10 136/14 141/25 145/23 148/14 161/6 161/16 164/2 169/1 173/2 173/23 185/16 <b>onto</b> [2] 97/18 124/25 <b>onward</b> [1] 166/24 <b>open</b> [1] 171/1 <b>operating</b> [1] 100/5 <b>opportunist</b> [2] 68/22 166/10 <b>opportunistic</b> [7] 44/10 53/17 65/19 103/3 111/22 113/17 167/12 <b>opposed</b> [1] 18/16 <b>opposite</b> [1] 37/4 <b>optimist</b> [1] 135/21 <b>optimum</b> [2] 171/17 171/17 <b>options</b> [4] 58/7 124/21 126/16 127/2 <b>or</b> [128] 9/25 10/5 10/18 11/25 13/25 14/10 14/21 16/20 17/8 17/8 17/25 18/1 18/18 19/6 19/11 20/1 20/11 20/11 21/3 21/9 21/11 27/14 27/20 27/25 28/3 28/22 29/4 29/6 32/17 33/7 33/8 39/9 41/22 43/3 44/5 44/5 46/15 49/14 51/15 60/5 64/25 65/2 65/2 65/4 69/21 70/16 70/20 73/1 77/9 77/16 77/20 78/11 79/6 79/9 83/3 84/13 87/21 88/3 88/11 89/18 89/20 91/8 91/10 91/10 91/10 91/12 92/23	93/8 93/19 94/13 94/13 97/9 106/14 108/16 110/15 111/18 116/14 119/18 121/25 122/1 122/4 122/18 125/18 126/24 127/18 127/21 130/13 131/24 136/7 141/6 142/8 144/4 145/13 147/7 149/11 149/16 150/17 152/9 153/15 155/1 155/14 160/10 162/17 165/13 166/3 167/7 167/11 167/22 167/22 169/23 169/23 171/2 171/8 172/8 175/12 175/21 176/14 176/15 176/15 177/10 180/15 185/13 186/10 187/17 188/11 191/2 193/23 193/23 <b>oral</b> [6] 8/24 40/2 41/22 43/21 61/3 66/17 <b>orange</b> [1] 139/7 <b>order</b> [6] 29/21 45/17 96/14 108/23 145/5 162/9 <b>ordinated</b> [2] 171/24 194/17 <b>ordinating</b> [1] 24/13 <b>ordination</b> [1] 24/15 <b>organisms</b> [1] 46/2 <b>origin</b> [1] 62/11 <b>originally</b> [3] 16/2 93/5 106/24 <b>Ormond</b> [1] 186/15 <b>other</b> [90] 1/7 1/10 5/20 7/19 7/23 8/5 9/6 11/19 12/13 15/14 16/11 17/3 19/4 20/10 24/21 26/20 26/23 27/9 27/13 27/18 27/19 28/6 29/10 30/20 34/16 44/1 47/15 49/7 57/16 59/20 62/12 64/8 64/9 65/10 69/1 69/4 69/5 71/4 71/6 71/16 72/7 72/20 73/8 73/15 74/11 77/3 79/15 80/8 84/19 85/9 86/3 91/6 94/10 100/13 102/24 104/3 104/10 107/10 110/5 113/22 116/1 117/21 118/22 122/9 123/6 133/25 134/7 139/16 140/19 146/15 146/24 147/5 148/6 148/9 152/17 158/18 163/6 163/25 166/21 169/8 170/16 170/23 172/2 172/17 175/25	177/7 178/8 180/22 192/4 192/23 <b>other reasons</b> [1] 172/17 <b>others</b> [5] 76/16 82/22 139/2 163/14 166/24 <b>otherwise</b> [1] 184/6 <b>ought</b> [1] 37/15 <b>our</b> [21] 2/18 5/18 12/8 15/5 46/13 46/15 55/20 57/3 80/19 90/18 108/1 110/21 112/8 134/20 135/3 135/4 135/8 151/1 163/19 178/13 191/25 <b>ours</b> [2] 34/22 83/17 <b>out</b> [49] 4/20 4/23 9/11 14/5 25/14 35/23 37/15 38/9 39/21 41/25 43/16 45/15 48/7 49/18 55/17 56/4 66/15 74/5 76/7 82/3 82/3 87/1 94/5 95/13 102/12 105/23 106/13 108/22 110/14 113/4 129/14 129/18 131/6 137/24 138/3 139/15 145/20 148/19 151/1 157/8 158/10 158/18 158/24 160/19 160/20 165/22 169/2 169/3 189/18 <b>out-patient</b> [2] 4/20 4/23 <b>outcome</b> [11] 53/20 61/21 64/15 64/19 103/13 105/17 114/19 115/2 115/15 115/23 117/19 <b>outcomes</b> [5] 79/14 134/4 135/12 161/16 162/3 <b>outline</b> [2] 92/5 100/25 <b>outpatient</b> [1] 122/5 <b>outside</b> [11] 22/6 79/20 80/20 81/22 86/10 154/17 180/23 181/13 186/15 191/7 195/5 <b>outwards</b> [1] 76/6 <b>over</b> [22] 2/23 3/10 12/7 14/16 30/12 62/21 87/18 89/23 98/9 102/24 110/23 117/10 128/21 151/16 152/1 153/10 155/2 163/18 164/5 167/4 171/16 174/23 <b>overall</b> [3] 150/16 177/16 192/25 <b>overestimate</b> [2] 76/11 76/24	<b>overrepresented</b> [1] 11/6 <b>overspeaking</b> [1] 143/10 <b>own</b> [10] 21/5 21/9 75/18 91/24 104/24 121/23 135/3 150/21 151/1 167/1 <b>oxygen</b> [2] 58/1 65/24
<b>P</b>	<b>package</b> [1] 166/4 <b>Paddington</b> [1] 2/10 <b>paediatric</b> [2] 36/1 70/11 <b>paediatrician</b> [2] 2/7 11/12 <b>paediatricians</b> [2] 37/13 186/17 <b>page</b> [15] 19/12 31/12 42/1 42/3 42/6 49/6 49/22 49/22 69/3 69/11 95/7 95/10 137/10 149/9 154/7 <b>page 21</b> [1] 19/12 <b>page 25</b> [2] 69/3 69/11 <b>page 3</b> [1] 137/10 <b>page 33</b> [1] 49/6 <b>page 51</b> [1] 95/7 <b>page 6</b> [1] 31/12 <b>page 61</b> [2] 149/9 154/7 <b>pages</b> [1] 137/11 <b>pain</b> [9] 43/12 44/18 51/5 51/6 51/12 55/6 77/8 77/16 95/16 <b>painful</b> [6] 50/24 52/3 57/20 57/22 61/2 61/5 <b>pains</b> [1] 76/7 <b>pale</b> [1] 19/17 <b>pancreas</b> [1] 127/19 <b>pancreatic</b> [1] 127/21 <b>pandemic</b> [1] 71/7 <b>panel</b> [8] 1/4 81/24 91/1 129/21 136/23 174/9 193/10 193/13 <b>paragraph</b> [1] 69/11 <b>parallel</b> [1] 69/24 <b>parent</b> [2] 149/22 181/21 <b>parents</b> [6] 75/19 84/4 112/12 112/18 150/20 152/3 <b>park</b> [2] 54/8 54/12 <b>part</b> [14] 21/15 65/25 85/15 91/20 126/5 142/22 151/12 159/11 171/9 173/19 183/15 193/14 194/3 195/25 <b>participants</b> [3] 81/17 136/22 172/6 <b>particle</b> [9] 9/16					



<b>P</b>	4/23 16/2 31/7 33/2 34/4 39/11 46/1 76/21 84/24 86/15 89/18 97/5 103/24 106/8 128/15 128/18 149/18 155/17 157/16 159/14 168/17 171/7 173/13 176/21 178/9 180/20 187/18	<b>period</b> [13] 22/1 22/6 22/10 22/20 22/22 22/24 32/5 123/19 133/10 163/19 167/13 168/2 189/16 <b>periodically</b> [2] 114/10 114/13 <b>peripheral</b> [7] 41/17 43/10 97/2 127/13 127/14 188/2 188/9 <b>person</b> [20] 8/19 9/14 11/14 11/21 12/21 13/20 35/13 38/14 46/24 58/12 79/9 149/2 151/10 160/13 161/16 165/25 168/12 171/8 171/23 180/13 <b>person's</b> [2] 26/21 45/24 <b>person-centred</b> [1] 171/23 <b>personal</b> [1] 151/25 <b>perspective</b> [5] 4/12 11/12 67/25 70/11 134/3 <b>persuading</b> [1] 84/4 <b>pessaries</b> [1] 42/16 <b>pharmaceutical</b> [1] 112/3 <b>pharmacist</b> [2] 167/23 167/25 <b>phase</b> [7] 24/2 24/4 24/5 25/6 33/5 33/5 34/8 <b>phenomenon</b> [1] 169/7 <b>phone</b> [3] 167/22 169/14 187/8 <b>photophobia</b> [1] 19/21 <b>physical</b> [5] 77/9 78/3 87/8 136/15 170/10 <b>physically</b> [3] 77/17 78/2 125/18 <b>physicians</b> [2] 14/12 186/17 <b>physiological</b> [1] 187/17 <b>physiologically</b> [1] 129/11 <b>pick</b> [7] 87/14 106/13 111/2 130/19 149/9 158/20 193/11 <b>picked</b> [1] 162/7 <b>picking</b> [2] 14/9 123/20 <b>picture</b> [2] 82/23 137/10 <b>pictures</b> [1] 100/19 <b>piece</b> [1] 164/5 <b>pigeon</b> [1] 54/8 <b>pigeons</b> [1] 54/8 <b>pill</b> [6] 102/25 108/3	109/23 114/1 114/4 122/13 <b>pills</b> [4] 108/15 108/22 109/7 116/14 <b>pink</b> [1] 19/18 <b>pivotal</b> [2] 114/5 114/16 <b>place</b> [6] 32/18 47/1 97/24 112/22 164/8 184/18 <b>places</b> [1] 68/13 <b>plainly</b> [1] 196/12 <b>plan</b> [1] 167/16 <b>planting</b> [1] 59/7 <b>plants</b> [1] 59/8 <b>platelet</b> [1] 39/10 <b>platelets</b> [1] 39/6 <b>play</b> [3] 38/12 99/7 129/13 <b>please</b> [6] 31/12 50/22 90/18 137/9 152/5 157/23 <b>pleased</b> [1] 1/9 <b>plus</b> [2] 104/22 125/7 <b>pm</b> [5] 45/9 90/19 137/2 137/4 196/24 <b>pneumocystis</b> [4] 37/11 63/13 65/6 68/15 <b>pneumonia</b> [2] 37/11 57/24 <b>point</b> [40] 25/15 25/25 26/1 27/11 27/20 28/13 32/7 38/19 43/15 49/8 56/2 56/6 76/7 83/23 85/24 87/19 96/6 100/18 104/25 105/1 105/9 105/22 105/24 105/24 111/7 115/14 120/1 123/17 123/20 126/10 131/17 135/22 147/21 152/2 159/21 162/7 166/12 168/18 178/5 193/13 <b>pointing</b> [1] 41/25 <b>points</b> [2] 15/4 27/5 <b>poison</b> [1] 186/13 <b>policy</b> [1] 5/23 <b>polyproteins</b> [2] 145/4 145/4 <b>pond</b> [1] 75/3 <b>poo</b> [1] 54/8 <b>pooled</b> [1] 15/9 <b>poor</b> [2] 56/16 80/25 <b>POPPY</b> [1] 194/9 <b>population</b> [24] 5/18 11/2 70/4 70/6 73/10 73/13 73/16 73/21 75/6 76/22 76/23 81/15 113/5 128/16 128/16 128/18 135/6 190/8 190/9 190/14	190/15 190/24 191/1 191/7 <b>populations</b> [5] 11/2 33/1 36/19 178/9 195/8 <b>port</b> [2] 58/12 58/18 <b>port-a-caths</b> [1] 58/12 <b>position</b> [4] 71/12 152/21 174/6 180/3 <b>positive</b> [15] 12/15 14/11 18/7 23/23 64/19 70/3 76/13 76/15 76/18 83/11 149/15 159/1 159/24 160/18 184/6 <b>possible</b> [11] 10/21 13/3 15/12 15/16 15/23 22/20 83/22 126/15 172/19 173/21 175/19 <b>possibly</b> [1] 55/19 <b>postpone</b> [1] 151/10 <b>posts</b> [1] 40/9 <b>potential</b> [1] 22/23 <b>potentially</b> [4] 100/22 159/24 177/24 182/22 <b>powder</b> [2] 94/25 109/8 <b>power</b> [1] 48/21 <b>powerful</b> [4] 39/1 47/5 47/5 60/25 <b>powerless</b> [1] 79/1 <b>pox</b> [3] 38/20 60/21 60/22 <b>practical</b> [2] 34/23 35/8 <b>practice</b> [9] 4/11 5/15 15/5 84/17 85/18 127/3 150/5 158/1 166/6 <b>practitioner</b> [2] 4/6 4/7 <b>practitioners</b> [3] 1/8 83/25 84/7 <b>pre</b> [1] 80/6 <b>pre-internet</b> [1] 80/6 <b>precautions</b> [2] 180/17 180/21 <b>precipitate</b> [1] 72/11 <b>predispose</b> [2] 71/25 72/6 <b>preface</b> [1] 154/23 <b>pregnancy</b> [1] 192/6 <b>pregnant</b> [1] 58/23 <b>prejudicial</b> [1] 134/16 <b>premature</b> [1] 130/17 <b>prepared</b> [1] 155/15 <b>prepares</b> [1] 152/15 <b>prescribe</b> [1] 112/9 <b>prescribed</b> [2] 116/21 184/1 <b>prescribing</b> [1] 109/15	<b>prescription</b> [5] 164/7 164/11 165/9 165/10 165/23 <b>presence</b> [3] 27/9 145/22 180/11 <b>present</b> [11] 15/13 20/8 53/2 62/13 65/11 68/15 68/17 68/19 68/20 113/13 178/24 <b>presentation</b> [1] 21/21 <b>presented</b> [3] 20/23 64/13 64/13 <b>presenting</b> [4] 20/4 21/15 53/19 166/17 <b>press</b> [2] 105/10 184/25 <b>pressure</b> [6] 53/12 80/22 105/17 105/18 105/20 133/20 <b>Presumably</b> [4] 16/3 33/12 44/23 132/15 <b>presumed</b> [1] 70/22 <b>presupposes</b> [1] 35/16 <b>pretty</b> [2] 7/12 130/24 <b>prevalence</b> [9] 70/9 133/18 190/6 190/21 190/23 191/9 191/10 191/19 191/25 <b>prevalent</b> [2] 54/6 65/1 <b>prevent</b> [10] 59/18 61/11 66/7 66/10 68/16 68/19 97/9 103/2 118/22 186/9 <b>preventative</b> [1] 68/23 <b>prevented</b> [1] 130/2 <b>preventing</b> [1] 65/22 <b>previous</b> [7] 47/15 49/22 93/1 106/16 117/2 123/5 132/12 <b>primarily</b> [1] 87/19 <b>primary</b> [27] 17/8 18/15 19/8 20/4 20/8 21/10 21/11 21/19 22/15 23/5 25/5 85/1 85/7 155/7 155/9 155/19 155/21 155/25 156/21 156/24 157/12 157/24 159/3 164/1 165/17 167/8 168/16 <b>print</b> [1] 10/23 <b>prior</b> [1] 12/18 <b>pro</b> [3] 142/2 144/12 144/14 <b>pro-viral</b> [3] 142/2 144/12 144/14 <b>probably</b> [28] 31/19 32/22 34/23 39/7 41/9 43/21 48/15 54/11 56/12 63/22 65/6 70/8 70/23 81/6 95/8
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------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------

<b>P</b>	2/20	125/11 153/3 187/16	<b>questions [34]</b> 6/5 6/16 48/5 82/7 87/15 90/14 91/19 91/19 136/22 148/18 149/3 149/8 156/9 156/14 156/19 156/20 160/16 165/1 171/15 172/4 172/5 178/15 178/21 180/2 180/22 182/15 189/11 190/10 192/1 192/10 195/10 195/15 195/18 196/17 <b>queue [1]</b> 143/1 <b>quick [2]</b> 113/7 168/18 <b>quickly [15]</b> 13/15 31/3 76/16 89/1 124/1 145/25 154/8 161/3 161/18 161/24 162/19 165/3 165/19 170/20 181/13 <b>quiescent [1]</b> 119/11 <b>quite [58]</b> 7/5 11/1 13/15 19/11 20/18 21/20 21/22 22/16 30/25 33/20 37/4 38/18 38/22 38/25 39/1 39/17 39/22 39/23 39/25 40/3 44/20 50/4 55/18 55/19 59/10 60/4 60/25 61/1 61/25 65/11 66/3 66/5 76/16 78/7 83/6 90/5 94/19 94/20 94/25 95/5 98/7 99/3 100/3 107/3 107/7 108/25 112/13 156/18 156/21 161/23 161/24 173/22 174/18 184/14 187/2 187/3 191/6 194/1 <b>quoted [2]</b> 176/8 190/17	<b>rate [5]</b> 74/3 116/16 134/25 188/11 193/1 <b>rates [5]</b> 71/25 105/2 192/8 192/11 192/15 <b>rather [15]</b> 35/22 64/14 65/20 89/20 102/11 105/16 118/16 122/25 130/16 138/8 139/15 140/10 145/23 150/13 186/6 <b>rationalise [1]</b> 111/10 <b>re [2]</b> 46/21 175/12 <b>re-emerge [1]</b> 46/21 <b>re-emerges [1]</b> 175/12 <b>reach [1]</b> 38/6 <b>reached [1]</b> 134/22 <b>reaction [2]</b> 66/16 186/5 <b>reactions [1]</b> 110/20 <b>reactivate [2]</b> 57/6 67/4 <b>reactivation [4]</b> 57/6 57/16 57/21 57/23 <b>read [2]</b> 6/10 97/16 <b>reading [2]</b> 140/17 187/8 <b>reads [1]</b> 140/15 <b>real [7]</b> 51/6 76/10 181/22 181/24 182/2 189/17 196/12 <b>really [96]</b> 2/25 8/18 21/20 28/25 29/2 30/10 32/20 39/16 41/12 43/11 44/8 44/11 44/18 44/20 48/23 48/24 49/16 51/12 52/19 54/1 54/5 54/16 54/24 55/4 55/8 55/9 55/11 56/10 56/13 57/11 58/7 58/9 58/21 58/23 58/25 59/9 59/15 59/24 61/5 61/14 63/10 64/12 70/1 71/1 75/9 76/1 76/11 76/13 76/24 80/10 85/4 87/23 89/25 90/25 94/7 95/5 96/13 96/19 97/12 106/17 107/1 107/20 108/7 109/15 110/4 110/6 110/14 110/25 120/14 121/17 122/7 123/12 131/6 132/19 132/24 135/13 135/22 145/16 151/7 151/22 152/1 152/4 152/5 152/10 153/17 161/25 162/18 170/24 173/23 174/21 177/20 181/19 182/25 186/2 186/19 186/25 <b>really be [1]</b> 28/25	<b>reason [9]</b> 11/9 18/5 29/16 91/15 99/20 118/3 118/6 173/24 178/7 <b>reasoning [1]</b> 151/9 <b>reasons [12]</b> 3/19 26/5 26/6 26/8 28/20 42/10 51/24 146/13 150/18 172/16 172/17 176/9 <b>reassembling [1]</b> 144/24 <b>reassembly [1]</b> 145/12 <b>reassure [2]</b> 2/3 90/13 <b>reassuring [1]</b> 182/3 <b>rebuild [2]</b> 118/20 119/1 <b>recall [1]</b> 20/25 <b>receive [1]</b> 93/12 <b>received [3]</b> 37/21 66/18 95/11 <b>receives [1]</b> 154/17 <b>recent [6]</b> 52/19 83/12 89/2 115/16 174/24 195/10 <b>recently [3]</b> 50/5 86/6 89/7 <b>receptor [4]</b> 29/11 29/22 138/19 139/1 <b>receptors [4]</b> 26/18 29/23 29/25 148/7 <b>recognise [2]</b> 17/18 183/5 <b>recognised [5]</b> 22/7 28/17 33/4 186/23 189/2 <b>recognising [2]</b> 36/6 36/20 <b>recombinants [1]</b> 13/22 <b>recommend [1]</b> 163/11 <b>recommending [1]</b> 90/1 <b>reconstitute [1]</b> 110/19 <b>reconstitution [1]</b> 166/16 <b>recorded [1]</b> 85/10 <b>records [1]</b> 191/13 <b>recover [2]</b> 118/18 118/19 <b>recovered [2]</b> 66/19 132/7 <b>recovery [2]</b> 21/5 57/14 <b>recurrence [1]</b> 61/11 <b>recurrent [1]</b> 44/16 <b>recurring [3]</b> 42/23 58/5 102/8 <b>red [1]</b> 84/13 <b>reduce [6]</b> 93/19
<b>probably... [13]</b> 128/19 135/3 136/25 154/7 159/9 162/15 163/23 164/3 165/8 167/13 168/25 175/2 176/10 <b>problem [14]</b> 39/8 47/4 48/14 49/2 49/10 49/15 57/3 93/21 99/16 107/22 127/15 127/17 133/13 159/12 <b>problematic [1]</b> 182/23 <b>problems [18]</b> 54/15 60/23 62/18 70/16 76/12 76/25 107/21 107/24 109/23 113/11 117/10 128/17 132/16 133/20 133/22 135/17 136/5 136/6 <b>procedure [1]</b> 58/18 <b>procedures [2]</b> 174/22 182/10 <b>process [15]</b> 25/10 48/6 129/22 143/9 147/5 147/18 154/25 155/4 160/21 162/13 174/4 175/17 195/22 195/24 195/25 <b>processes [3]</b> 25/13 28/8 78/1 <b>processing [1]</b> 165/22 <b>produce [6]</b> 17/20 112/4 140/16 141/13 145/1 145/2 <b>produced [5]</b> 36/9 76/19 140/25 141/5 187/24 <b>produces [2]</b> 18/1 18/3 <b>producing [2]</b> 17/24 145/3 <b>product [5]</b> 9/25 10/11 13/4 16/4 16/24 <b>products [14]</b> 9/3 10/18 11/21 15/9 62/7 121/13 140/16 141/11 150/13 150/15 172/8 175/24 178/3 193/6 <b>professional [1]</b> 161/6 <b>professionals [2]</b> 78/25 162/16 <b>PROFESSOR [15]</b> 1/14 1/15 1/19 2/1 2/20 2/25 5/2 81/13 87/3 112/20 134/2 148/25 150/1 158/20 180/10 <b>Professor Anderson</b> <b>[2]</b> 87/3 158/20 <b>Professor Cooke [1]</b>	<b>Professor Rockstroh</b> <b>[1]</b> 180/10 <b>Professor</b> <b>Tudor-Williams [5]</b> 2/1 2/25 112/20 134/2 150/1 <b>profile [2]</b> 107/13 108/4 <b>profound [6]</b> 54/25 55/11 60/8 65/23 67/9 136/12 <b>profuse [2]</b> 181/19 181/20 <b>progress [1]</b> 23/11 <b>progressed [2]</b> 102/1 121/22 <b>progresses [1]</b> 88/25 <b>progressing [1]</b> 190/4 <b>progression [8]</b> 29/13 34/11 35/18 35/23 89/6 98/20 105/2 115/7 <b>progressive [3]</b> 57/5 60/6 67/1 <b>project [1]</b> 3/24 <b>projects [1]</b> 3/21 <b>prolonged [1]</b> 88/17 <b>prominent [3]</b> 46/22 62/15 184/15 <b>promised [1]</b> 90/23 <b>pronounce [1]</b> 54/17 <b>proofread [2]</b> 13/17 101/11 <b>properly [4]</b> 8/2 51/15 53/7 53/15 <b>prophylaxis [1]</b> 111/21 <b>proportion [7]</b> 9/25 10/24 30/11 76/3 82/19 83/14 175/10 <b>protease [12]</b> 104/17 106/18 107/15 107/18 107/25 108/12 110/4 141/7 141/12 141/24 144/23 145/12 <b>protect [2]</b> 17/25 97/14 <b>protective [1]</b> 16/16 <b>protein [1]</b> 141/8 <b>proteins [1]</b> 144/24 <b>provide [3]</b> 36/23 155/22 155/23 <b>provided [6]</b> 5/25 6/3 6/9 91/4 152/25 155/24 <b>providing [1]</b> 100/5 <b>provision [1]</b> 169/22 <b>psychiatric [1]</b> 124/18 <b>psychological [14]</b> 44/22 74/13 75/1 76/12 77/9 77/15 78/1 78/4 81/8 87/5 125/10	<b>psychologically [4]</b> 43/24 44/13 75/10 77/16 <b>psychologists [1]</b> 80/17 <b>psychosocial [2]</b> 153/20 162/6 <b>puberty [3]</b> 151/8 151/9 151/19 <b>public [7]</b> 32/24 76/19 115/14 161/17 191/9 191/16 195/6 <b>publicised [3]</b> 173/14 173/16 174/20 <b>publish [1]</b> 133/4 <b>published [2]</b> 192/16 192/18 <b>pull [1]</b> 31/11 <b>pulse [1]</b> 188/11 <b>punctures [1]</b> 53/11 <b>purple [1]</b> 62/21 <b>purpose [1]</b> 94/8 <b>purpura [1]</b> 39/6 <b>put [23]</b> 2/4 13/9 32/14 35/19 36/22 39/4 67/24 74/6 89/21 97/7 97/18 109/14 109/15 128/15 134/19 136/22 142/20 154/15 178/13 184/18 185/13 192/3 193/10 <b>putting [3]</b> 9/18 75/2 144/3 <b>Q</b> <b>qualified [1]</b> 5/8 <b>qualitative [1]</b> 86/2 <b>quality [8]</b> 18/25 27/24 73/18 103/6 115/25 133/15 134/1 162/1 <b>quantitative [2]</b> 83/17 86/1 <b>quantity [1]</b> 9/23 <b>quasi [1]</b> 13/19 <b>quasi-species [1]</b> 13/19 <b>question [35]</b> 71/11 78/19 82/9 82/10 82/17 83/6 87/9 91/16 120/17 124/1 126/8 126/11 129/20 130/20 131/2 131/5 132/25 134/3 146/9 163/3 173/18 174/7 176/15 176/18 177/9 182/9 183/23 183/24 185/9 187/7 187/9 187/14 193/14 194/3 195/19 <b>questioning [1]</b> 20/24 <b>questionnaire [2]</b> 82/4 82/14	<b>radical [1]</b> 174/18 <b>raised [3]</b> 6/5 19/15 95/24 <b>raltegravir [1]</b> 113/22 <b>randomised [5]</b> 103/12 103/17 103/21 114/12 120/21 <b>range [1]</b> 108/25 <b>rapid [2]</b> 28/10 35/22 <b>rapidly [1]</b> 13/16 <b>rare [3]</b> 21/2 21/7 47/13 <b>rarely [3]</b> 61/16 163/25 164/1 <b>rash [6]</b> 19/18 66/15 107/5 185/25 186/1 186/2 <b>rashes [1]</b> 107/4		



<b>R</b>					
<b>reduce...</b> [5] 97/14 120/18 120/19 120/22 133/24	161/18	81/19 81/24	<b>responsibility</b> [2] 152/8 159/12	145/18 145/19 147/11 148/5 149/3	44/15 51/13 55/1 55/20 62/5 67/16
<b>reduced</b> [2] 111/22 117/15	<b>relatives</b> [1] 3/25	<b>reporting</b> [1] 81/17	<b>responsible</b> [4] 7/16 92/10 145/3 181/10	<b>rightly</b> [1] 100/2	82/19 87/10 99/21
<b>refer</b> [2] 86/9 158/12	<b>release</b> [1] 187/23	<b>reports</b> [3] 14/4 84/11 180/18	<b>rest</b> [5] 64/24 91/20 125/5 127/8 164/20	<b>ringing</b> [1] 40/11	101/10 101/23 102/18
<b>reference</b> [2] 91/17 176/11	<b>relevant</b> [4] 22/12 22/13 22/14 36/24	<b>representational</b> [1] 138/1	<b>resting</b> [3] 30/17 142/4 187/12	<b>ripples</b> [2] 75/3 76/6	102/21 108/12 115/22
<b>referenced</b> [1] 193/7	<b>reliant</b> [1] 45/24	<b>represented</b> [1] 129/17	<b>restrictions</b> [1] 106/22	<b>rise</b> [3] 28/15 63/5 177/11	116/23 117/13 130/22
<b>referral</b> [4] 158/19 169/19 170/20 170/21	<b>relies</b> [1] 173/22	<b>reproduce</b> [3] 141/23 141/25 141/25	<b>result</b> [18] 10/6 21/12 59/3 66/21 67/9 81/20	<b>risk</b> [40] 9/22 10/1 10/9 11/16 11/17	133/2 133/13 135/11
<b>referrals</b> [1] 86/13	<b>reluctance</b> [1] 87/6	<b>repulsive</b> [1] 112/7	83/7 97/1 114/16	11/21 11/25 12/5 12/6	162/12 171/7 196/8
<b>referred</b> [5] 71/21 79/6 79/7 156/3 161/8	<b>rely</b> [1] 124/22	<b>requested</b> [1] 190/3	137/20 140/17 150/12	12/7 16/22 34/11	<b>Saint</b> [3] 2/9 5/4 5/6
<b>referring</b> [5] 31/25 65/8 120/12 154/4 158/5	<b>remain</b> [4] 59/17 135/21 142/3 189/6	<b>require</b> [3] 89/12 160/17 168/3	150/25 155/13 177/7	64/11 72/20 73/6 73/7	<b>same</b> [37] 2/24 7/20
<b>reflect</b> [1] 165/16	<b>remaining</b> [1] 187/11	<b>required</b> [4] 34/19 53/10 68/22 162/8	185/17 186/1 186/3	73/8 73/14 73/20	7/21 8/5 11/15 16/4
<b>reflects</b> [1] 134/15	<b>remains</b> [5] 24/18 31/7 35/11 71/14 194/21	<b>requires</b> [4] 9/14 157/11 165/22 174/17	<b>resulted</b> [1] 105/12	73/22 74/1 83/1 85/16	23/13 26/25 28/7
<b>refractory</b> [1] 10/25	<b>remember</b> [16] 14/4 14/15 37/8 42/15 59/2	<b>research</b> [3] 3/3 3/20 174/3	<b>results</b> [4] 93/6 166/2 184/20 188/21	97/10 97/14 115/7	35/11 42/21 50/16
<b>regard</b> [1] 25/17	59/5 59/12 70/17	<b>researching</b> [1] 4/10	185/17 186/1 186/3	120/22 121/2 121/5	63/16 73/25 85/25
<b>regarded</b> [1] 186/12	74/17 80/5 96/16	<b>reservoir</b> [1] 142/5	<b>retinitis</b> [3] 59/10 59/22 68/18	135/1 135/2 172/13	86/6 88/6 93/22 94/14
<b>regime</b> [1] 164/25	97/17 105/25 108/21	<b>resident</b> [1] 170/1	<b>retroviral</b> [27] 5/1 11/15 35/17 39/19	176/9 176/12 177/11	94/23 101/2 102/13
<b>regimen</b> [1] 116/13	109/10 186/7	<b>resistance</b> [19] 26/17 52/17 84/6 86/14	41/19 43/8 43/10	177/17 177/25 178/22	121/11 123/17 125/5
<b>regimens</b> [5] 103/15 114/2 114/3 116/25 117/3	<b>remembering</b> [2] 84/3 110/12	86/19 102/6 106/6	49/11 66/4 74/10	179/17 186/10	125/19 134/3 135/6
<b>regimes</b> [1] 164/16	<b>reminder</b> [2] 122/12 122/15	117/1 117/1 118/1	80/24 90/11 104/11	<b>risks</b> [5] 16/25 90/9 91/9 175/23 177/14	146/1 147/5 154/20
<b>regional</b> [2] 79/22 79/23	<b>renal</b> [4] 74/3 106/22 133/21 183/16	118/1 123/6 126/20	109/22 115/4 116/9	<b>ritonavir</b> [5] 106/24	164/4 165/4 166/4
<b>regular</b> [1] 66/6	<b>render</b> [1] 137/21	127/1 145/24 147/14	117/14 118/5 122/21	107/20 108/18 112/6 112/9	179/25 183/7 192/21
<b>regularly</b> [2] 68/14 183/20	<b>reoccur</b> [1] 38/24	148/11 148/13 148/15	127/6 165/19 166/11	<b>RNA</b> [11] 92/12 139/5 139/10 139/11 139/15	<b>saquinavir</b> [2] 107/23 108/19
<b>rein</b> [1] 129/3	<b>repeated</b> [2] 11/20 16/3	<b>resistant</b> [4] 12/4 101/6 101/9 113/4	166/14 166/19 167/18	139/22 139/24 140/10	<b>sarcoma</b> [4] 62/4 63/1 63/3 178/2
<b>reinforce</b> [1] 120/1	<b>repetitive</b> [1] 53/11	<b>resolve</b> [3] 21/5 21/9 21/23	168/20 185/24	145/1 145/2 145/3	<b>satisfaction</b> [1] 158/6
<b>Reiterate</b> [1] 179/21	<b>replaced</b> [1] 128/8	<b>resonates</b> [1] 166/5	<b>retrovirals</b> [1] 71/13	<b>road</b> [1] 2/9	<b>saw</b> [4] 58/25 98/7 113/18 182/20
<b>relapse</b> [4] 59/18 59/20 59/20 102/13	<b>replicate</b> [5] 18/13 24/8 94/17 140/13 145/21	<b>resource</b> [2] 169/21 195/9	<b>retroviruses</b> [1] 6/23	<b>robust</b> [2] 32/16 128/2	<b>say</b> [57] 1/23 2/6 19/12 20/21 21/1
<b>relapsed</b> [2] 173/5 185/17	<b>replicates</b> [4] 13/16 18/11 37/5 101/10	<b>respect</b> [4] 37/18 151/2 155/23 162/9	<b>return</b> [1] 78/19	<b>Rockstroh</b> [2] 148/25 180/10	22/24 32/23 33/18
<b>related</b> [12] 26/5 26/5 26/8 27/10 28/20 28/25 60/21 62/12 69/17 135/1 180/7 183/21	<b>replicating</b> [2] 25/20 145/19	<b>respected</b> [1] 129/15	<b>returned</b> [1] 2/14	<b>role</b> [2] 4/25 99/7	37/12 41/8 41/21
<b>relates</b> [3] 15/24 45/24 64/3	<b>replication</b> [7] 30/20 31/6 31/8 92/8 119/17 120/6 130/1	<b>respond</b> [5] 17/17 17/17 26/23 26/24 26/25	<b>returning</b> [1] 187/11	<b>roles</b> [1] 5/21	86/25 91/3 100/2
<b>relating</b> [1] 134/20	<b>report</b> [58] 2/4 6/3 6/6 6/9 6/10 12/9 14/23 17/7 19/12 20/14 21/1 23/19 26/4 27/2 27/8 29/11 29/15 31/12 33/7 35/20 36/22 36/24 40/8 45/23 49/6 50/8 62/5 63/5 68/25 81/16 91/4 95/8 101/23 112/19 134/4 134/20 147/3 148/23 149/6 149/9 150/1 150/2 153/24 154/6 154/15 157/19 171/5 171/16 173/17 175/5 178/14 180/7 190/17 190/18 191/19 191/22 191/25 193/8	<b>responded</b> [1] 40/25	<b>reverse</b> [21] 92/9 92/9 92/16 93/3 93/4 94/10 104/13 104/14 139/14 139/18 140/23 141/11 141/19 141/20 142/11 145/10 145/14 146/14 146/22 147/15 147/16	<b>room</b> [2] 54/11 59/7	102/11 103/14 103/22
<b>relation</b> [9] 91/12 91/19 91/23 95/20 116/6 152/17 187/9 191/22 193/18	<b>reported</b> [3] 14/11	<b>responding</b> [2] 21/16 30/19	<b>reversible</b> [1] 60/10	<b>round</b> [7] 8/5 27/18 53/6 97/19 139/16 140/2 144/14	108/7 110/8 111/13
<b>relations</b> [1] 120/24		<b>responds</b> [6] 16/16 17/2 19/1 25/4 27/17 30/15	<b>revised</b> [1] 35/25	<b>route</b> [4] 8/22 9/1 16/19 28/3	112/19 114/22 119/9
<b>relationship</b> [5] 125/13 125/15 125/15 151/25 163/3		<b>response</b> [25] 16/21 17/15 17/20 17/21 17/24 19/5 23/13 24/10 24/13 24/16 25/4 26/12 27/24 28/2 29/18 36/8 59/13 71/24 72/14 82/17 83/16 95/3 123/3 175/11 183/1	<b>revolutionised</b> [1] 80/7	<b>routes</b> [3] 8/20 177/14 177/16	121/10 128/19 130/19
<b>relationships</b> [3] 77/23 80/19 151/21		<b>responses</b> [6] 9/20 17/23 18/2 24/17 24/20 26/21	<b>rid</b> [2] 174/16 182/12	<b>routine</b> [2] 15/5 39/10	135/23 135/25 138/7
<b>relatively</b> [12] 11/9 16/9 52/19 61/13 61/16 67/7 70/6 70/8 89/7 124/19 158/1		<b>responsibilities</b> [1] 1/11	<b>right</b> [45] 6/7 6/8 8/4 14/19 16/21 20/15 45/20 63/2 63/8 66/23 78/21 82/20 92/20 93/18 96/4 104/3 110/9 110/15 116/22 117/25 118/15 119/5 120/25 121/11 132/1 132/17 133/5 133/19 137/15 138/16 139/13 139/25 140/9 140/22 141/3 141/17 142/19 143/2 144/17 144/22	<b>runs</b> [1] 76/14	141/6 141/6 151/4 152/3 152/22 155/6 158/7 161/2 164/11 165/6 166/7 171/22 177/18 179/6 182/7 183/18 185/20 194/19 195/23 196/10



<b>S</b>	<b>sensation [1]</b> 65/16 <b>sense [10]</b> 10/7 30/23 33/2 34/11 39/15 69/22 73/7 77/19 114/8 177/2 <b>sensitive [2]</b> 23/7 120/10 <b>sensitivity [3]</b> 120/13 120/16 186/5 <b>sensory [1]</b> 59/8 <b>separate [3]</b> 28/8 139/22 143/12 <b>separately [2]</b> 69/21 162/17 <b>separation [2]</b> 75/18 75/19 <b>septrin [3]</b> 66/7 66/13 66/15 <b>sequelae [2]</b> 67/13 133/11 <b>series [2]</b> 36/19 68/21 <b>serious [5]</b> 55/10 62/3 89/1 96/14 96/14 <b>seroconversion [3]</b> 21/11 23/20 24/6 <b>seroprevalence [1]</b> 192/8 <b>served [1]</b> 84/5 <b>service [4]</b> 81/19 125/14 158/3 158/23 <b>services [11]</b> 3/2 79/8 80/22 81/9 81/10 82/5 86/10 86/14 87/7 100/5 169/23 <b>session [2]</b> 132/12 136/23 <b>set [10]</b> 5/11 20/20 25/14 26/1 27/5 27/11 27/19 38/9 74/5 159/16 <b>sets [5]</b> 45/15 49/2 89/13 95/13 153/2 <b>setting [16]</b> 28/14 40/4 52/5 57/4 61/21 121/23 154/18 154/19 155/7 157/3 157/12 159/19 169/18 179/4 182/23 183/2 <b>settings [5]</b> 83/25 126/2 157/18 159/4 159/5 <b>several [2]</b> 21/12 21/22 <b>severe [19]</b> 19/11 21/10 21/22 36/20 50/2 53/17 62/18 68/21 93/8 94/20 96/22 99/4 107/4 107/5 115/8 132/9 184/11 185/25 186/2 <b>severely [2]</b> 21/18 37/17 <b>sex [7]</b> 8/24 8/24 8/24	9/9 163/4 178/8 178/23 <b>sexual [18]</b> 8/23 8/25 8/25 10/8 10/9 10/13 10/14 15/19 16/25 86/24 120/24 121/15 159/19 163/10 176/5 179/7 179/13 180/14 <b>sexually [2]</b> 151/18 178/6 <b>shall [5]</b> 6/20 8/21 56/19 63/20 162/25 <b>share [5]</b> 77/8 77/14 77/16 134/8 196/15 <b>shared [2]</b> 154/10 177/16 <b>sharing [3]</b> 77/13 154/24 164/3 <b>shelf [1]</b> 92/25 <b>shift [1]</b> 110/4 <b>shifting [1]</b> 42/23 <b>shingles [4]</b> 38/17 40/24 44/15 44/16 <b>shiny [1]</b> 128/24 <b>shock [1]</b> 156/7 <b>shoe [1]</b> 109/9 <b>short [14]</b> 19/17 45/8 51/9 65/12 65/14 91/5 93/25 99/10 137/3 169/20 170/17 180/25 185/16 185/19 <b>short-lived [1]</b> 93/25 <b>short-term [4]</b> 99/10 170/17 185/16 185/19 <b>shortening [1]</b> 74/25 <b>shorthand [1]</b> 32/21 <b>shortness [1]</b> 65/17 <b>should [30]</b> 17/5 28/25 28/25 29/1 40/11 75/18 91/8 91/22 115/13 118/21 149/7 149/10 153/25 154/1 154/10 154/11 154/12 155/22 157/18 159/7 159/9 159/13 163/11 170/2 170/4 170/17 179/1 179/12 179/22 180/20 <b>should have [1]</b> 28/25 <b>showed [9]</b> 101/20 103/12 103/17 104/21 114/17 115/4 184/20 192/18 192/21 <b>showing [4]</b> 33/7 33/8 139/8 155/20 <b>shown [5]</b> 94/18 104/23 138/18 141/2 196/6 <b>shows [2]</b> 135/3 137/24 <b>SIAN [6]</b> 1/16 3/7 44/15 51/16 82/16 122/10	<b>Sian's [1]</b> 50/23 <b>sick [12]</b> 32/10 35/14 51/22 58/7 67/25 125/8 125/9 160/4 165/13 167/11 184/18 184/19 <b>sicker [1]</b> 184/23 <b>side [48]</b> 19/22 38/3 67/23 68/2 94/1 94/19 95/6 95/14 95/23 96/11 96/13 96/14 96/21 97/1 98/14 99/4 99/13 103/8 103/9 103/14 104/2 104/4 104/5 107/13 107/21 108/4 108/6 109/2 109/24 113/8 114/22 116/5 116/16 116/19 116/20 123/7 128/14 129/2 157/6 182/16 182/21 182/24 183/7 183/14 183/19 184/24 185/8 185/22 <b>sides [1]</b> 117/5 <b>sight [1]</b> 53/16 <b>sign [2]</b> 40/9 169/12 <b>signal [1]</b> 39/13 <b>signalling [2]</b> 40/9 48/25 <b>signals [1]</b> 127/25 <b>significant [5]</b> 7/14 21/8 60/4 163/14 171/8 <b>significantly [3]</b> 7/6 8/16 33/21 <b>signposting [1]</b> 154/3 <b>signs [1]</b> 21/2 <b>similar [10]</b> 7/12 10/4 10/5 20/1 20/10 28/13 36/2 56/23 80/9 177/9 <b>Similarly [1]</b> 89/5 <b>simplex [4]</b> 60/16 60/18 60/22 61/4 <b>simply [6]</b> 14/14 14/15 112/16 134/15 155/16 156/21 <b>since [5]</b> 2/10 2/14 4/18 139/18 190/9 <b>single [8]</b> 14/15 15/7 92/12 114/3 125/19 139/24 139/25 140/1 <b>sir [3]</b> 131/16 136/20 148/20 <b>sit [3]</b> 8/4 33/3 112/14 <b>site [5]</b> 92/18 142/20 143/13 143/15 146/21 <b>sites [4]</b> 110/20 146/3 146/20 146/25 <b>sitting [1]</b> 55/15 <b>situation [2]</b> 15/20 86/6 <b>situations [1]</b> 181/15 <b>six [6]</b> 25/10 25/12	25/25 37/15 101/5 168/11 <b>six-monthly [1]</b> 168/11 <b>sixth [1]</b> 25/24 <b>size [1]</b> 16/22 <b>skills [3]</b> 160/23 162/8 162/15 <b>skin [6]</b> 39/23 39/24 60/19 62/15 62/22 72/25 <b>sleeping [1]</b> 124/6 <b>sleepless [1]</b> 153/14 <b>slightly [8]</b> 23/16 23/17 83/14 88/9 89/10 143/12 143/13 189/19 <b>slip [1]</b> 145/20 <b>slow [1]</b> 65/10 <b>small [10]</b> 9/15 10/22 10/23 12/3 30/11 97/4 111/25 158/1 168/19 173/21 <b>smaller [2]</b> 85/19 176/7 <b>smart [1]</b> 114/5 <b>smear [1]</b> 43/5 <b>smoke [1]</b> 72/6 <b>smoker [2]</b> 73/25 74/1 <b>smoking [1]</b> 73/6 <b>so [422]</b> <b>societal [1]</b> 82/23 <b>some [107]</b> 6/2 6/4 7/8 8/1 8/6 9/18 9/19 20/14 21/17 24/20 28/5 31/21 34/10 34/16 38/16 38/19 40/11 40/22 40/25 41/3 41/9 45/12 46/19 49/24 50/1 52/17 55/24 58/3 61/3 61/7 61/21 62/12 62/23 64/5 64/5 66/18 66/19 67/19 69/23 71/19 72/1 72/5 72/7 72/7 72/19 75/21 76/15 80/12 85/16 85/22 86/22 87/16 87/17 88/8 89/10 90/5 92/2 95/14 96/7 96/23 96/25 97/1 98/23 98/25 99/7 102/20 104/4 106/13 109/13 109/24 111/5 111/21 113/8 113/10 114/8 119/5 122/16 124/7 127/12 129/3 135/2 135/16 135/18 138/19 143/3 143/4 148/6 148/7 150/4 157/12 160/16 164/21 165/3 165/5 165/10 165/11 170/15 172/7 172/12	176/10 178/15 185/4 185/6 185/24 191/8 192/1 194/22 <b>somebody [58]</b> 5/7 6/25 7/11 9/16 11/22 12/15 17/13 20/19 21/15 32/10 33/7 33/8 33/15 42/6 47/24 48/18 49/1 54/18 54/22 61/8 69/20 76/12 77/20 92/4 122/19 122/24 129/20 130/23 130/25 131/4 154/17 155/6 158/7 159/10 159/18 159/20 159/22 160/13 161/3 161/4 163/4 163/12 163/20 165/12 165/16 167/10 167/17 167/21 168/6 168/15 169/7 169/15 171/11 171/14 171/18 172/2 172/19 180/16 <b>somebody's [3]</b> 32/16 48/22 171/8 <b>somehow [1]</b> 74/22 <b>someone [45]</b> 13/24 17/16 18/6 18/10 18/14 18/16 19/1 19/2 19/7 22/16 22/22 23/5 24/25 31/12 64/19 64/20 73/2 74/4 76/15 116/8 116/10 117/7 117/8 117/12 123/1 123/7 123/9 123/11 123/13 123/21 130/9 131/23 133/5 133/6 133/9 133/16 166/8 177/10 180/18 183/24 184/5 184/11 185/20 190/19 195/21 <b>someone's [1]</b> 28/15 <b>something [55]</b> 20/11 20/25 29/3 33/16 34/6 39/13 40/12 40/16 45/4 47/20 48/14 48/22 55/23 57/12 60/13 62/4 62/5 66/14 68/8 68/10 70/15 76/13 81/23 84/13 85/17 93/18 95/4 96/2 98/20 99/14 108/17 108/20 111/11 118/12 122/11 128/11 128/24 132/3 137/6 143/12 145/10 145/11 145/12 145/21 156/6 169/9 169/13 178/4 178/9 181/16 181/19 183/5 183/16 190/3 196/11 <b>sometimes [21]</b> 10/5 19/11 19/18 42/16 43/13 47/23 55/6 75/6
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(71) scientists - sometimes

<b>S</b>	<b>species [1]</b> 13/19 <b>specific [15]</b> 8/11 29/17 39/17 39/22 40/4 40/14 41/14 43/14 63/9 78/19 89/7 167/14 180/7 186/6 192/1 <b>specifically [7]</b> 23/24 24/12 41/6 83/6 92/24 129/21 134/10 <b>spectrum [6]</b> 31/20 33/3 48/20 85/9 85/10 173/2 <b>spend [1]</b> 112/2 <b>spending [1]</b> 125/6 <b>spent [4]</b> 2/12 3/3 3/11 3/16 <b>spliced [2]</b> 144/11 145/5 <b>spoken [1]</b> 95/22 <b>sport [1]</b> 85/16 <b>spot [1]</b> 84/13 <b>spread [2]</b> 51/1 54/9 <b>spreading [1]</b> 76/6 <b>spreads [1]</b> 18/12 <b>St [2]</b> 3/18 5/10 <b>stabilises [2]</b> 25/14 169/2 <b>stable [1]</b> 168/6 <b>stage [25]</b> 19/13 19/23 25/19 26/1 30/12 32/1 34/25 43/17 45/11 48/9 52/12 138/11 138/12 139/3 139/9 140/22 140/23 141/2 141/16 147/4 155/11 162/24 167/5 184/23 187/1 <b>stages [6]</b> 17/6 17/8 31/22 138/23 147/3 147/22 <b>stand [1]</b> 152/16 <b>standard [6]</b> 40/24 41/1 43/6 103/22 159/6 182/10 <b>standards [4]</b> 158/23 159/15 160/2 163/11 <b>standing [1]</b> 127/15 <b>start [31]</b> 1/22 1/25 2/5 6/16 6/20 8/21 17/11 50/22 56/19 63/20 66/15 87/12 114/25 125/3 146/17 151/20 160/19 161/22 161/22 163/1 163/15 164/17 164/19 164/24 166/11 166/19 166/22 167/17 167/23 168/1 168/19 <b>started [7]</b> 3/8 70/17 113/18 114/1 114/2 124/24 130/21 <b>starting [13]</b> 88/22	116/8 116/10 116/12 117/7 118/23 118/25 161/18 166/14 168/2 168/10 168/25 187/3 <b>starts [6]</b> 18/11 43/9 137/17 141/23 154/6 167/17 <b>state [7]</b> 25/11 30/17 30/21 31/4 31/9 71/22 187/12 <b>States [3]</b> 34/16 93/7 186/8 <b>statistical [1]</b> 177/11 <b>status [1]</b> 81/20 <b>stavudine [3]</b> 94/13 94/23 127/18 <b>stay [2]</b> 35/7 46/16 <b>steady [1]</b> 25/11 <b>stem [4]</b> 173/4 173/4 173/7 173/8 <b>step [2]</b> 103/20 164/21 <b>Stevens [1]</b> 186/1 <b>sticking [2]</b> 116/4 149/25 <b>stigma [22]</b> 74/14 74/19 74/23 75/11 77/4 78/11 81/15 85/17 100/23 151/13 153/7 153/20 193/15 193/18 193/23 193/23 193/24 194/4 194/18 194/21 195/1 195/3 <b>stigmatised [2]</b> 74/22 85/8 <b>stigmatising [3]</b> 62/22 99/13 100/3 <b>still [59]</b> 2/18 5/15 35/15 36/12 56/13 56/15 60/10 60/13 67/5 72/18 78/18 80/12 80/23 80/25 80/25 81/4 81/11 83/13 83/15 89/12 96/24 96/25 97/5 97/12 97/13 97/25 98/10 98/11 98/16 99/7 99/14 99/16 99/17 100/4 100/7 101/23 101/23 102/1 102/1 107/6 109/18 111/16 119/10 122/13 124/6 124/18 124/22 129/3 129/5 129/18 145/21 174/21 174/25 181/6 189/21 190/1 190/2 192/22 194/24 <b>stomach [1]</b> 112/15 <b>stone [1]</b> 75/3 <b>stones [1]</b> 106/23 <b>stop [7]</b> 93/4 98/20 102/5 119/12 145/17 145/18 147/7	<b>stopped [4]</b> 93/9 102/3 145/10 145/11 <b>stops [4]</b> 43/23 143/17 144/13 145/18 <b>straight [3]</b> 158/15 159/23 166/13 <b>straightforwardly [1]</b> 175/20 <b>strand [5]</b> 139/11 139/23 139/24 139/25 140/3 <b>stranded [2]</b> 92/11 92/12 <b>strands [2]</b> 139/22 140/1 <b>Street [1]</b> 186/15 <b>strength [2]</b> 19/1 27/24 <b>stretch [1]</b> 169/2 <b>strides [1]</b> 83/9 <b>stroke [2]</b> 68/20 105/18 <b>strokes [1]</b> 67/8 <b>strong [5]</b> 40/15 71/14 96/3 108/19 125/16 <b>strongly [1]</b> 185/5 <b>struck [1]</b> 24/8 <b>structure [3]</b> 7/24 7/25 8/3 <b>struggled [1]</b> 86/17 <b>stuck [1]</b> 51/5 <b>studied [1]</b> 99/2 <b>studies [9]</b> 105/9 105/22 192/15 193/3 193/15 194/3 194/4 194/7 195/9 <b>study [11]</b> 81/14 82/2 82/4 82/14 83/10 83/17 114/5 114/5 194/10 194/16 195/12 <b>stuff [1]</b> 109/9 <b>stupid [1]</b> 37/13 <b>subcutaneous [2]</b> 99/12 110/18 <b>subject [1]</b> 86/8 <b>subjective [1]</b> 82/17 <b>subsequent [2]</b> 3/11 164/6 <b>subsequently [3]</b> 57/15 78/8 78/12 <b>substrate [1]</b> 143/15 <b>subtype [4]</b> 13/21 13/24 13/25 28/21 <b>subtypes [7]</b> 7/8 7/9 7/11 8/15 13/12 13/14 28/2 <b>successful [7]</b> 24/21 24/22 24/23 24/25 88/22 97/11 148/14 <b>such [8]</b> 11/6 67/22 72/21 72/21 72/22 97/23 107/15 167/3 <b>suck [1]</b> 42/17	<b>suffer [1]</b> 69/3 <b>suffered [2]</b> 66/20 131/9 <b>suffering [3]</b> 44/25 50/15 80/9 <b>sufficient [3]</b> 22/17 102/5 114/9 <b>suggest [4]</b> 11/8 16/9 134/4 134/12 <b>suggested [1]</b> 70/14 <b>suggests [3]</b> 11/13 47/25 132/5 <b>suicide [2]</b> 78/22 135/9 <b>suit [1]</b> 161/21 <b>suiting [1]</b> 167/21 <b>summarise [1]</b> 9/19 <b>summarised [1]</b> 23/9 <b>summary [2]</b> 6/2 171/22 <b>sunken [2]</b> 100/12 100/12 <b>super [3]</b> 15/15 15/17 15/24 <b>super-infection [1]</b> 15/24 <b>super-infections [1]</b> 15/15 <b>supplemental [8]</b> 6/6 9/20 12/9 36/23 157/10 157/19 171/16 171/19 <b>supplementary [4]</b> 6/5 178/14 191/25 196/17 <b>supplies [1]</b> 55/2 <b>support [23]</b> 75/5 77/10 78/24 78/24 79/4 79/12 79/13 79/19 79/24 80/4 81/5 81/9 126/1 126/4 155/21 157/6 161/9 168/3 168/13 187/6 188/6 188/6 188/21 <b>suppose [5]</b> 15/14 24/6 26/7 32/20 106/17 <b>suppress [3]</b> 106/5 117/23 118/4 <b>suppressed [1]</b> 105/11 <b>suppression [7]</b> 36/21 40/5 106/7 115/21 115/23 118/14 119/16 <b>sure [10]</b> 43/1 44/6 59/5 71/3 111/17 126/2 127/2 178/4 191/12 194/1 <b>surgical [1]</b> 58/17 <b>surprise [1]</b> 155/14 <b>surrogate [1]</b> 105/13 <b>surveillance [1]</b> 192/5	<b>survey [2]</b> 76/19 83/12 <b>surveys [1]</b> 76/18 <b>survival [3]</b> 134/11 134/17 193/1 <b>survive [3]</b> 80/3 96/24 110/23 <b>surviving [2]</b> 2/19 135/13 <b>susceptibility [2]</b> 10/3 172/15 <b>susceptible [2]</b> 35/1 101/14 <b>suspect [1]</b> 138/1 <b>suspension [1]</b> 112/6 <b>swallow [1]</b> 51/4 <b>swallowing [1]</b> 51/2 <b>sweats [5]</b> 39/20 39/21 41/14 44/7 44/8 <b>switches [1]</b> 30/4 <b>sworn [4]</b> 1/3 1/13 1/15 1/17 <b>Sydney [1]</b> 3/10 <b>sympathise [1]</b> 77/10 <b>sympathy [1]</b> 75/6 <b>symptomatic [3]</b> 33/15 36/9 184/5 <b>symptoms [38]</b> 19/8 19/9 19/10 19/13 19/14 19/20 19/23 19/25 20/9 20/10 20/14 20/20 21/8 21/8 21/23 22/15 33/7 33/9 33/13 33/13 33/13 34/5 35/10 38/3 39/15 41/12 41/24 43/19 47/24 50/16 52/10 64/25 66/23 109/4 124/7 127/14 159/8 196/10 <b>syndrome [1]</b> 166/16 <b>system [73]</b> 7/1 7/4 16/15 17/2 17/17 18/20 18/23 18/24 21/16 21/18 22/17 24/9 24/18 25/8 26/9 27/17 28/7 30/13 30/15 30/16 30/18 30/21 31/4 32/16 33/20 34/25 35/15 35/24 36/8 36/10 38/13 41/16 44/2 45/24 46/11 46/18 47/1 47/19 48/13 48/20 48/24 49/1 50/25 54/14 54/15 57/5 57/14 60/24 71/14 72/17 73/13 84/20 102/8 105/12 118/15 118/19 118/21 119/1 132/7 133/8 133/10 137/23 166/15 173/9 176/20 176/22
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<b>S</b>	<b>talked [8]</b> 33/25 42/8 77/4 86/8 88/11 116/2 176/2 177/4 <b>talking [34]</b> 12/19 29/8 29/9 32/2 32/3 32/5 34/1 37/14 38/5 38/8 39/16 69/3 71/19 75/17 109/4 111/3 125/9 125/20 136/2 136/2 136/16 136/17 145/7 153/5 156/2 160/8 174/9 179/24 181/4 181/15 181/22 182/11 182/17 195/20 <b>target [2]</b> 37/5 142/10 <b>targeted [4]</b> 92/7 92/16 92/17 184/9 <b>targeting [1]</b> 146/13 <b>targets [1]</b> 169/19 <b>task [1]</b> 196/16 <b>taste [1]</b> 112/17 <b>tasted [1]</b> 112/10 <b>TB [5]</b> 145/23 145/23 146/1 166/18 166/18 <b>teaching [1]</b> 3/13 <b>team [19]</b> 80/18 112/8 155/21 156/2 156/3 156/16 158/4 159/7 159/12 159/24 160/5 160/7 160/9 160/11 160/22 161/14 162/14 162/17 193/9 <b>teams [2]</b> 156/23 160/25 <b>technically [1]</b> 29/23 <b>technologies [1]</b> 105/5 <b>technology [1]</b> 14/20 <b>teenagers [1]</b> 169/3 <b>teeth [3]</b> 143/24 143/25 144/6 <b>tell [13]</b> 27/11 29/14 31/13 69/10 71/11 104/9 123/16 128/10 137/8 150/7 150/23 153/10 192/14 <b>telling [2]</b> 151/3 163/6 <b>temperature [1]</b> 19/15 <b>tempering [1]</b> 128/21 <b>ten [2]</b> 152/9 174/23 <b>tend [13]</b> 11/6 28/18 34/11 72/6 116/18 128/25 129/15 130/15 133/17 133/21 146/12 183/6 186/25 <b>tended [2]</b> 112/24 112/25 <b>tends [3]</b> 17/25 18/7 34/7 <b>tenof [3]</b> 128/4 128/4 128/5 <b>tenofovir [7]</b> 107/12 113/24 128/3 128/4	128/9 183/13 183/14 <b>tens [2]</b> 18/18 142/5 <b>term [28]</b> 4/13 23/19 57/13 61/8 67/9 67/12 72/12 83/23 99/10 122/17 127/5 127/19 127/23 128/1 131/7 133/11 135/11 136/16 170/17 185/16 185/19 193/16 193/19 193/20 193/24 194/4 194/6 194/22 <b>terminal [1]</b> 78/9 <b>terminating [1]</b> 92/20 <b>terminology [2]</b> 120/2 185/1 <b>terms [79]</b> 7/13 7/21 8/15 12/8 16/22 17/1 17/14 17/14 20/6 21/20 21/23 22/22 23/11 26/7 26/14 26/17 26/21 27/13 27/15 27/16 27/22 34/23 44/14 50/7 57/18 72/16 73/6 73/12 73/23 79/22 81/11 85/14 94/1 94/8 95/3 98/19 100/3 101/20 102/11 102/14 103/18 103/24 104/21 106/8 107/1 107/12 108/3 108/3 113/14 113/23 114/23 115/7 115/9 115/20 115/23 119/22 121/11 124/2 130/9 131/25 132/2 133/11 134/6 150/5 154/3 154/13 156/24 161/11 175/1 175/15 177/3 180/15 180/21 185/11 188/1 188/14 188/20 191/24 194/18 <b>terotin [1]</b> 54/2 <b>terrible [4]</b> 55/4 55/4 59/14 96/19 <b>test [23]</b> 14/9 18/6 22/21 23/1 25/22 34/2 34/7 39/10 91/13 120/14 120/16 155/7 155/11 159/1 159/11 159/18 159/22 159/23 160/18 163/23 164/18 166/1 177/20 <b>tested [3]</b> 93/1 168/15 177/23 <b>testimony [1]</b> 91/24 <b>testing [7]</b> 14/19 155/9 155/12 158/24 159/4 159/21 160/1 <b>tests [12]</b> 14/22 23/11 43/6 49/17 120/7 120/12 149/14 156/25 157/2 163/24 167/15	194/24 <b>text [1]</b> 187/8 <b>texts [1]</b> 176/11 <b>than [53]</b> 15/12 15/20 16/24 20/8 20/21 26/12 35/23 42/22 47/15 54/7 64/14 65/20 69/4 74/3 75/14 81/6 89/20 101/6 103/18 103/25 104/24 106/16 112/22 114/11 116/10 118/11 118/24 121/7 123/1 123/9 123/11 124/14 128/16 130/16 131/9 131/12 133/8 134/13 139/15 140/10 146/3 150/13 155/10 160/18 167/14 174/24 175/21 175/24 177/24 178/8 190/5 190/24 190/25 <b>than affecting [1]</b> 65/20 <b>thank [12]</b> 1/11 2/19 4/14 5/3 5/25 63/18 136/24 143/22 148/17 196/1 196/13 196/19 <b>thankfully [2]</b> 57/10 60/12 <b>thanking [1]</b> 196/13 <b>that [959]</b> <b>that's [100]</b> 6/8 9/8 10/12 11/1 11/8 13/13 14/24 18/2 18/5 23/22 31/3 33/19 34/13 34/15 34/20 36/24 39/7 39/8 43/1 47/4 47/16 48/14 49/9 54/6 55/1 56/14 60/25 62/4 62/6 66/24 79/10 85/6 85/23 87/2 87/25 89/9 89/23 91/3 92/20 99/9 101/9 117/18 118/18 119/22 120/25 121/15 125/1 128/8 129/13 131/17 132/13 132/19 137/11 137/17 137/17 138/1 138/4 138/16 139/13 141/1 141/2 141/3 141/5 141/17 141/24 142/22 143/2 143/7 143/21 144/17 144/23 146/7 148/16 151/5 152/23 157/7 160/20 161/23 164/19 166/12 170/1 170/1 170/15 170/24 171/15 173/19 176/18 177/7 178/4 178/7 181/24 182/5 183/4 185/9 185/20 187/25 190/3 191/3 194/12 194/16 <b>the panel [1]</b> 91/1	<b>their [94]</b> 2/18 10/4 12/16 12/21 13/25 17/14 21/5 21/9 26/18 26/24 30/9 33/19 34/5 35/24 37/1 38/20 40/10 43/17 44/2 48/19 49/2 52/10 53/16 53/20 62/21 75/17 75/20 75/20 78/13 81/20 84/4 84/5 84/12 84/12 84/18 85/11 86/4 86/9 86/18 87/7 89/13 91/24 96/9 97/10 103/6 104/24 108/3 108/4 109/20 111/17 112/12 113/1 114/14 114/14 116/15 117/2 117/15 123/15 123/24 124/5 126/5 127/8 132/7 132/8 134/23 136/9 136/10 149/13 150/6 150/21 150/25 151/15 151/15 154/13 156/2 156/2 156/16 157/3 161/11 165/8 167/1 167/2 168/6 168/21 168/23 169/1 171/11 171/11 172/23 179/15 185/18 186/18 191/13 195/21 <b>them [67]</b> 6/18 6/19 7/12 10/19 15/1 33/2 36/25 38/2 40/22 41/9 43/5 44/5 46/17 50/21 51/23 67/17 69/19 82/1 84/14 98/23 98/25 100/1 103/2 104/7 106/12 114/10 114/11 114/24 115/18 117/4 117/11 124/15 124/16 125/19 141/15 144/24 146/4 151/6 152/15 153/2 153/6 153/11 154/2 154/3 154/4 154/6 155/18 155/18 155/20 155/21 156/1 156/5 156/7 156/15 157/6 158/11 158/12 162/2 162/3 167/19 168/9 168/23 168/24 169/1 170/19 187/3 196/7 <b>theme [1]</b> 58/5 <b>themselves [8]</b> 67/20 72/10 77/22 119/23 150/6 150/20 151/11 152/16 <b>then [153]</b> 2/14 3/24 4/22 6/16 10/2 10/8 10/12 10/14 10/23 15/11 15/19 16/5 17/2 17/5 17/9 18/8 18/12 18/20 22/1 22/21	24/23 25/24 26/3 28/20 30/12 32/5 32/7 36/5 37/19 41/22 45/10 46/19 46/23 47/19 48/13 49/12 51/1 51/6 52/21 53/2 57/13 57/23 58/19 59/19 59/19 61/8 63/3 66/19 68/17 68/17 68/19 68/20 70/8 70/16 70/22 71/16 73/24 74/1 74/12 74/21 75/2 78/18 79/7 84/24 86/4 91/12 92/1 93/17 94/25 96/9 100/20 100/24 101/17 102/6 102/7 102/12 102/25 103/23 104/8 104/15 105/3 105/6 105/11 109/3 109/9 112/16 113/13 114/21 114/24 115/12 115/24 115/24 116/4 120/9 120/10 120/10 127/6 128/16 128/20 129/1 130/1 132/8 132/21 137/1 139/1 139/1 139/2 139/3 139/4 139/9 140/11 140/13 140/14 140/16 140/20 140/24 141/4 141/20 143/6 143/25 144/18 144/23 146/4 146/10 148/13 148/13 148/15 149/5 149/5 149/9 149/25 152/14 153/23 156/15 156/23 158/9 159/17 162/4 164/5 164/7 164/9 165/14 165/18 166/19 167/4 167/5 168/5 169/1 174/2 177/5 182/15 184/12 191/2 <b>then they [1]</b> 59/19 <b>theoretically [1]</b> 15/11 <b>theory [1]</b> 173/20 <b>therapeutic [2]</b> 108/14 108/25 <b>therapies [8]</b> 41/3 41/5 92/6 92/7 99/16 113/2 122/25 122/25 <b>therapy [83]</b> 5/1 34/10 35/17 39/19 41/19 43/8 43/10 50/9 60/10 66/5 68/23 80/24 90/12 93/21 100/24 101/15 101/19 101/22 102/2 102/15 104/8 104/10 105/25 106/4 109/19 109/22 109/23 110/24 113/1 113/15 114/15 114/25 115/4 115/5 115/13 115/15
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<b>T</b>	103/11 103/19 104/18 105/7 106/3 106/9 107/25 111/4 111/7 111/8 113/2 113/8 113/10 114/8 123/22 124/3 124/9 125/18 128/20 129/23 130/24 133/24 136/15 136/16 153/23 156/12 156/13 171/14 174/19 174/19 174/22 180/2 183/19 184/16 192/9 <b>they</b> [231] <b>they'd</b> [1] 100/17 <b>they're</b> [4] 151/7 151/7 163/25 167/1 <b>they've</b> [2] 136/9 156/9 <b>thing</b> [35] 7/23 15/9 15/14 34/13 38/18 43/25 49/7 55/1 56/8 56/13 57/9 58/18 59/24 64/8 74/23 77/3 79/15 82/13 82/24 102/13 110/9 111/13 113/7 122/10 123/3 124/25 125/13 146/1 155/25 156/6 158/18 166/21 169/11 171/4 190/20 <b>things</b> [55] 19/9 27/14 34/12 38/6 38/17 38/24 39/4 41/2 41/11 41/12 41/16 44/4 44/10 44/14 46/9 46/19 48/8 48/10 48/23 61/7 62/24 67/16 71/4 71/15 71/16 71/19 71/20 85/13 86/16 97/6 103/20 109/15 110/6 111/1 114/21 116/1 118/8 124/8 124/17 126/11 126/17 142/17 163/2 165/14 166/8 167/16 172/2 187/20 188/11 189/1 189/2 191/5 194/19 194/23 195/4 <b>think</b> [205] <b>thinking</b> [9] 47/17 47/22 48/18 59/12 60/7 63/9 85/2 114/7 164/23 <b>third</b> [2] 12/23 139/3 <b>thirds</b> [1] 13/1 <b>this</b> [149] 1/6 9/20 15/18 16/8 17/12 19/12 19/23 20/20 21/1 22/2 25/18 28/25 30/11 30/12 31/5 31/13 31/15 31/17 31/24 32/5 32/20 33/3	33/5 33/5 35/16 35/17 35/17 36/2 38/9 40/7 41/8 43/17 44/10 44/11 44/24 48/18 48/22 49/2 49/8 50/12 52/1 52/12 52/15 53/3 54/11 54/16 55/14 55/15 56/3 56/9 59/7 59/15 61/14 62/6 66/7 66/10 67/8 68/7 72/14 73/17 74/12 75/14 77/8 78/16 79/9 80/6 80/11 82/13 83/7 83/18 83/21 84/1 84/10 86/8 86/25 87/3 90/1 91/21 93/3 94/6 94/15 96/15 97/23 98/18 100/2 100/11 102/25 103/23 106/11 107/13 110/17 111/2 111/6 113/18 122/6 123/17 124/25 125/2 125/4 126/12 126/21 131/21 132/2 132/20 133/3 133/4 136/11 136/20 137/12 138/13 138/18 140/24 144/1 150/14 151/15 152/4 152/8 152/18 152/24 154/7 155/3 156/6 158/14 158/20 161/8 161/15 162/11 162/13 165/7 169/12 171/9 171/10 171/12 172/9 172/10 172/11 172/11 173/6 174/13 174/18 178/5 178/17 185/6 186/22 187/9 187/14 192/3 193/10 195/12 <b>Thomas'</b> [1] 3/18 <b>those</b> [130] 1/6 2/21 4/1 12/23 14/22 17/23 17/23 19/9 19/22 20/9 21/7 21/8 21/9 21/13 24/23 27/5 29/19 29/25 34/3 35/4 38/3 39/18 41/5 41/20 42/7 42/15 43/8 43/13 44/25 45/19 46/4 46/16 46/19 48/4 49/25 54/24 58/3 62/6 64/24 65/1 69/2 73/22 74/8 77/25 78/3 78/12 79/13 79/20 80/20 80/20 82/1 86/12 86/13 87/4 88/14 88/18 88/20 91/1 91/2 91/18 91/23 93/11 93/12 95/21 96/21 96/23 96/24 96/25 97/1 103/25 105/8 109/7 113/3 115/17 115/19 116/23 117/14	122/22 122/25 122/25 124/24 126/25 127/22 129/18 131/10 132/21 132/23 133/19 134/5 134/12 134/13 138/21 141/12 144/5 145/4 148/24 149/3 151/25 154/5 157/11 157/17 159/14 159/17 162/7 162/10 163/1 163/16 169/2 172/17 173/8 175/15 177/23 178/23 180/9 182/18 184/7 186/24 187/18 188/6 188/12 188/25 189/1 190/10 191/11 192/12 193/4 193/8 193/16 195/10 195/14 <b>though</b> [5] 25/21 85/19 138/17 171/22 181/14 <b>thought</b> [8] 72/19 73/5 79/9 130/14 143/21 145/8 156/12 184/12 <b>thousand</b> [1] 150/17 <b>thousands</b> [2] 18/18 18/19 <b>threatening</b> [1] 184/10 <b>three</b> [24] 17/7 19/6 21/4 22/22 22/25 44/5 45/25 46/4 104/20 106/20 108/22 122/1 135/8 138/23 141/10 141/10 141/13 146/3 146/18 146/19 147/5 147/25 158/9 169/4 <b>threshold</b> [1] 37/8 <b>thresholds</b> [1] 36/20 <b>throat</b> [1] 19/15 <b>thrombocytopenic</b> [1] 39/5 <b>through</b> [57] 3/24 8/23 25/21 28/11 31/21 36/15 46/1 46/6 48/18 49/17 50/2 50/4 50/16 51/11 52/10 54/12 55/18 57/1 62/7 75/16 75/22 76/14 77/15 77/25 81/10 83/3 86/25 100/18 106/12 107/9 107/10 112/15 118/4 119/16 120/24 129/6 131/18 138/14 138/25 139/8 150/13 150/15 151/1 151/9 151/19 153/3 155/19 160/24 164/13 167/24 172/8 172/22 173/3 178/23 180/19 181/9 193/5 <b>throughout</b> [1] 18/12	<b>thrush</b> [6] 40/1 41/2 42/9 42/10 50/24 51/8 <b>Thursday</b> [1] 1/1 <b>thus</b> [1] 100/23 <b>tick</b> [1] 82/17 <b>ticking</b> [1] 82/15 <b>tide</b> [1] 110/23 <b>time</b> [92] 1/10 2/10 3/3 3/9 4/8 5/13 12/8 22/5 22/24 23/13 25/9 28/7 43/7 48/21 61/22 63/16 65/11 65/13 71/10 71/16 74/18 75/10 76/17 78/9 80/10 83/4 85/25 88/6 89/4 89/23 93/22 98/5 98/16 98/23 98/25 102/10 102/24 103/16 103/20 104/25 105/24 109/19 110/22 114/8 114/11 114/15 114/18 116/1 116/13 117/8 117/21 119/5 119/20 121/24 122/12 123/2 123/19 123/22 125/5 125/19 125/21 126/14 126/18 126/23 127/8 129/10 132/6 132/20 133/8 133/10 136/20 136/21 149/13 151/6 151/19 151/20 152/15 153/16 163/19 163/22 163/22 164/4 164/21 165/11 184/24 185/2 186/20 187/2 189/9 189/16 191/19 196/14 <b>time-limited</b> [1] 102/10 <b>time-line</b> [1] 25/9 <b>timely</b> [2] 123/14 156/4 <b>timers</b> [1] 96/16 <b>times</b> [7] 10/9 68/6 98/19 106/20 108/16 108/22 169/4 <b>timetables</b> [1] 1/10 <b>Timothy</b> [1] 174/23 <b>tired</b> [1] 41/13 <b>tiredness</b> [1] 19/17 <b>tissue</b> [2] 65/21 70/25 <b>title</b> [2] 2/4 29/1 <b>to</b> [1020] <b>today</b> [17] 60/12 66/20 80/13 80/21 86/23 87/2 87/10 97/6 97/13 97/25 111/17 116/9 116/13 117/8 124/19 127/2 135/23 <b>today's</b> [3] 14/25 103/15 126/13 <b>together</b> [7] 13/23 63/15 88/3 147/22 162/17 164/5 166/1	<b>toilet</b> [1] 56/4 <b>told</b> [8] 37/9 115/10 122/21 136/8 142/9 142/10 149/2 149/13 <b>tolerable</b> [3] 114/4 114/22 114/23 <b>tolerate</b> [4] 103/16 107/8 110/25 124/14 <b>tolerated</b> [7] 106/16 107/17 108/10 116/15 124/4 126/16 127/7 <b>tombstone</b> [1] 151/13 <b>tomorrow</b> [7] 135/24 149/1 180/9 193/8 193/11 193/13 196/20 <b>tongue</b> [2] 40/1 42/19 <b>too</b> [5] 10/22 58/15 58/25 59/2 112/2 <b>took</b> [1] 95/12 <b>tool</b> [1] 32/24 <b>top</b> [5] 72/4 110/9 110/15 137/16 144/4 <b>topic</b> [1] 137/6 <b>totally</b> [1] 147/13 <b>touched</b> [1] 60/15 74/12 74/14 87/3 88/8 89/25 92/2 123/12 182/25 186/22 <b>toxic</b> [7] 54/2 58/4 58/8 90/5 94/22 97/23 103/7 <b>toxicities</b> [3] 89/16 97/16 185/8 <b>toxicity</b> [7] 58/10 88/19 90/9 94/1 98/8 183/16 186/24 <b>toxo</b> [1] 70/22 <b>toxoplasmosis</b> [8] 47/10 47/15 47/18 67/2 67/12 68/20 69/18 70/20 <b>train</b> [2] 143/3 143/20 <b>trained</b> [2] 5/4 5/6 <b>training</b> [1] 157/17 <b>transcript</b> [1] 138/5 <b>transcriptase</b> [13] 92/9 92/10 92/16 93/4 94/11 104/13 104/15 141/11 141/19 142/12 146/14 146/22 147/16 <b>transcribing</b> [1] 143/9 <b>transcription</b> [8] 139/14 139/16 139/19 140/24 141/20 145/10 145/15 147/15 <b>transfer</b> [1] 84/25 <b>transferred</b> [1] 181/12 <b>transform</b> [1] 145/20 <b>transformative</b> [2] 121/18 122/8 <b>transformed</b> [2] 56/14 121/21
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<b>T</b>	164/19 164/24 165/8 167/16 167/24 168/1 168/2 169/23 169/25 170/14 172/25 173/5 175/6 179/5 179/22 180/4 181/23 182/15 182/17 183/23 185/4 185/10 185/19 189/3 <b>treatments [33]</b> 38/3 40/17 41/14 42/24 43/14 50/1 50/3 50/19 52/19 53/23 54/2 58/4 59/18 61/7 61/20 61/22 67/17 67/20 67/22 67/22 67/24 68/1 68/3 74/8 89/8 92/6 94/5 136/16 142/9 150/9 182/18 182/22 186/24 <b>tremendously [2]</b> 44/25 59/13 <b>trial [12]</b> 93/7 93/9 101/19 103/12 105/19 105/19 114/12 114/16 115/3 184/15 184/16 184/18 <b>trials [19]</b> 5/1 67/18 94/2 97/8 103/17 103/21 104/20 104/22 104/25 105/23 112/21 120/21 122/16 128/13 128/13 129/16 129/17 147/25 148/3 <b>tried [5]</b> 16/7 67/18 67/19 109/10 134/19 <b>tries [2]</b> 18/20 24/19 <b>triple [12]</b> 104/8 104/10 105/24 106/3 109/19 109/22 110/24 113/15 121/18 145/8 146/7 146/18 <b>tropic [1]</b> 30/2 <b>true [10]</b> 14/14 22/4 37/3 66/24 70/11 85/6 88/7 127/3 177/8 182/6 <b>truly [1]</b> 108/9 <b>trumped [1]</b> 135/23 <b>trust [2]</b> 5/24 152/5 <b>truth [1]</b> 80/12 <b>Truvada [2]</b> 183/8 183/11 <b>try [17]</b> 17/20 31/20 49/18 54/17 56/2 59/18 61/9 61/11 66/9 85/21 94/4 96/14 100/6 111/11 155/17 174/18 186/9 <b>trying [23]</b> 24/8 24/9 48/7 48/17 69/14 69/22 75/20 82/1 86/13 86/18 95/4 98/19 112/3 119/19	126/15 133/24 142/25 143/1 157/6 164/5 174/15 186/17 189/18 <b>tube [4]</b> 51/2 112/14 143/3 143/20 <b>tuberculosis [3]</b> 46/23 46/25 47/2 <b>tubes [2]</b> 51/11 112/14 <b>TUDOR [6]</b> 1/14 2/1 2/25 112/20 134/2 150/1 <b>TUDOR-WILLIAMS [1]</b> 1/14 <b>Tuesday [3]</b> 75/17 77/5 136/12 <b>tumour [1]</b> 49/14 <b>turn [6]</b> 49/5 90/24 95/7 137/5 193/25 195/15 <b>turned [1]</b> 37/15 <b>Turning [3]</b> 28/20 37/19 132/21 <b>twice [5]</b> 94/2 102/20 110/18 117/3 176/24 <b>two [51]</b> 1/5 3/20 6/1 7/4 7/7 11/4 13/1 17/22 18/8 18/9 19/7 23/5 28/8 36/13 36/14 44/5 88/2 90/18 101/18 103/7 103/18 103/25 103/25 104/20 104/23 104/24 111/19 116/14 128/3 139/21 140/1 145/13 146/13 147/23 148/4 150/16 154/15 158/8 159/8 160/2 160/3 167/19 172/20 173/2 174/19 174/25 181/6 189/1 189/2 192/10 194/2 <b>Two o'clock [1]</b> 90/18 <b>type [6]</b> 15/12 15/20 15/25 28/22 29/11 29/17 <b>types [1]</b> 13/12 <b>typically [1]</b> 169/5 <b>typo [1]</b> 191/21	77/14 117/24 <b>unacceptable [1]</b> 83/21 <b>unanswered [1]</b> 189/11 <b>unborn [1]</b> 9/4 <b>uncertainty [1]</b> 189/17 <b>unclear [4]</b> 4/19 51/7 67/7 189/22 <b>uncomfortable [3]</b> 42/18 65/16 186/20 <b>uncommon [4]</b> 20/4 21/20 39/19 70/8 <b>under [3]</b> 61/9 129/17 168/25 <b>under-represented [1]</b> 129/17 <b>underestimate [3]</b> 121/18 125/1 136/1 <b>undergoing [1]</b> 174/20 <b>underlying [3]</b> 49/12 49/19 185/18 <b>understand [15]</b> 33/6 50/8 103/10 124/3 134/6 137/7 137/13 147/2 152/16 153/19 161/10 174/4 175/5 180/2 189/13 <b>understandable [1]</b> 187/4 <b>understanding [6]</b> 63/2 84/22 125/25 137/15 155/23 189/12 <b>understood [1]</b> 87/20 <b>undertakers [2]</b> 86/17 86/19 <b>undertook [1]</b> 3/20 <b>undetectable [16]</b> 115/20 117/15 119/4 119/7 120/1 120/3 120/14 121/4 123/23 152/19 152/23 168/10 172/25 179/1 179/23 180/5 <b>undiagnosed [1]</b> 20/13 <b>undifferentiated [1]</b> 47/24 <b>undoubtedly [1]</b> 99/1 <b>unexpected [2]</b> 132/4 135/17 <b>unfortunately [3]</b> 22/1 100/15 113/4 <b>uniform [1]</b> 10/13 <b>uninfected [1]</b> 120/23 <b>unit [3]</b> 3/17 5/12 80/3 <b>United [2]</b> 93/7 190/7 <b>United Kingdom [1]</b> 190/7 <b>United States [1]</b> 93/7 <b>universal [2]</b> 161/1	180/21 <b>universally [1]</b> 27/23 <b>University [1]</b> 4/22 <b>unmet [2]</b> 80/23 81/1 <b>unpleasant [2]</b> 54/1 54/4 <b>unprotected [1]</b> 11/16 <b>unrecognised [2]</b> 19/11 20/2 <b>until [11]</b> 22/25 46/18 60/24 89/6 105/21 105/21 106/18 108/7 115/2 186/25 196/25 <b>untransmissible [1]</b> 152/20 <b>untransmittable [2]</b> 119/6 120/18 <b>untreated [3]</b> 17/6 35/18 187/22 <b>unusual [9]</b> 11/1 11/10 13/8 15/6 15/8 61/13 77/11 146/10 170/23 <b>unwell [5]</b> 19/8 78/2 109/17 125/21 166/17 <b>unwise [1]</b> 135/15 <b>up [44]</b> 2/2 5/11 11/24 14/9 21/4 23/8 31/11 39/12 44/9 53/6 74/2 78/22 81/2 83/10 87/15 96/16 102/7 109/14 109/15 111/2 112/13 113/3 113/13 119/13 123/20 126/17 130/19 135/5 137/23 149/9 150/24 152/20 153/2 156/25 158/16 158/21 159/11 162/1 162/7 169/9 171/24 181/24 188/11 193/11 <b>up-to-date [1]</b> 162/1 <b>upon [1]</b> 82/12 <b>upper [1]</b> 57/21 <b>us [35]</b> 1/11 27/12 29/14 31/13 46/12 46/14 46/17 54/11 57/2 69/10 71/11 76/2 82/3 85/5 90/17 92/5 100/25 104/9 106/13 108/2 112/4 113/13 115/10 122/21 128/10 134/8 136/25 142/9 142/10 150/7 150/23 151/10 152/5 189/12 195/12 <b>use [27]</b> 4/13 5/18 9/7 18/5 22/19 23/7 23/19 24/1 29/25 30/3 34/7 34/12 42/16 47/9 54/4 58/9 61/25 97/8 97/13 98/6 107/7 119/8 128/25 142/24 176/12 186/9 192/18	<b>used [34]</b> 22/19 22/21 23/14 31/16 31/19 31/25 32/21 32/23 34/13 42/13 43/12 47/13 57/9 58/4 58/11 63/10 63/12 72/9 89/17 94/2 94/3 96/8 96/16 97/13 97/25 99/5 102/17 106/25 107/2 124/14 124/20 155/10 184/22 190/5 <b>useful [3]</b> 34/7 56/11 98/4 <b>users [1]</b> 121/12 <b>uses [2]</b> 29/12 30/1 <b>using [6]</b> 8/12 79/22 98/3 110/21 173/6 186/12 <b>usually [12]</b> 9/9 21/5 28/16 30/4 30/14 34/9 52/14 64/13 70/14 162/23 169/17 173/3 <b>utterly [1]</b> 112/7
				<b>V</b>	
				<b>vaginal [5]</b> 8/24 11/16 12/22 42/21 42/24 <b>vaguely [1]</b> 84/15 <b>valid [1]</b> 85/23 <b>value [1]</b> 196/12 <b>Vancouver [2]</b> 106/1 110/3 <b>variable [2]</b> 40/20 190/22 <b>variation [1]</b> 10/25 <b>varies [1]</b> 11/1 <b>variety [7]</b> 7/7 32/10 38/11 42/10 43/12 159/5 162/14 <b>various [1]</b> 14/5 <b>vary [2]</b> 172/16 192/9 <b>vein [1]</b> 58/15 <b>veins [2]</b> 58/11 58/16 <b>venous [1]</b> 58/13 <b>vent [1]</b> 67/7 <b>version [2]</b> 128/5 128/8 <b>versions [1]</b> 183/13 <b>versus [4]</b> 104/20 114/15 115/3 147/25 <b>vertical [5]</b> 9/10 12/14 121/1 121/3 150/12 <b>vertically [1]</b> 9/3 <b>very [197]</b> <b>via [2]</b> 121/8 178/2 <b>view [7]</b> 36/1 56/2 56/6 87/19 111/7 135/22 196/5 <b>vilified [1]</b> 186/12 <b>viral [80]</b> 9/13 12/25 13/1 16/5 16/10 16/13 17/3 18/10 18/14 19/5 22/16 24/24 25/1	



<b>V</b>	<b>visit</b> [2] 165/8 166/2 <b>visits</b> [3] 164/6 165/24 166/1 <b>visual</b> [1] 187/12 <b>voices</b> [2] 76/18 83/12 <b>vomiting</b> [2] 95/16 104/6	177/1 196/1 <b>ways</b> [2] 43/20 175/25 <b>we</b> [365] <b>we'll</b> [4] 12/9 52/9 52/10 168/25 <b>we're</b> [36] 25/25 31/13 31/25 32/3 32/5 34/1 47/16 50/1 50/11 52/16 63/23 69/14 69/22 71/12 71/19 73/13 76/7 77/18 83/13 83/14 136/1 136/2 145/7 147/23 152/21 155/22 157/5 169/4 174/6 174/25 179/24 181/22 182/11 182/17 189/15 194/25 <b>we've</b> [21] 16/7 28/24 46/15 46/15 46/17 73/11 74/12 74/14 76/6 92/2 111/8 113/12 117/22 136/11 152/1 154/14 154/15 161/14 176/10 178/5 194/23 <b>weak</b> [6] 34/25 47/2 47/19 48/24 48/25 60/24 <b>weakening</b> [4] 38/13 46/11 57/5 177/5 <b>weakens</b> [1] 46/19 <b>weaker</b> [1] 50/25 <b>website</b> [1] 192/7 <b>week</b> [2] 1/6 166/3 <b>weeks</b> [13] 17/22 18/8 18/9 19/7 21/4 21/12 21/22 23/6 158/9 159/8 160/2 167/13 167/19 <b>weight</b> [8] 19/19 41/13 51/25 52/1 55/8 152/13 168/21 168/22 <b>weight and</b> [1] 55/8 <b>welcome</b> [3] 1/4 1/7 1/7 <b>well</b> [69] 2/6 8/4 13/12 16/3 16/16 16/18 22/7 25/7 25/17 26/10 26/22 31/7 34/4 34/20 35/9 39/22 48/2 54/23 57/23 58/20 58/21 62/18 66/16 67/7 68/5 68/11 69/21 72/8 74/15 79/9 81/3 84/20 96/18 101/1 103/22 109/4 109/17 121/5 123/10 124/25 127/6 128/11 129/12 129/15 129/17 131/2 132/18 137/23 144/10 147/8 148/8 151/4 153/9 160/3 161/7 161/17 163/15 164/10 169/24	171/5 172/24 173/13 174/19 178/12 184/6 186/17 188/8 194/20 195/3 <b>well-being</b> [1] 195/3 <b>well-recognised</b> [1] 22/7 <b>went</b> [7] 59/23 102/4 115/8 120/9 120/10 120/11 185/17 <b>were</b> [184] 1/6 3/25 6/5 12/19 14/6 14/7 14/12 14/12 22/11 30/24 30/24 35/14 36/12 37/10 40/17 41/5 41/15 41/18 41/24 42/23 43/3 43/14 43/19 44/25 45/10 45/14 49/24 49/25 50/20 53/25 54/1 55/11 58/4 58/6 58/9 58/12 59/1 59/7 59/11 59/23 61/24 61/24 62/24 64/25 67/20 67/21 68/3 68/12 73/25 74/18 75/8 75/16 75/16 75/22 76/8 76/9 77/12 77/14 77/14 79/7 79/14 79/17 79/19 79/24 79/25 80/1 80/2 81/13 81/17 82/4 82/8 82/14 84/5 84/21 85/8 86/1 86/13 86/21 88/21 88/21 89/11 89/17 91/4 92/22 93/16 94/10 94/18 94/19 95/2 95/2 95/2 95/5 95/5 95/25 96/4 96/7 96/13 96/22 97/17 99/3 99/5 99/15 100/21 102/1 102/10 102/20 102/21 103/4 103/11 103/15 104/5 104/18 104/23 105/4 106/10 106/21 107/4 107/16 108/2 108/10 109/5 109/12 109/13 109/15 110/2 110/21 110/21 111/3 111/4 111/5 111/9 111/19 112/6 112/11 112/11 112/17 112/22 113/1 113/2 113/20 114/2 119/12 125/9 131/7 134/5 134/12 134/14 134/16 136/4 136/9 149/13 149/15 150/12 150/14 150/18 150/18 150/19 150/20 150/21 153/5 155/6 166/7 174/9 174/12 181/17 182/3 182/15 182/18	182/20 183/25 184/4 184/18 184/19 185/12 186/16 186/25 187/5 189/10 192/16 192/20 192/22 195/4 195/20 196/8 <b>weren't</b> [5] 2/22 41/14 82/15 91/2 113/10 <b>West</b> [3] 7/18 29/6 29/7 <b>West Africa</b> [3] 7/18 29/6 29/7 <b>western</b> [1] 63/5 <b>what</b> [154] 6/17 7/10 7/13 7/13 8/20 9/13 17/12 22/12 23/20 24/3 24/7 24/17 25/2 26/2 31/3 31/13 31/13 34/12 35/3 37/14 37/24 38/1 38/2 44/23 44/24 46/8 46/15 48/3 48/18 48/20 49/9 49/18 49/24 50/6 51/13 51/22 55/17 56/14 58/11 59/13 66/3 69/14 73/20 75/21 76/9 77/14 78/2 80/12 81/24 82/7 83/20 87/20 88/1 91/7 91/13 91/20 92/14 93/3 94/9 100/5 100/11 100/15 108/11 111/10 112/10 112/11 114/7 115/25 117/11 117/16 117/23 118/14 118/18 118/21 120/12 121/21 122/2 123/24 127/5 127/23 128/14 129/22 130/22 131/6 131/12 131/21 131/25 132/10 132/14 132/22 135/15 137/10 137/13 138/8 139/11 141/5 142/12 143/11 143/21 144/12 145/8 145/19 148/10 149/10 151/16 152/18 153/8 154/1 154/9 154/18 154/22 155/6 155/16 157/7 157/10 157/17 158/10 161/8 161/20 161/21 162/2 162/7 162/8 162/12 162/25 163/3 163/4 163/5 164/8 164/24 165/25 167/5 167/5 168/13 171/16 171/17 173/8 174/10 174/25 175/15 181/1 181/7 185/9 185/11 187/14 187/16 187/18 189/4 189/17 190/6 192/2 192/11 195/24 196/10	<b>what's</b> [15] 30/12 32/25 48/7 86/5 100/12 126/13 131/2 132/2 154/24 158/3 161/20 162/23 162/24 166/15 191/4 <b>whatever</b> [4] 49/13 79/6 172/14 179/24 <b>whatsoever</b> [1] 159/8 <b>when</b> [82] 5/7 6/25 10/9 17/13 17/16 20/18 20/18 22/11 22/15 26/10 26/12 26/13 29/7 30/14 31/25 34/1 35/2 37/10 39/6 41/4 41/19 47/22 47/22 48/24 51/21 52/12 53/25 61/16 61/17 61/24 62/14 64/13 67/10 70/17 75/21 77/6 86/25 87/21 100/20 101/16 102/24 104/10 108/8 108/19 110/14 111/3 111/10 113/18 125/3 125/6 126/23 127/1 128/14 132/6 140/14 141/24 142/15 142/15 144/25 145/7 146/17 150/9 151/8 152/10 154/10 155/1 155/14 161/21 168/19 172/7 173/24 174/8 174/15 174/22 181/3 181/23 188/10 189/6 189/9 189/16 189/21 195/20 <b>where</b> [95] 5/15 6/20 8/21 11/7 13/22 14/6 15/9 16/1 18/11 18/17 21/7 22/20 24/23 24/25 25/25 31/20 32/1 32/6 32/8 32/18 33/3 34/12 41/21 44/18 45/15 46/14 46/25 47/16 48/2 48/19 49/25 51/2 56/13 56/19 57/17 57/22 57/23 58/2 63/20 73/14 78/11 78/17 79/5 79/18 81/7 81/10 86/22 96/7 100/12 100/18 106/13 106/14 108/18 115/2 115/4 116/5 117/9 121/16 121/23 124/16 128/22 128/23 132/19 133/23 137/8 137/17 147/24 152/2 152/16 154/12 155/8 158/23 160/1 160/15 163/7 166/1 166/8 167/14 170/25 171/2 171/2 174/1 175/17 179/4
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(76) viral... - where



<b>W</b>	173/16 173/23 176/18 176/20 178/1 179/22 181/19 185/3 186/1 186/6 187/24 191/22 194/10 195/1 195/12 196/11 196/12 <b>while</b> [4] 10/10 25/13 47/3 78/1 <b>whilst</b> [2] 97/15 135/16 <b>white</b> [4] 6/23 29/17 42/18 42/19 <b>who</b> [155] 1/6 2/18 2/21 3/25 4/1 4/1 5/18 9/7 10/14 10/24 10/25 11/14 12/3 13/7 13/20 14/12 14/16 15/2 16/15 20/19 26/16 27/4 29/5 29/6 30/5 30/7 30/7 30/9 31/24 35/21 35/24 40/10 46/24 47/12 51/20 54/11 56/8 58/6 59/1 59/2 59/7 59/11 59/21 61/15 62/21 63/4 64/9 64/10 64/11 64/19 64/20 66/18 67/6 74/4 74/21 75/16 76/2 76/13 76/15 78/7 78/14 80/9 81/6 82/4 82/25 83/15 86/12 86/14 86/17 86/18 87/4 87/10 88/15 89/12 91/23 93/8 93/11 93/11 93/12 96/9 97/5 98/11 98/14 99/15 99/15 99/17 99/18 100/19 103/1 107/7 109/18 111/17 111/24 115/17 115/19 116/23 117/8 117/12 117/14 121/4 123/8 123/21 123/23 126/4 129/4 129/9 131/21 131/23 133/6 133/9 134/14 134/21 135/8 135/12 136/3 136/4 150/12 150/14 150/17 150/21 150/22 155/7 159/1 159/22 159/22 160/14 160/22 161/3 167/10 167/23 169/25 172/21 172/22 173/3 175/11 178/8 179/11 180/18 180/18 181/16 181/17 182/18 183/6 184/3 184/4 184/9 184/17 184/19 186/16 188/19 194/10 194/11 194/11 194/14 194/15 <b>who's</b> [4] 18/17 130/23 152/7 161/4 <b>whole</b> [12] 33/14	36/19 76/14 78/20 78/23 82/7 83/19 85/17 121/20 136/5 147/18 151/13 <b>whom</b> [1] 1/5 <b>whose</b> [3] 47/1 51/19 150/20 <b>why</b> [15] 22/12 29/16 46/4 63/11 84/22 99/20 118/6 147/13 160/5 166/22 172/7 174/11 178/7 183/24 189/12 <b>wider</b> [3] 75/6 78/6 162/4 <b>widespread</b> [1] 86/20 <b>will</b> [104] 9/7 9/19 10/15 12/16 16/10 16/21 17/3 17/17 17/17 17/18 17/19 17/19 18/13 18/18 20/14 20/21 25/6 27/19 28/1 28/6 38/13 46/7 47/7 47/23 47/24 70/18 76/15 91/20 96/25 101/15 111/2 112/14 116/9 116/13 116/14 116/16 116/17 116/24 118/5 118/6 122/19 123/10 128/10 128/11 129/3 130/2 135/23 136/21 138/24 139/1 139/3 140/16 141/25 142/2 142/3 144/10 145/1 145/2 145/21 145/24 146/22 152/24 155/2 155/12 155/16 156/3 156/4 156/7 156/19 157/18 158/1 158/2 158/14 160/11 160/13 160/14 160/14 162/2 162/13 165/20 165/24 165/25 166/3 168/2 168/12 168/14 168/24 169/17 172/3 175/6 175/7 175/10 176/8 177/12 179/24 181/12 191/11 191/22 192/3 193/9 193/13 195/9 196/20 196/21 <b>WILLIAMS</b> [11] 1/14 1/18 2/1 2/25 4/15 4/16 23/12 95/24 112/20 134/2 150/1 <b>window</b> [3] 22/10 22/21 23/15 <b>wiping</b> [1] 182/12 <b>wish</b> [2] 37/3 196/18 <b>wishes</b> [1] 151/3 <b>with</b> [332] <b>withdrawn</b> [1] 99/6 <b>withhold</b> [2] 149/17	149/21 <b>within</b> [22] 7/8 13/19 13/19 18/1 18/3 76/9 81/18 82/23 94/17 117/4 126/2 159/7 159/9 159/13 159/14 159/17 159/19 159/25 163/10 163/19 168/14 176/5 <b>without</b> [9] 10/22 11/7 35/17 57/3 63/25 85/2 88/24 124/4 189/12 <b>woman</b> [4] 43/9 87/10 121/4 179/11 <b>women</b> [5] 5/17 43/3 58/23 129/15 178/16 <b>won't</b> [5] 12/4 87/13 143/25 154/25 160/9 <b>wondered</b> [1] 81/23 <b>wonderful</b> [1] 59/13 <b>work</b> [32] 3/5 3/8 3/13 4/23 8/1 8/1 8/4 8/7 26/10 48/7 49/12 49/15 86/23 88/18 91/11 95/4 97/12 102/12 111/18 112/4 116/19 142/14 143/17 143/25 148/12 159/11 160/20 161/23 165/1 171/2 173/15 194/18 <b>worked</b> [6] 5/12 8/6 8/8 92/21 94/14 125/8 <b>workers</b> [3] 81/21 82/21 86/7 <b>working</b> [10] 2/11 2/22 5/9 28/11 54/14 80/18 144/8 157/17 159/17 168/8 <b>works</b> [1] 146/8 <b>world</b> [11] 7/19 14/5 14/25 15/2 32/22 34/17 46/15 47/12 47/16 63/6 192/24 <b>Worldwide</b> [1] 8/22 <b>worried</b> [1] 169/11 <b>worries</b> [2] 169/8 169/16 <b>worry</b> [3] 126/23 150/10 169/12 <b>worrying</b> [1] 179/12 <b>worse</b> [3] 64/17 79/14 103/25 <b>worth</b> [13] 14/2 25/17 33/23 35/6 41/25 49/23 67/15 69/25 100/10 110/12 181/14 186/21 186/22 <b>would</b> [169] 7/23 10/19 11/15 11/20 11/23 11/25 12/20 12/24 13/6 13/8 15/8 16/5 16/11 16/13 16/23 16/24 16/25	18/21 20/6 22/24 22/24 28/5 32/23 34/9 37/12 37/25 38/2 38/4 40/19 40/23 40/23 40/25 41/3 41/8 42/13 42/16 42/17 42/22 43/6 43/12 48/5 48/15 49/7 49/16 50/18 51/3 51/6 51/9 52/16 52/25 53/2 53/8 53/15 53/22 54/4 55/3 58/13 58/15 58/17 59/16 59/17 59/19 59/21 61/1 61/6 63/17 65/2 66/3 66/10 66/14 66/14 67/12 68/6 68/7 68/8 68/9 69/17 70/19 71/10 78/6 78/8 84/11 84/13 85/7 88/1 90/1 90/4 90/5 90/7 90/10 93/19 95/4 96/9 98/20 98/22 98/23 99/24 103/16 104/4 108/7 108/17 108/21 114/10 120/8 121/10 123/13 123/16 124/11 124/16 132/4 134/12 135/14 145/9 151/4 151/10 154/22 155/6 155/8 155/17 155/19 158/12 159/16 160/15 160/25 161/1 161/4 161/5 161/21 162/25 163/21 163/23 163/24 164/1 164/2 164/3 165/5 165/7 165/16 166/2 167/18 167/22 167/25 168/4 168/8 168/10 170/7 171/12 175/18 175/21 177/18 177/19 179/2 179/7 179/8 182/9 182/12 182/22 183/24 184/8 184/19 185/6 187/14 188/10 189/11 189/12 190/20 191/12 192/12 194/19 <b>wouldn't</b> [6] 11/23 13/8 70/9 97/22 112/7 164/11 <b>write</b> [1] 156/11 <b>written</b> [3] 28/24 84/16 113/12 <b>wrong</b> [1] 137/8 <b>wrote</b> [3] 83/10 171/5 171/10 <b>Y</b>	14/16 36/13 89/3 116/11 117/9 117/10 123/11 125/7 126/12 127/16 127/21 128/21 131/10 132/22 133/7 134/24 142/5 151/16 152/1 152/9 174/24 174/25 196/11 <b>yes</b> [76] 9/20 10/21 12/1 19/24 20/16 20/22 21/21 27/7 28/13 28/24 33/11 33/14 36/6 42/3 42/5 42/23 44/12 45/1 49/4 49/22 51/19 66/2 67/11 71/10 73/11 73/11 76/10 83/8 87/25 89/23 90/9 90/22 94/4 95/9 102/3 103/14 110/1 112/1 113/15 116/19 119/7 123/7 127/4 132/17 133/22 138/6 138/23 139/17 139/20 141/9 143/2 143/2 143/5 143/21 144/17 144/25 146/11 146/23 147/9 147/10 149/4 153/22 157/14 157/20 157/23 158/22 176/1 177/13 179/4 180/1 182/13 183/18 191/15 192/14 193/12 196/22 <b>yesterday</b> [13] 1/6 2/22 15/10 22/10 81/3 82/16 87/16 87/17 88/8 88/12 91/3 174/7 175/17 <b>yet</b> [8] 22/17 90/1 127/25 151/8 165/6 165/7 165/11 175/13 <b>you</b> [498] <b>you'd</b> [2] 70/21 70/21 <b>you're</b> [5] 37/14 111/10 122/13 144/4 172/24 <b>you've</b> [8] 15/9 26/1 37/7 61/3 62/5 95/13 140/5 148/23 <b>young</b> [20] 2/11 2/17 37/6 76/4 111/8 113/6 126/16 134/21 135/4 135/12 136/5 150/5 150/11 150/24 151/3 151/10 151/22 152/6 153/7 168/20 <b>younger</b> [6] 26/10 26/12 131/11 134/13 134/18 153/12 <b>youngster</b> [1] 152/7 <b>your</b> [72] 2/2 2/3 16/5 16/6 17/7 19/12 22/2 23/19 25/4 25/6 25/7
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(77) where... - your

<b>Y</b> <b>your... [61]</b> 26/4 26/9 27/2 27/8 29/10 33/6 34/25 35/10 35/14 39/6 39/6 45/23 47/19 50/8 50/24 50/25 51/14 53/6 54/13 54/15 62/5 68/25 74/1 74/2 75/4 77/7 77/7 77/7 78/19 82/12 83/7 91/4 96/20 123/5 125/5 125/14 125/15 125/15 125/16 134/3 134/4 136/18 146/9 147/3 148/23 149/6 150/1 150/2 152/6 152/7 152/8 152/22 153/24 158/3 163/17 164/20 166/5 170/8 171/3 175/5 196/15 <b>yourselves [2]</b> 1/23 196/16 <b>youth [1]</b> 182/5 <b>Z</b> <b>zalcitabine [5]</b> 94/12 94/20 99/3 99/6 101/17 <b>Zambia [1]</b> 3/11 <b>zero [1]</b> 182/4 <b>zidovudine [13]</b> 92/23 93/9 93/18 93/21 97/9 97/13 97/19 99/7 101/5 101/6 102/17 104/6 142/18 <b>zip [6]</b> 143/23 143/24 143/25 144/4 144/6 144/7 <b>zoster [7]</b> 38/17 40/25 41/22 44/14 44/19 60/15 60/21					
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