Witness Name: GRO-B

Statement Number: WITN2475007

Dated: 6 April 2021

INFECTED BLOOD INQUIRY

THIRD WRITTEN STATEMENT OF GRO-B

I provide this statement in response to a request under Rule 9 of the Inquiry rules 2006 dated 1 October 2020. I adopt the paragraph numbering in the Rule 9 request for ease of reference.

I, GRO-B will say as follows:-

1.Introduction

1. This is my 3rd statement, and I apologise that it will need to be read in tandem alongside just page 1 – 8 of my First Witness Statement (FWS) dated 1st April 2019. I also made a very brief Supplemental Statement (SS) dated 23rd April 2019 which is represented here solely at 'How Affected' point 16 within this final statement. *My statements are anonymous re myself and my 2 hospitals, otherwise I would never have written them.*

2. Around the September 2019 Inquiry Hearings, I read that Sir Brian had written to express disappointment at lack of attention paid in a recent report into Hep C through blood transfusion, *'This approach risks perpetuating the failure to identify, test and diagnose people infected through transfusion.'* In talking to a blood group leader re finding my list of ten 1995 – 1997 raised ALTs (re Liver Function Tests results) for which I was never Hep C tested, nor for my 2008 – 2009 raised ALTs occurring whilst on biological drugs, we realised Hep C diagnosis through raised ALTs, or even a past history of raised ALTs (as underlying Hep C can also be quiet for long periods and not disturb ALT results) could still help to find some historically infected blood transfused people.

3. When I began my FWS I was overwhelmed with how much information I'd have to provide so I didn't request ALT records from my previous hospital and made only brief reference in less than 3 pages to both my 1995 – 1997, and also my 2008 – 2009 raised ALTs (for which I had only incomplete records, meaning some questions went unanswered). For me, the main point of my FWS had been to explain my resulting legacy of Hep C induced, MRI-evidenced and permanent MCD brain condition that my Consultant has also referred to as 'associated neurocognitive disorder related to hep C' (HCV AND).

4. Novembere 2019 I began typing my own notes, then from January 2020 I tried to understand and collate specific ALT records belatedly requested from my previous hospital. This statement is mostly my copy-typing of medical records, ALT research and 'point 16', but I absolutely struggled to retain sequences and facts of different paperwork, to identify then organise key items and merge with my own comments to provide it all as 'Further ALT details'. Notably*, the ALT related clinic letter from each of my two Consultants suggests raised ALT as an indicator of underlying Hep C from transfused blood was not a consideration in 1995 – 1997 when I was on DMARDs and steroids, nor a decade later in 2008 – 2009 when I was also on biological drugs. In contrast, while still on biologics in late 2009, the Consultant at my current hospital requested a Hep C blood test after just 1 raised ALT.
   (*Not to 'criticise' but to show raised ALT relevance to undiagnosed underlying Hep C.)

5. I have found key research advising specific raised ALT levels at which underlying Hep C can be reactivated in 'at risk' patients, supporting raised ALT as a marker for transfused Hep C infected blood diagnosis, which I hope validates the purpose of this 3rd statement. I apologise for the *disjointed* end result of my statement/s overall, as I provide relevant medical and research details. And if, in light of the current Inquiry it hasn't already happened, my hope is that Hep C screening, to include a targeted pre '91 transfused blood question, should become compulsory pre all biological drug use.

6. **Further ALT details** relate to/can replace FWS points listed below; while unchanged FWS points 4,6,10,11,14,15+17 can / should be read in tandem at 2.How Affected when using the FWS/3rd statement together. Section 8.Other Issues: concludes raised ALT. (3.Other Infections: Non-ALT, but necessary HC MCD corrections detailed here.) Fully covering raised ALT *was* too much paperwork, and too much for me to cope with.

2. How Affected

1. Re FWS Page 2, *point 1*. 1980s blood transfusions - *read after existing FWS point 1.*
   I was Hep C infected by blood transfusions in the 1980s, the decade which fell between Dr David Owen's stated planned policy 'with the aim of NHS self-sufficiency by 1977. . .' and an *eventual* safe haven of post 1991 Hep C screened NHS blood. Unfortunately, for so many people receiving 1980s transfusions and blood products, and for reasons I hope to learn from the Inquiry, 'the government moved away from a policy of self-sufficiency' when Dr Owen left as Minister for Health, so infected blood was allowed to continue.

2. Re FWS Page 2, *point 2*. August 1995 – May 1997 Raised ALT: *now replaced by* 2a – 2p

   a. From October 1989, a year after my 1988 transfusion I attended GRO-B GRO-B for nearly twenty years, cared for as both an in- and out-patient. As my rheumatoid arthritis (RA) was still aggressive I was regularly seen by the rheumatology team in clinic; and from 1991 I had regular FBC + LFTs (full blood count and liver function tests, normal ALT range at this hospital 7 - 55) to monitor a weekly 10mg dose of Methotrexate (MTX). But in one 21-month period between August 1995 – May 1997, 10 out of 17 LFT tests showed raised ALT and I attended 8 general RA clinic appointments during the ALT relevant dates. My doctors increased, decreased, or stopped MTX to try and help bring raised ALT under control for me, whilst during the same clinic-time helping me manage pain, recover from joint surgeries, and cope with all other aspects of my aggressive RA.

   b. In hoping to help find other undiagnosed Hep C infected blood transfused people, I will try to use just main facts of my own ALT related medical history to help me highlight how even long-term patients could be seen very regularly in clinic, receiving frequent blood tests to specifically monitor changes in the liver, yet still have undetected Hep C even decades later because the possibility of underlying Hep C from infected blood transfusions and its potential to raise ALT level results had, it seems, simply not been considered. As I've *since* learned, this was despite growing medical awareness of the 'Contaminated blood scandal' leading to all blood given by NHS being pre-screened for Hep C from September 1991, just 2 months after I started MTX and only 4 years before the 10 raised ALTs began. But perhaps this was the norm? I certainly had no suspicion of Hep C in either 1995–1997 or 2008–2009.

c. Relevant ALT comments from 8 clinics follow, but in particular key points f, I and o highlight: my initial raised ALT clinic; advice given for medical treatment of abnormal 3 x ALT to reduce MTX further; and final clinic doctor's thoughts re his full retrospective view of continuously raised ALTs leading him to consider liver biopsy.

d. 12 June 1995: Clinic Dr: noting 7 years of steroids had led to diminished bone density, suggested reducing Prednisolone by 0.5mg a month (I was very happy about this). I only mention this *in case* a disturbance re balance of drugs affected the next point.

e. 4 September 1995: GP rang '*Stop MTX, Aug raised ALT of 209*'; and wrote to my Consultant '*as a matter of urgency*'. Sincere thanks to my GP for 20 years of support.

f. 25 September 1995 clinic: Original Rheumatology Consultant, his only ALT clinic: re 1st high ALT of 209, nearly 4 x higher than 7-55 normal ALT, '*Thank you for asking me to see this girl as a matter of urgency . . . she is not an abuser of alcohol . . . as a rule I would strongly resist the temptation to stop MTX unless there is clearcut evidence that the patient is unwell with a real liver problem . . . There is a school of thought in the sense that in the absence of alcohol abuse there is no use in doing liver function tests.*' I can see three indicators that raised ALT as a consequence of Hep C from infected blood transfusion does not seem to be on the radar. If it had been, a simple Hep C test might have been arranged at this point to rule it out (although obviously I can only say this with hindsight). My Consultant does want to maintain MTX for me, advising '*push the MTX back up to 10mg*'; and regarding his 3 key points, even after just one single albeit very high 4 x ALT, he is open-minded in his consideration of hepatitis from '*abuse of alcohol*', but while he mentions the need for *'clearcut evidence of patients being unwell with liver problems'*, this doesn't appear to lead him to consider the possibility of underlying Hep C taken in the context of common practice, but pre-1991 possibly infected, RA-anaemia blood transfusions; and while he definitely does not give the last point of '*in the absence of alcohol abuse there is no use in doing LFTs*' as his own view, only that this was a way of thinking at the time, the idea itself might help towards an understanding of how some patients were not identified until years later and perhaps some not at all.

g. Crucially, for many of us who were Hep C infected by blood transfusion these raised ALTs might have been the only way for some of us at least to have been 'noticed medically' and eventually diagnosed with a condition we wouldn't necessarily have been aware of until it was well advanced. (Exactly how I was diagnosed 14 years later, immediately after 1 unexpectedly raised ALT of only 86 whilst on biologics.) At this 1st clinic, my Consultant asked how much I drank. I said I was practically 'teetotal' as alcohol wasn't part of my lifestyle; and considering it took between 21 – 29 years from my 1980s blood transfusions to a diagnosis of infected-blood Hep C, that early choice may well have contributed to my still being here today.

h. Patients did not see clinic letters at the time (and I don't remember if the Consultant used the word 'hepatitis' during clinic) so I never, until diagnosis, made a connection between raised ALTs re alcohol-hepatitis '*she is not an abuser of alcohol*', which I now see his letter shows he'd briefly considered, and raised ALTs re undiagnosed Hep C from infected blood transfusion, just one step away, which it seems he had not. But my Consultant did start by taking a broader view on my behalf, in considering that the cause of raised ALT might be something other than MTX which, with the exception of the final ALT-relevant clinic letter 'considering' a liver biopsy (30 June 1997) is almost the only time medication, namely and almost exclusively MTX, wasn't necessarily assumed to be both cause + remedy for initially intermittent then continuous raised ALTs over a nearly 2-year period (and unfortunately again in '08.)

i. Following a high 209 ALT in August 1995, during 1996 I had 5 *(out of 9)* intermittent raised ALTs: 83 January, 99 April, (next clinic was pushed back due to replacement surgery) *63 September, 64 October and very high 213 in December* and in 1997 I had 4 (out of 4) final raised ALTs *127 January, 146 February, 162 April and 65 May.* Those in italics were *continuously* raised.

j. My two highest ALTs were both nearly 4 x ALT, but I don't remember being worried at the time as, *although highlighting it now,* raised ALT would have been just one part of a bigger 'aggressive RA' picture for me and my doctors. And as a patient, it never occurred to me that regular liver monitoring tests specifically designed to safeguard my liver since starting MTX in 1991, and then more intensely over 21 months of disturbed ALTs, weren't already monitoring anything and everything to do with my liver. As it was, throughout the whole period until the possibility of biopsy was mentioned at my final ALT related clinic, no non-medicine investigations were

considered; instead MTX was stopped, increased, decreased or stopped again at least 7 times, so like my doctors I'd have had no thoughts of possibly contaminated-blood transfused Hep C, only that my raised ALTs were MTX–dosage related (then after ALT had resolved itself by September 1997, I wouldn't have given ALT another thought).

k. At 3 clinics, November 1995 - May 1996 raised ALT still intermittent: concerns were noted re future 'Transaminase' (ALT) levels; monthly LFTs were suggested; it was noted at one point that *MTX, Diclofenac or Sulphasalazine might be responsible for raised ALT;* and LFTs were confirmed as being checked. At a 5th clinic on 16 September 1996, my 2nd Consultant referred back to acknowledge awareness re 1st initial highly raised ALT of 209, '*she did have some transient disturbances in liver function last year.*'

l. 2 January 1997, letter to GP: Not relating to a clinic but response to being copied into blood results re 12 December 1996 very high ALT result 213 or 3.87 x ALT, 3 weeks earlier; the 2nd spike of this size and highest ALT practically 4 x normal. My 2nd Consultant noted, '*Liver functions are becoming abnormal again - at this stage I would be inclined to reduce her MTX to 7.5mg, then repeat the LFTs mid-Jan. If ALT remains above 3 times normal, will need to reduce her MTX further.*'

m. 6 January 1997 clinic, he noted *'we need to keep a close eye on her LFTs . . . I'll try to keep an eye on her blood results'.* Blood sheets in my file seem to show the 4 most recent sets of LFTs, and the 7 April clinic doctor noted raised ALTs of December 213 and January 146 but unable to see Febuary's ALT (162), *'thought it advisable to discontinue MTX and restart only once the liver enzymes come down.*' Sorry to note I'd forgotten my MTX card (and at 5th clinic) but I couldn't have supplied the *most* recent result either time, as it would only have been written-up at my *next* blood test, after that clinic. Even the doctor with all the results at the next and final clinic didn't think 'Hep C!'.

n. Extracts from individual dates in my GP's notes show frequent clinic adjustments to MTX dosage were still not controlling or even directly affecting raised ALT levels, yet no clinic doctor had mentioned a Hep C test; I don't even know if the Hep C test was used at the hospital in those days . . . '*Raised ALT 209, stop MTX; ALT 83, going up again; ALT 99;* (raised ALTs of 63, 64 not noted); *ALT 213 abnormal, doctor will discuss, reduce MTX if ALT remains more than 3 x normal; ALT 127, ALT has dropped to half of what it was without altering MTX dose; ALT 146, in view*

of persistent LFT changes and nausea had already reduced MTX; ALT 162, even higher despite reducing MTX, reduce dose again' (hospital clinic notes also show *Withhold MTX; MTX 5mg;* while final raised ALT of 65 not noted by GP)'. Were my 10 raised ALT results just not high enough? As a patient I don't know how high ALT must be, or how many high ALTs cause enough concern for a doctor to consider the need for a Hep C blood test? But my next clinic doctor, though not a Consultant, did finally 'consider' a non-medication alternative via a liver biopsy investigation; he also provides an answer to my 'how high' ALT question in his following clinic letter.

o. 30 June 1997: Clinic Dr: Crucially, this doctor highlights 7 results spanning 9 months of unceasingly abnormal ALTs. '*I note she has had persistently elevated LFTs since Sep last year. Currently they* (LFTs, with final raised ALT 'only' 65) *are less than 2 times the upper limit of normal and I would therefore continue with her MTX. However, should her LFTs remain abnormal over the next 2 or 3 months we would strongly have to consider a liver biopsy before continuing with MTX.'* Many thanks to this vigilant doctor. Normal ALT at the hospital was given as 7–55, his reasoning suggests 2 x ALT, so an ALT of 110 taken in context, as a benchmark for concern and action. This would include 5 of my 10 highest ALTs at x 3.80, 3.87, 2.31, 2.65 and 2.95, the last 4 within the last 6 months. I very much appreciate this doctor's recognition of long-standing unresolved ALT issues and need for a different method of investigation to identify the cause, yet at this point my ALT results did return to sustained normal at 30, 24 and 18 over 3 months (and for several years), so unfortunately for me there was no biopsy, things just moved on without me realising the significance of a missed opportunity to identify underlying Hep C. My ALTs returned to normal for no MTX-related reason, just as they had fluctuated randomly over 21 months despite adjustments to control them with lower or higher dose MTX. Sadly, even whilst his full retrospective view of the run of most of my highest ALTs concerned him enough for a liver biopsy, a Hep C test was not considered if only to narrow down what had been aggravating liver function results over a long period.

p. However, the reason I've been boringly breaking down raised ALT values above for example ALT of 213 is 3.87 x ALT, is to highlight the positive link between the final clinic doctor's 2 x ALT reasoning to more recently researched understanding of the ALT level at which underlying Hep C can be reactivated, as fortunately I recently found key research clarifying RA 'risks' at a specific raised ALT level of 2–3 x ALT. My final clinic doctor had referred to a sustained 2 x ALT as the decisive point at which he would, *'strongly consider a liver biopsy'*; whilst my 2nd Consultant referred

to above 3 x ALT as his point for concern and medication-related action, '*If ALT remains above 3 x normal, we will need to reduce her MTX further.*' And now years later, both these levels taken together are supported by key research identifying a range of 2–3 x ALT to benchmark when Hep C screening should be considered for *'patients who had been exposed to risks' prior to biological drug use'.* In Omer Karadag's 2015 research report he says: '*Viral hepatitis screening guideline before biological drug use in rheumatic patients: Biological drugs (TNFs, RTX etc) may cause HCV reactivation. Therefore, screening of patients before biological treatment and application of prophylactic treatment are recommended when necessary . . . reactivation risk is higher when more than one immunosuppressive drug is used.*' (I was, and still am, on both steroids and DMARDS.) '*If the serum ALT level increases 2 – 3 x more than baseline* (mine was at times nearly 4 x ALT), *and the level of HCV RNA increases . . . HCV reactivation occurs. HCV screening should not be conducted for all patients, but only for those specified . . . Patients exposed to risk . . . who underwent blood to blood product transfusion before 1994*' (so not '91?) *'and those who underwent surgery'*. I met all 4 reactivation risks. (Other types of hepatitis are also tabled). This 'biological' research states that it also applies to DMARDs (eg MTX) used with corticosteroids (eg Prednisolone), my long-time medications, reinforcing the fact that, in the '90s, my final clinic doctor was absolutely right to feel concern at raised ALTs even as low as 2 x ALT. Again, thank you to this doctor.

3. FWS Page 3 *point 3* 2008-2009 Raised ALT/biologicals: *read after existing FWS pt 3.* I can now state that my '*Protocol for starting anti-TNF therapy for adults with RA (2004)*' did confirm, as I'd thought, that there was no required pre-screening for Hep C. However, under the question, '*Are there any other contraindications to starting anti-TNF therapy?*', 5 given examples did include a blanket term of '*uncontrolled infection*', but this would have meant nothing to me at the time, as underlying Hep C can't be 'felt' until later stages, so at that point I had no idea I'd had Hep C for at least 20 years. However, what Protocol did stress very strongly was that the Infliximab/MTX combination should be used together whenever possible for the most effective outcome. *'Protocol: 'Is the patient taking Remicade? Remicade (Infliximab anti-TNF) is licensed in this country to be given only with MTX. If the patient is not taking MTX and yet can tolerate it, this should be commenced immediately'.* I was already on MTX.

4. Re FWS Page 4, *point 4*: Only 2 joint surgeries post '08, originated by accident not RA.

5. Re FWS Page 4, *point 5*. re September – October 2008, 3 raised ALTs: further details 5a – 5d

   a. 24 September 2008: My 6th infusion, due in September, was cancelled and MTX withheld due to 3 raised ALTs of 120, 119 and 126 (or 2.18, 2.16 and 2.29 x ALT) on 19, 24 September and 1 October. Yet, 10+ years after 1995–1997 string of abnormal ALT, 3 raised ALTs out of the blue while on powerful biologics unfortunately still did not cause my (2nd, now only) Consultant to consider historic Hep C infected transfused blood awareness.

   b. 3 October 2008: Consultant to me: '*some slight disturbances with LFT, we need to keep an eye on this with more frequent tests until it improves. It is possible we can use Infliximab alone without MTX or perhaps consider another form of anti-TNF.*'

   c. 18 October 2008: Clinical Nurse Specialist (CNS) letter to my Consultant: '*Her ALT is now back to normal . . . MTX was stopped from 24/9/08 and last Infliximab infusion was 30/07/08 . . . kindly advise whether we restart MTX at a lower dose and continue Infliximab whilst monitoring the bloods or a different regime?*'

   d. 28 October 2008: GP notes: '*She was feeling unwell last week since off anti-TNF and MTX since September due to LFTs. For U/S scan to exclude GS as some upper abdo pain; refer U/S investigation Liver/Gallbladder.*' (Confirms FWS GP scan request.)

6. Re FWS Page 5, *point 7*. November 2008 – January 2009, RA without MTX: further details 7a – 7h

   a. 3 November 2008: Rang my surgery re increased burning pain in joints, needing something stronger; I rang the hospital advice line, leading CNS to email Consultant: '*She contacted us wishing to restart Infliximab; I will wait to hear from you whether treatment is to be recommenced.*' And '*I understand from her that her GP is arranging a USS of her liver.*' Consultant: '*Let's go ahead (with infusion) on 12th*'.

   b. 5 November 2008: Consultant to CNS: '*most recent LFTs from 28 Oct show improvement with the ALT well within normal limits at 29 (normal 7 – 55) and ALP 140 (normal 35–104). I think for the moment we should continue to withhold MTX'.*

c. 12 November 2008: 6th infusion given, 7 weeks without MTX, 4 weeks of normal ALT.

d. 17 November 2008: Cons to GP: Re 3 raised ALTs, all 2+ x ALT, over 3 weeks. '*I would be grateful if LFTs could continue to be monitored 2 weekly just in case the disturbance was due to infliximab although I think it more likely related to MTX. We may well try to introduce a small dose of MTX in the near future in order to reduce the risk of development of chimeric antibodies that can be associated with Infliximab infusion reactions. This seems more common with Infliximab than with other anti-TNF agents, which is why MTX is often used in combination. However, some patients are maintained on Infliximab alone and this would remain an option.*' Obviously, the Consultant is being cautious re MTX on my behalf, but never did *'introduce a small dose of MTX in the near future'* to reduce an acknowledged risk; and as he only *'thought it more likely'* to be MTX, he could perhaps have considered a Hep C test to clarify next steps, in case raised ALT was also some sort of '*Infliximab infusion reaction*'. Perhaps I wasn't tested because the introduction of 1991 Hep C screened NHS blood was historically so far away from 3 raised ALTs in 2008, it just simply didn't come to mind that a very long-term patient could have been infected all those years ago, pre 1991, yet never been Hep C identified? Could this have happened / still be happening to others with Hep C? As it was, the '*Infliximab alone without MTX*' option was maintained but with MTX withheld since 24 September my decline in health continued.

e. 18 November 2008: GP: '*US Upper abdomen normal. No action*'. Many thanks to my GP for trying to find a cause. (Although following a positive Hep C test at my current hospital in December 2009 just a year later, my January 2010 'non-drinker' liver biopsy reported '2,1,1' abnormalities, and March 2010 my first Hep C viral load reading was 8,693,848).

f. 17 December 2008 / letter 9 January, Extra-clinic Dr: '*I reviewed her as an extra patient, she has been struggling with increased activity of her RA. . she restarted infliximab infusions 5 weeks ago. .no longer taking MTX. . .also stopped her Hydroxychloroquine in Sep 2008 as until then her rheumatoid had been very well controlled since introduction of anti-TNF. We would consider reintroducing HCQ in the future*'. I'd forgotten I'd had support of 2 DMARDs on anti-TNF; so now struggling without support of either.

g. 7 January 2009: 7th anti-TNF infusion given,15 weeks without MTX (or HCQ), RA flaring.

h. 26 February 2009: GP: '*Still reacts very well to Anti TNF but* (without MTX) *it wears off after 5 weeks* (of an 8-week cycle) *and she has 3 awful weeks to wait till next time'*, re my increasingly painful lead-ups to each of the Nov, Jan and Mar infusions.

7. Re FWS Page 5, *point 8*. March 2009 Infusion not allowed; UTI: Further details 8a– 8e

a. 4 March 2009: Infusion not allowed due to UTI. 5 March 2009: Anti-TNF Nurse to Consultant re non-infusion, '*due to a raised WBC and UTI symptoms we delayed it. She was wondering if she can go back on her MTX as she feels the Infliximab is only working 5 weeks without it. Her bloods have returned to normal.*' As ALTs were normal since 15 October, even allowing for a safe period of stabilisation, I don't quite understand why such a long absence of MTX was necessary especially as Protocol had strongly emphasised how important it was for '*best outcome'* that MTX be used alongside anti-TNF whenever possible, and I was having painful RA problems without it. My Consultant himself had noted as early as 5 November 2008, *'for the moment . . . we should continue to withhold MTX'*, and 17 November 2008, '*we may try to introduce a small dose of MTX … in the near future'*. I'd have liked to have re-tried MTX (or HCQ).

b. NB 19 June 2008: clinic letter of the previous year, when I was doing so well RA-wise, Anti-TNF nurse: '*Since commencing Infliximab she has had 2 UTIs which require antibiotics. . .she has experienced UTIs in the past. . .but it is well-known side-effect of Infliximab so at this stage we are unsure of the cause.*' See FWS *point 9* + below.

c. 9/10 March 2009: 6 days later, night-time emergency hospitalisation at my local hospital

d. 11 March 2009: GP notes Externally Entered: Emergency Dept, GRO-B Hosp

e. 11 March 2009: 2nd Consultant: '*I have had a letter from the anti-TNF team mentioning Infliximab infusion deferred because of possible UTI. She wondered whether she could restart MTX as Infliximab is not giving her full cover. I think this*

would be reasonable especially as recent blood tests have shown normal liver function with ALT well within normal range.' (… as it had been for several months).

8. Re FWS Page 5, *point 9*. Mar-May septic arthritis/RA flare, *read with existing point 9.* Consultant, '*UTI crossing into bloodstream could have caused sepsis of both hips.*' 11 March Urgent Reports, 7 of 9 abnormal results include: WBC 24.9 (4.5-11) 'Neutrophil Leukocytosis'; Platelets 616 (150-450) 'Thrombocytosis' (underlying infection); Neut 23.7 (1.8-7.7). 13 March: hb fell from 9.8 2 days earlier to 8.1 (12-16); ESR 108 (<20), CRP 142.3 (<6). 18 May clinic: Consultant, *'assumed to be septic arthritis* linked to UTI. *Furthermore she had a fever and a greatly raised WBC. During this time, she stopped MTX and Infliximab not surprisingly her RA has flared and she had to be put on 30mg Prednisolone daily to control her symptoms . . . clearly needs to go back on anti-TNF . . . will arrange for admission as soon as possible for an Infliximab infusion'*. (FWS Page 6 *point 11*. NB: '7, *not 5,* successful months of anti-TNF/MTX combined')

9. Re FWS Page 7, *point 13*. Hep C diagnosis: Add to end of 2nd sentence, . . . *'following a raised ALT of just 86 considered in context of my RA history. (10 further fluctuating raised ALTs of between 63–149 return to normal on commencing Hep C therapy.)'*

10. Re FWS Page 8 *point 16*: 2nd sentence should be final sentence *point 15* in conclusion of an important point made there. 'On 8 February 2010, Dr **GRO-B** advises the following '*she can restart MTX now we know Hepatitis C is responsible for the raised ALT markers.'* And 1st sentence, 'I appreciate everything my Consultant . . . 48-week Hepatitis C treatment', chronologically misplaced, should become penultimate sentence *point 17*. An empty *point 16* should now state the essence of the crucial point I was panicking to make re RTX and viral load in my Supplemental Statement (SS), having accidentally been unable to include it in my FWS: Significantly, on 1 March 2010 following on from January's still-raised ALT of 126, my initial Hep C viral load was 8,693,848 but had already fallen away by half to 4,068,845 on 29 July, a month before my 48-week course of Hep C therapy even began. I believe this gives a good picture of what my body was going through as a direct result of receiving biological drugs particularly RTX in 3 transfusions building from July to September 2009, feeling so well from the end of Octobeer yet having raised ALT of 86 late Dec, *all without my first having been Hep C tested pre anti-TNF or RTX* . . . . . And I believe my March post-RTX viral load high, followed by July's dramatic viral load fall, validates my FWS statement as it strongly supports Consultant opinion given in the document evidenced

letter 9 February 2010 that biological drugs can aggravate and re- activate underlying Hep C, therefore that he's right in saying that all patients should as a matter of routine be pre-tested for Hep C prior to receiving biological drugs. This powerful agitation is one of the most important points I'm raising, together with 30 years of underlying Hep C cumulatively injuring my brain *(HC MCD research)*, perhaps key to what happened in terms of permanent MRI damage re decision making / memory, ability to *quickly grasp, hold onto and use information* *in real time, real life situations*; and whilst writing this statement, 'am I understanding this information correctly when I can't always *re-find!* or carry-over in my head, medical strands or facts I've *just* read?' *(Read final ALT point 17 FWS; raised ALT conclusion at* *8.Other Issues* *page 13 below)*

3.Other Infections

(FWS corrections re research of HC MCD brain condition, *to read later with FWS pages 9-11.)*

1. Page 9, *point 2*, line 6: Exhibit cited here is misplaced belonging to point 1, line 4 after *'some form of dementia'*; at same line 6, reinstate key diagnostic sentence deleted by the exhibit, *'in the last 5 years. He explained, 'This is not the clinical picture of degenerative dementia which would likely involve grey matter in a different area of the brain.'* (This sentence was in my original Consultant supported letter to Inquiry.)

2. Page 10, *point 4*: HC MCD research: Maurier '10, Forgot to give title 'Hepatitis C virus causes brain inflammation leading to neuron injury', *'We saw the virus in brain of a deceased patient who had hep C. Normally very difficult for any virus to pass blood brain barrier'* cumulative attack *'triggering inflammatory changes that ultimately result in damage to neurons.'* Irwin/Terrault 2008, begins 'Cognitive abnormalities are more common in people with comorbidities', my RA/Osteoporosis? Solinas 2015, begin, *'irrespective of the grading of liver fibrosis. . HVC brain dysfunction may be associated with white matter neuronal loss. . .unclear to what extent* HCV triggers irreversible neurodegenerative brain damage. . . evaluations should be associated with. . MRI . .'

4.Consent

5.Impact

6.Treatment/Care Support

7.Financial Assistance

1. I have no amendments or any further evidence to add to any of the above.

8.Other Issues

Finally, to re-state the aim of this 3rd statement

1. My hope is that pro-active pre-screening for Hep C before biologics, to specifically include historically infected blood transfused patients, will become routine. A Janruary 2012 NRAS (National Rheumatoid Arthritis Society) article '*Biologics: The story so far*' asks '*Are there any medical reasons why I can't take these drugs? You are screened before starting treatment to exclude an increased risk of side effects (TB, MS etc) . . . You will have a chest x-ray and also blood tests to check blood count, liver function tests* and in some cases to exclude hepatitis B and C. All of these are routine for your protection and safety.'* So, if Hep C tests are among those considered 'routine for our protection' prior to biologics, but only in 'some cases', what's the criteria for who is considered in those cases and does pre-screening include a question specifically aimed at finding pre 1991 infected blood transfusion patients not yet Hep C tested? I finally found *NICE December 2012: Guidelines on raising awareness of and testing for hep B and C infection* but couldn't face reading it! (*Hep C may be quiet/not cause raised ALTs for long periods.)

2. As anti-TNF 'Protocol (2004)' used in hospitals in 2008 did not mention Hep C screening, I'm concerned for other patients in any hospital who might have gone on to show raised *ALTs* on biological treatment; were any others eventually Hep C tested and identified as infected by historic blood transfusion? In 2008, my current hospital routinely screened patients for Hep C pre anti-TNF, so they would have 'found' me before I received any biological treatment if I'd been their patient at that time; yet my previous hospital, therefore I'm sure there would be other hospitals, did not pre-screen for Hep C in 2008, but they were obviously not in the wrong at all because clearly it was not a Protocol consideration at the time. So, was pre-screening disparity between different hospitals generally just a case of best practice, a matter of luck/or not for the patient? Is it still?

3. Similarly, my current hospital has only 42, not 55, as upper range ALT limit. I now understand that some 'normal blood ranges' can vary depending on which lab supplies which hospitals (so must be just accepted practice) but it does mean that if I'd been at my current hospital in the 1990s, my 2 highest '95 – '97 ALTs would have been 5 x ALT which again might possibly have led to a Hep C test years earlier and, with treatment, would have left me in a better position to benefit from anti-TNF later. (I wonder . . . what range for upper ALT level did Omer Karadag base the 2015 research on?!)

4. If nothing's changed and patients are still dependent on 'best practice' of local hospitals rather than on a universal standard of Hep C testing pre anti-TNF, could NICE level-up the situation to standardise Protocol by considering Omar Karadag's 2015 research advice to actively seek those affected? 'Viral hepatitis screening guideline before biological drug use in rheumatic patients' recommends that for Hep C rheumatic at risk patients *'An Anti-HCV test must be performed in individuals in the risk group for HCV infection . . . but anti-HCV screening must be conducted for all patients to take RTX.'* Purely by chance, I had neither. Obviously, non-RA people also had historic blood transfusions for various reasons (accident, maternity, op) and may have come into the system later in life to use biological drugs for other illnesses, so every pre 1991 possibly Hep C-infected blood transfused person about to start on biologics, from any medical setting, deserves a safety net of targeted and potentially life changing pre-screening.

5. Just before I signed off at above, I was stunned to learn from an Inquiry hearing that on 11 Jan 1995 DOH announced a 'Lookback' exercise to find people with Hep C through infected blood transfusion. I couldn't believe the date! Lookback lasted from 1995-1998 easily covering my 1995-1997 string of ALTs. Realising it in hindsight stopped me in my tracks; have I got it all wrong? Clearly my memory's unreliable but *I just can't remember* associating my hospital with a Lookback . . . I felt too stressed to write more but had no choice. It's taken at least 3 months to write/re-write extra points 5-6 in trying to join the dots, as *I need facts to base my memories of events on.* But the medical records I copy-typed contained no letter sent to me re Hep C blood infected transfusions, no reflections of Lookback awareness (not even evidence of a *negative(!)* hep C test) to comment on. Otherwise, at my 1st ALT relevant clinic 8 months after Lookback began, on seeing my original RA Consultant *'as a matter of urgency'* re my GP's initial *'raised ALT of 209'* concern, his first Lookback question could have been, 'Did you have a pre 1991 RA-related blood transfusion?', in place of his non-Lookback aware comment (not his view) that *'in the absence of alcohol abuse there is no use doing liver function tests*'. But equally, I could have done more to help myself IF I'd known about Lookback, as there would be no reason for me not to volunteer my own transfusion details, so I'm sure I didn't know either. If there were Hep C forms in the waiting room prompting patients to discuss blood transfusions in clinic, I'd have listed my 3 stated transfusions, plus (almost certainly) re 1984 replacement, and definitely re my 1988 childbirth, records now irretrievable. Earlier, in June 1991, my 1st Consultant noted RA *'has deteriorated very considerably'* admitting me urgently. Due

to past medication side effects (Gold rash, duodenal bleeds) I was very anxious about even stronger MTX, but on the ward *'Following lengthy discussion with'* Consultant and 2nd doctor *'she agreed to start MTX.'* I felt so reassured when it was explained that regular targeted full *blood* and *liver function* tests would monitor and highlight 'any' liver concerns. I'm sure (although I do now know about 1991 pre-screening of all NHS blood 2 or 3 months later) we weren't talking about Hep C at the time, but *sometime* post 'ALT' clinics when I became aware of media Hep C interest, I'd have felt fortunate to be regularly tested and *naively* have believed that if a Hep C-specific marker(!) ever appeared in my blood it would somehow flag up an urgent alert in blood test results . . . yet that must be exactly what already had happened in 1995-1997 although we didn't realise it. The correct marker for hepatitis had flagged up an alert although not specifically shouting 'Hep C infected blood!' alone, but warning 'not so specific / open to interpretation raised ALT' – within a few clinics permanently linked to MTX dosage, except for an eventual short-lived consideration of liver biopsy in 1997. *And I've just realised, perhaps this final doctor was thinking of a Hep C diagnosis via biopsy* but didn't specifically name it in his clinic letter. If only a Hep C blood test had been taken. If anything, not needing a biopsy could only have reinforced my trust in my blood being Hep C-clear, a belief lasting until January 2010 when after an ALT of 86 my current Consultant asked if I'd ever had a Hep C test and I answered 'No'. . . as I'd always have believed, *correctly* it seems, my Consultant's explanation unless told otherwise by another doctor. IF I'd ever been asked, I'd have said, and did say to my own family, 'I've been having regular liver blood tests for years, I'm fully covered'. But I was *wrong* too, as I wouldn't have known liver tests alerts could be misunderstood, and as a patient I didn't think for myself, so just didn't realise the Hep C message also applied to me. I've tried to explain my ALT history in a balanced way, but I accept the hospital may want to point out something I've clearly missed.

6. I hope this statement is a fair account to help find some of those still unidentified by highlighting raised ALTs as a possible marker for Hep C infected blood diagnosis, and for Hep C screening for all biological drugs to include a targeted pre 1991 transfused blood question. For whatever reason, I wasn't identified during '90s Lookback, or 2008 Protocol-supported pre-biological routine screening, or re 3 x raised ALTs later in 2008 whilst I was on biological drugs, but 21 - 29 years after my 1980's blood transfusions I was eventually identified through a single raised ALT of 86 in context of my RA history.

Statement of Truth

I believe that the facts stated in this witness statement are true.

Signed... GRO-B .....

Dated... 6th April 2021