REVIEW OF THE SUPPORT AVAILABLE TO INDIVIDUALS INFECTED WITH HEPATITIS C AND/OR HIV BY NHS-SUPPLIED BLOOD TRANSFUSIONS OR BLOOD PRODUCTS AND THEIR DEPENDANTS

Contents

Section 1 – Introduction to the review and scope

1.1 On 14 October 2010, the Government announced that it would review the support available to those who had been infected with hepatitis C and/or HIV by NHS-supplied blood transfusions or blood products.

1.2 The scope of the review was defined by the following terms of reference:

1. To review the following aspects of provision and support for those affected with HIV and/or hepatitis C via NHS-supplied contaminated blood and blood products:

a. to review the level of *ex-gratia* payments made to those infected with hepatitis C, including:
   - the consideration of financial support for their dependants;
   - the eligibility date for entry to the current scheme;
   - comparison with the *ex-gratia* payments made in the UK to those infected with HIV (and their dependants);

b. to review the mechanisms by which the *ex-gratia* payments for HIV and hepatitis C are made;

c. to consider the provision of insurance for those infected, (having regard to similar access available to other groups) including:
   - life assurance cover;
   - mortgage protection cover;
   - travel insurance;

d. to consider the issue of prescription charging for those infected;

e. to review the provision of, and access to, nursing and other care services in the community for those infected.

2. To develop options arising from the above, and make recommendations to Ministers for their consideration by the end of the year.

1.3 A copy of the Written Ministerial Statement announcing the review is attached at **Annex 1**.

1.4 Issues that were raised during the House of Commons backbench debate on contaminated blood on 14 October 2010 have also been considered. These are:

- *ex-gratia* payments for individuals infected with HIV and their dependants in light of the options considered for individuals infected with hepatitis C;
- exemption from the Department for Work and Pensions (DWP) Work Capability Assessments; and
- access to dentistry for individuals infected with hepatitis C.

1.5 This review examines these issues for all individuals (people with haemophilia and others) who were infected with HIV and/or hepatitis C by NHS-supplied blood transfusions or blood products, regardless of whether these were sourced from the UK or other countries.

The Archer review

1.6 In 2007, Lord Archer of Sandwell set up an independent inquiry into infections arising from NHS-supplied blood and blood products, which reported on 23 February 2009. In his report he did not seek to apportion blame but made a number of recommendations about financial and other support, focusing on people with haemophilia. A full list of the Archer recommendations is at www.archercbbp.com/report.php.

1.7 Some of Lord Archer's recommendations were already accepted and in place before his inquiry began. These were:

- Free access to GP services, physiotherapy, home nursing and support services;
- Testing people with haemophilia and blood donations for specified infectious agents;
- *Ex-gratia* payments to those infected with both HIV and hepatitis C (although not at the levels recommended by Lord Archer). *Ex-gratia* payments disregarded for the purposes of calculating income tax and eligibility for calculating other state

benefits. Payments made if infection is confirmed, and there is an appeals mechanism.

1.8 The previous Government's response to Lord Archer's report was published on 20 May 2009. The following changes were implemented as a result:

- Introduction of flat-rate payments of £12,800/year for HIV-infected individuals from that date (previously the average charitable payment to infected individuals and their dependants was around £6,400);
- Increased discretionary payments to both those infected and their dependants, through the charitable trusts;
- A review of the Skipton Fund in 2014 (in April 2010 a government announcement brought it forward to later in 2010);
- A £100,000 annual grant to the Haemophilia Society from 2010/11 to 2014/15;
- Twice-yearly meetings between the Department of Health and the Haemophilia Alliance (as opposed to the statutory Haemophilia Committee proposed by Lord Archer);
- A look-back exercise to identify individuals with bleeding disorders who might have been infected with hepatitis C but remain unaware of the fact. This is currently underway.

Section 2 – Background and current financial support

Historical context

2.1 Before heat treatment of blood products was introduced in 1985, and a test for hepatitis C was developed and introduced in 1991, 4,675 people with haemophilia in the UK were infected with hepatitis C by NHS-supplied blood products during the 1970s and 1980s. It is estimated that 2,807 of these individuals are alive today. Published epidemiological estimates suggest that up to 28,043 other individuals might have been similarly infected with hepatitis C by whole blood transfusions in the UK. See **Annex 2** for details of estimates of numbers of infected individuals.

2.2 Over roughly the same period, approximately 1,200 people with haemophilia and 100 other individuals were infected with HIV by NHS-supplied blood products or blood transfusions in the UK before the introduction of heat treatment of blood products, and the development and introduction of a test for HIV, in 1985. It is estimated that 400 of these individuals are alive today.

2.3 Some of these individuals were co-infected with both hepatitis C and HIV, and there are around 361 co-infected individuals alive today.

2.4 In recognition of the special circumstances of these infections, a financial support package was developed and set up in the late 1980s and early 1990s for individuals infected with hepatitis C and/or HIV by NHS-supplied blood transfusions and blood products. Payments are *ex-gratia* payments which there is no liability to make. Three *ex-gratia* payment schemes were set up, and they have paid out £206 million since their establishment. *Ex-gratia* payments are disregarded for the purposes of calculating income tax and eligibility for calculating other state benefits. Payments are made if infection is established.

Ex-gratia payment schemes for HIV infected individuals and their dependants

2.5 The Macfarlane Trust was established in November 1987 as a charitable trust to make payments to people with haemophilia who had been infected with HIV from NHS-supplied blood products. It was

initially funded by the Government with £10 million, and in 1989 a further grant of £19 million was made.

2.6 In 1988, approximately 1,000[1] people with haemophilia who had been infected with HIV by blood transfusions and blood products brought litigation against the Government. On their own legal advice, the litigants settled the case out of court in 1991. A further £42 million was granted to the Macfarlane Trust in 1991 following this litigation. It is now funded annually.

2.7 The Eileen Trust was established in 1993 as a charitable trust to make payments to other individuals who had contracted HIV from NHS-supplied blood transfusions and blood products. It was initially funded by the Government with £500,000, received a further £500,000 in 2001, and is now funded annually.

2.8 **Annex 3** provides additional information on the Macfarlane and Eileen Trusts.

2.9 HIV infected individuals received lump a sum of £20,000 each in 1990, and an additional lump sum in 1992 of up to a maximum of £80,500 (for a married individual with children). Currently, HIV-infected individuals receive a flat-rate annual payment of £12,800 via MFET Ltd, a separate payment vehicle established in 2009 to make flat-rate payments to registrants of the Macfarlane and Eileen Trusts. Some also receive additional discretionary payments for themselves and their dependants through the Macfarlane and Eileen Trusts. These discretionary payments are decided by the Trustees, within the overall financial resources available to the Trusts.

2.10 Dependants of those with HIV may also receive discretionary payments from the Macfarlane and Eileen Trusts following bereavement. The Trustees' current approach is to top up the dependants' annual household income to a total of £15,000. Individuals with an income in excess of £15,000 per annum do not receive any discretionary payments.

[1] Government documents suggest various numbers of litigants, possibly because additional litigants joined during the course of the case.

Ex-gratia payments to hepatitis C infected individuals

2.11 The Skipton Fund was announced on 29 August 2003 to make payments to individuals infected by hepatitis C by NHS-supplied blood transfusions and blood products. It is a company limited by guarantee, acting as an agent of the Department of Health and the Devolved Administrations. **Annex 3** provides additional information on the Skipton Fund.

2.12 Individuals who develop chronic hepatitis C receive a lump sum payment of £20,000, and those who go on to develop severe liver disease (cirrhosis, decompensated cirrhosis and hepatocellular carcinoma) receive an additional lump sum of £25,000. The individual's clinician completes the part of the application form relating to the individual's hepatitis C infection. No recurrent payments are made.

2.13 The Skipton Fund does not provide financial support for dependants. Only those infected and still alive on 29 August 2003 are eligible for payments. Where the claimant died after 28 August 2003, but before payment was received, the payment was made into their estate. The Skipton Fund has an appeals panel that is independent of the Skipton Fund Ltd.

Payments to those individuals co-infected with hepatitis C and HIV

2.14 Co-infected individuals are eligible for payments via the relevant separate schemes for both their HIV and their hepatitis C infections.

Section 3 – The review process

3.1 The review was conducted by the Department of Health, supported by input from relevant external experts, including the Chairs of the Macfarlane and Eileen Trusts and the Skipton Fund.

3.2 Scientific and clinical advice on hepatitis C and HIV was obtained from a joint working group of the Advisory Group on Hepatitis (AGH), the Expert Advisory Group on AIDS (EAGA), the UK Haemophilia Centre Doctors Organisation (UKHCDO), the Hepatitis C Trust and the Health Protection Agency (HPA). This scientific and clinical review of the evidence base is at **Annex 4**.

3.3 Advice on insurance was obtained from the Association of British Insurers (ABI) and Hannover Life Assurance (UK) Ltd.

3.4 There was also liaison with the following Government Departments: HM Treasury (HMT); the Department for Work and Pensions (DWP); HM Revenue and Customs (HMRC), and with the Blood and Tissue Policy Unit of the Department of Health and Children in the Republic of Ireland.

3.5 Representatives of the affected community and members of Parliament were engaged throughout the process. Anne Milton MP, the Under Secretary of State for Public Health, met representatives of the main campaign groups (the Haemophilia Society, Tainted Blood, Contaminated Blood Campaign Coalition, Manor House Group and the Hepatitis C Trust), as well as the Chairs of the current *ex-gratia* payment schemes, and others, at meetings in July and November 2010. Written submissions and correspondence were also received from these groups and affected individuals (see **Annex 5** for campaigners' requests in relation to support packages).

3.6 The conduct of the review was based on the scope set out in the Terms of Reference, and the following principles:

- to reduce the current anomalies between the HIV and hepatitis C payment schemes and avoid the creation of new anomalies;
- to ensure the review is evidence based, where evidence is available;

- that payments are made in recognition of the special circumstances of these individuals as a result of their infection, and their financial need.

Section 4 – The expert scientific review

4.1 An expert scientific review was undertaken of the evidence base on the spectrum and impact of disease associated with hepatitis C infection. This evidence base informed this review and its recommendations.

4.2 Expert scientific and clinical advice was sought from a joint working group of EAGA, AGH, UKHCDO, the Hepatitis C Trust and the HPA. The following paragraphs are a summary of the review report, which is attached at **Annex 4**.

4.3 Most individuals experience few, if any, clinical symptoms during the acute phase of hepatitis C infection. Some individuals clear the infection naturally in the acute phase. However, the majority of individuals will progress to chronic infection.

4.4 Chronic hepatitis C infection is associated with a range of non-specific symptoms and a demonstrable loss in quality of life. In addition, chronic infection has been associated with a range of extra-hepatic symptoms, including neurocognitive effects that impact on daily life, but many of these are difficult to attribute to hepatitis C infection in an individual.

4.5 Drug therapy is able to achieve effective viral clearance in the majority of individuals treated before cirrhosis has developed. However, therapy itself is associated with a range of side-effects, which may be significant. Following successful treatment, the prognosis for disease progression and quality of life largely improves.

4.6 A proportion of individuals with chronic infection will progress to cirrhosis, decompensated cirrhosis, or hepatocellular carcinoma. This substantially reduces quality of life, which is liable to deteriorate over time, and has a substantial impact on life expectancy. Even if a sustained virological response can be achieved in cirrhotic individuals, liver fibrosis is not completely reversed and the risk of decompensation or of developing hepatocellular cancer remains. Some individuals will be eligible for liver transplantation, but this in itself involves considerable morbidity and re-infection occurs in nearly all individuals. Those who develop life threatening hepatitis C-related tumours, specifically B-cell non-Hodgkin's lymphoma (B-cell NHL) will

experience ongoing hardship of a similar level to those who develop cirrhosis and severe liver disease.

4.7 Co-infection with HIV can increase the rate of progression to chronic hepatitis C infection and cirrhosis.

4.8 The following conclusions have been drawn from the evidence base presented in the expert scientific review report:

i) The lack of morbidity associated with acute hepatitis C infection supports the current position that individuals who experience acute infection, and do not progress to chronic infection, should not receive financial support;

ii) The evidence does not support a strong case for making a change to the current Skipton Fund stage 1 payment for all individuals with chronic hepatitis C infection, as this payment is designed to take account of the range of symptoms caused by hepatitis C infection, as well as the side-effects of treatment. Nevertheless, some individuals may experience financial hardship in this phase of the disease, particularly if they are unable to work during periods of treatment;

iii) The development of serious liver disease or B-cell NHL associated with chronic hepatitis C infection will substantially reduce quality of life, and have an impact on life expectancy. Therefore, there is a strong case for improving the current provision for payments to this group.

Section 5 – Payments for hepatitis C infection

5.1 There are some clear discrepancies between the provision of *ex-gratia* payments to those infected with hepatitis C and those infected with HIV through NHS-supplied blood transfusions and blood products. There are no annual payments for those infected with hepatitis C and there are no discretionary payments to those infected with hepatitis C or their dependants. Further, payments were not made in respect of individuals infected with hepatitis C who died before 29 August 2003.

5.2 The case for greater similarity between *ex-gratia* payments for HIV and hepatitis C infection is based on the arguments that the impact on quality of life of living with chronic hepatitis C is at least as great as that of living with HIV. In addition, those who are living with chronic hepatitis C are now more likely to die prematurely if they develop severe liver disease. Dependants of an infected individual can experience financial hardship, irrespective of whether the individual was infected with HIV or hepatitis C, and whether the infected individual is still alive. There is thus a case that those infected with hepatitis C and their dependants should have access to a financial support scheme that is broadly comparable with that available to those affected by HIV infection.

5.3 A wide range of views has been received from campaigners on the level of payments that this patient group should receive (see **Annex 5**).

<u>Options for payments to individuals infected with hepatitis C</u>

5.4 In this review, options were identified for payments to those living individuals infected with hepatitis C. These options are not mutually exclusive and are summarised in Table 1.

5.5 It is not possible to quantify precisely the financial implications of the options considered because there are too many unknowns, which include: numbers of surviving infectees, past and future mortality rates, disease progression and severity trajectories, numbers of potential claimants who may not have come forward and numbers of dependants, availability of evidence to support future claims, and the outcomes of discretionary decisions of Trustees. Hence all cost estimates, in all the Tables in this Review, are subject to unknown levels of inaccuracy.

5.6 The expert scientific review presented evidence that indicated that the current Skipton Fund stage 1 payment level is appropriate for those with chronic hepatitis C infection, but that some individuals might experience additional financial hardship, particularly if they cannot work during prolonged courses of treatment. The introduction of needs-based discretionary payments for these Skipton Fund stage 1 recipients would be the best way of targeting resources at those in greatest need (Option 1.4).

5.7 The expert scientific review demonstrated that those with serious hepatitis C-related illness may have a reduced quality of life and significant morbidity and mortality. The evidence supports an increase in the Skipton Fund stage 2 payment (Option 1.2).

5.8 The expert scientific review provided evidence for a strong case for improving the current provision for those who have developed B-cell NHL as a result of their hepatitis C infection, due to the impact on quality of life and life expectancy. The evidence supports the case for these individuals to receive Skipton Fund stage 2 payments.

5.9 There is also a case for the introduction of recurrent financial support for Skipton Fund stage 2 recipients, and provision for needs-based discretionary payments for these recipients (Option 1.3 and Option 1.4 respectively). This would remove an anomaly between HIV and hepatitis C financial support and would improve the financial security of these individuals. Flat-rate recurrent financial support for stage 2 recipients would need to be made at the same level as for those with HIV (£12,800 is the current flat-rate annual payment for HIV infection), to avoid creating a new anomaly between patient groups. These payments could be uprated annually, in line with the CPI, to keep pace with living costs.

5.10 The review also considered options for increasing the level of flat-rate recurrent financial relief for the hepatitis C patient group (Option 1.3b). An option that was costed was the total average annual payment to an HIV-infected individual in 2009/10 - £17,400. This figure is a combination of the flat-rate recurrent payment and additional discretionary payments based on need. However, this average figure is likely to be lower in 2010/11, because certain special discretionary

payments formerly made by the Trust will cease, though general needs-based discretionary provision remains.

5.11 However, an advantage of setting the level of payment at £12,800 is that, given the current fiscal context, funding available could be most effectively directed at those in greatest need through discretionary payments. On balance, taking into account the aim of maintaining discretionary payments and overall affordability of the wider package of measures within the overall fiscal context, the review concluded that the flat-rate recurrent payment should be £12,800.

5.12 With regards to those individuals who are co-infected with both hepatitis C and HIV, they would receive separate recurrent annual payments in respect of each infection, in addition to the various lump sum payments.

5.13 The possibility of making available a lump sum as an alternative to annual payments to those with HIV and hepatitis C was not taken forward due to concerns that it would create unfairness in the system with people who have longer lives once infected not realising the same levels of support as those who pass away soon after receiving the lump sum payment.

Table 1 – Options considered for payments to individuals infected with hepatitis C

Option	**Description**	**Estimated additional cost in England***
1.1	Keep Skipton Fund stage 1 and stage 2 payments at current levels – do nothing additional.	£0
1.2	Keep Skipton Fund stage 1 payments at the current level, but increase the payment at stage 2 by £25,000, both prospectively and retrospectively.	£19 million in the first year for retrospective payments £2 million in the first

		year for prospective payments, declining in subsequent years
1.3	(a) Introduce annual flat-rate payment of £12,800 for living Skipton Fund stage 2 recipients, prospectively, uprated annually by the CPI. (b) Introduce higher annual flat-rate payment for living Skipton Fund stage 2 recipients, prospectively. As an example, an increase of £4,600 for the higher rate was costed.	(a) £8 million in the first year, increasing to £9 million in subsequent years[2] (b) £11 million in the first year, increasing to £13 million in subsequent years.
1.4	Introduce discretionary payments for Skipton Fund stage 1 and stage 2 recipients, based on need and the same principle as those available for HIV.	At the discretion of the Trustees, within the resources allocated for discretionary payments.

* All costs are as of 2010 and rounded to the nearest million. Uprating of annual payments will increase the cash costs in future years, depending on the level of the Consumer Price Index.

5.14 Options 1.2, 1.3a and 1.4 were taken forward as recommendations (see Section 11 of this report).

<u>Options considered for payments either in respect of, or to the dependants of, individuals who were infected with hepatitis C and who have died</u>

5.15 When the Skipton Fund was set up, the decision was made not to make payments in respect of those who had died prior to the announcement of the scheme on 29 August 2003. Campaigners have long argued that this cut-off is unfair and that payments in respect of those who would have been eligible to receive payments from the Skipton Fund, but who died prior to 29 August 2003, should be available.

[2] These figures are at 2010 prices but would be higher in future years when expressed as cash costs, owing to annual uprating by the CPI.

5.16 In addition, many infected individuals have expressed concern about the financial security of their dependants after they die. Representations have been received for some provision of financial security for dependants after the death of the infected individual.

5.17 The review proposed options for payments either in respect of, or to the dependants of, individuals who were infected with hepatitis C and who died. These options are not mutually exclusive and are summarised in Table 2.

5.18 To address the representations concerning those who died before 29 August 2003, a proposed option is the payment of the equivalent of a Skipton Fund stage 1, or both a stage 1 and stage 2, lump sum payment to the estate of an individual who was infected with hepatitis C but died before 29 August 2003, in accordance with Skipton Fund eligibility criteria (Option 2.2).

5.19 A key consideration in the implementation of Option 2.2 is the medical evidence needed in respect of those who died before 29 August 2003, in order to qualify for a payment, in line with the current eligibility criteria of the Skipton Fund, since medical records may not be available. Therefore, it is possible that in some cases it may not be possible to provide the necessary evidence. It might be difficult, or even impossible, to make payments in all genuine cases, without relaxing the requirements which would introduce a significant risk of inappropriate claims. This is an identified risk, although the aim would be to strike the right balance between meeting genuine claims and avoiding inappropriate ones.

5.20 Another option considered the introduction of a new lump sum payment for bereaved dependants, including the dependants of individuals who died prior to 29 August 2003 (Option 2.3). This would meet representations from campaigners in respect of providing financial support for dependants. However, other measures proposed in this report (lump, flat-rate recurrent and discretionary payments) would also go a considerable way to enabling infected individuals and their dependants to receive the support they have sought. The sums involved are potentially very large, because this would need to also apply to the bereaved dependants of all infected individuals (HIV and hepatitis C),

in order to avoid creation of a new anomaly. Therefore, this option was not taken forward.

Table 2 – Options considered for payments either in respect of, or to dependants of, individuals who were infected with hepatitis C but who have died

Option	Description	Estimated additional cost in England*
2.1	Keep as is currently – do nothing additional.	£0
2.2	A Skipton Fund stage 1 and/or stage 2 payment in respect of an otherwise eligible individual who died prior to 29 August 2003, using existing Skipton Fund eligibility criteria.	£30-59 million in the first year[3] The figure used for stage 2 in this calculation includes the proposed increase of £25,000 to the stage 2 payment (Option 1.2)
2.3	A lump sum payment of £50,000 for a dependant on the death of the infectee (hepatitis C and/or HIV), to be applied prospectively and retrospectively in respect of all infected individuals.	£42 million, with an upper estimate of £66 million, in the first year Additional average £1 million per

[3] This estimated cost range may not reflect the true cost of this option. It has been calculated using the estimated number of deaths of Skipton fund beneficiaries between 1995 and 2003, which are outlined in Annex 2 (Table A2.1). The number of deaths before 29 August 2003 is very uncertain but reflect our best estimates based on the published literature and expert advice. The lower figure of the quoted cost range is obtained by assuming that successful applications are made in respect of 30% of the total estimated number of infected individuals. The upper figure similarly assumes successful applications are made in respect of 30% of stage 1 claims, but increases to 100% for the stage 2 claims. This higher estimate for successful stage 2 claims is based on an assumption that many of the family members who have campaigned for payment in respect of those who died before 29 August 2003 will have documentary evidence to support a successful stage 2 claim.

		annum These figures are for dependants of those who have died of hepatitis C and/or HIV
2.4	Needs-based discretionary payments for the dependant of an individual infected with hepatitis C but who has died	At the discretion of the Trustees within the resources allocated for discretionary payments.

*All costs are as of 2010 and rounded to the nearest million.

5.21 Options 2.2 and 2.4 were taken forward as recommendations (see Section 11 of this report).

Provision for dependants of living individuals infected with hepatitis C

5.22 The review has considered new discretionary payments in respect of the dependants of living individuals infected with hepatitis C by NHS-supplied blood transfusions or blood products (Option 3.1).

5.23 Although focusing resources on payments to infected individuals should address the needs of families and children, new discretionary arrangements to reflect individual circumstances and hardship would be a fair and appropriate use of the funds. These would be made at the discretion of the Trustees to either the infected individual in life and their dependants thereafter or, in certain circumstances, their dependant directly, as is the case for HIV. This option was taken forward as a recommendation (see Section 11 of this report).

Table 3 – Option for provision for dependants of living individuals infected with hepatitis C

Option	**Description**	**Estimated additional cost**

3.1	Needs-based discretionary payments for the dependant(s) of an individual infected with hepatitis C.	At the discretion of the Trustees within the resources allocated for discretionary payments.

Section 6 – Making *ex-gratia* payments through the Department for Work and Pensions benefits system

6.1 Evidence given to the Archer inquiry from affected individuals suggests that applying to the existing charitable Trusts is viewed as demeaning for some individuals, who see themselves as being forced to beg for hand-outs. Lord Archer argued in his report that making *ex-gratia* payments through the DWP benefit payment systems would give the Government direct responsibility for providing these resources. However, the written submission from the campaign groups stated that they wanted the existing charitable Trusts to continue providing support.

6.2 From this review, it is not clear that there would be any tangible benefits from making *ex-gratia* payments through the benefits system. Firstly, it would be inappropriate for the DWP to administer these *ex-gratia* payment schemes as they address health specific issues and it would not be consistent with DWP's role. Secondly, the mechanism for administering the schemes is well established through the Trusts, and incorporates the necessary health expertise to determine eligibility. This option was therefore not taken forward as a recommendation.

Section 7 – Access to insurance

7.1 This review sought to address the concerns of individuals infected with hepatitis C and/or HIV by NHS-supplied blood transfusions or blood products about access to insurance. The biggest representation from these individuals is the desire to ensure that their dependants have a degree of financial security in the case of their death, as well as concerns about travel and mortgage payment protection insurance.

7.2 The review considered a range of options to address any potential additional detriment to accessing insurance, arising directly as a consequence of becoming infected with hepatitis C and/or HIV from NHS-supplied blood transfusions or blood products.

7.3 In preparing options, advice has been taken from HM Treasury, the Association of British Insurers (ABI), Hannover Life Reassurance (UK) Ltd, and the Department for Health and Children in the Republic of Ireland.

Background

7.4 Insurers assess risk, taking into account the applicant's medical history, when deciding whether or not insurance can be offered, and if so with what premium loading and/or exclusion.

7.5 Individuals who have been infected with HIV and/or hepatitis C by NHS-supplied blood transfusions or blood products may face difficulties in obtaining insurance that is assessed using medical history. Infected individuals, either with or without underlying haemophilia, may be subject to significant premium loadings, some of which might be prohibitive for them. People with haemophilia who are co-infected with HIV and hepatitis C will not be able to obtain some forms of insurance such as life cover. For individuals who are mono-infected with HIV or hepatitis C some insurance terms may be available (see **Annex 6**).

7.6 The review noted that people with severe haemophilia may, in any event, have difficulty obtaining life cover, even if not infected with HIV or hepatitis C, purely as a consequence of their haemophilia.

Life cover

7.7 Accidental death insurance, which pays out in the event of, for example, a traffic accident (and not any underlying health condition) is available, irrespective of the individual's medical history.

7.8 The table at **Annex 6,** provided by ABI, considers the availability of life cover for individuals infected with hepatitis C and/or HIV by NHS-supplied blood transfusions or blood products. It is an indicative example only and does not purport to represent the industry. The table shows that people with haemophilia who are not infected with hepatitis C or HIV can have access to life cover, albeit with increased premium loadings of up to 300% for those with severe haemophilia. Those who are additionally infected with hepatitis C are likely to be subject to an additional premium loading that might be within the range of 75% to 250%. People who are infected with HIV may be able to obtain life insurance for example for a period of ten years, up to the age of 60, with an additional premium that might be within the range of £3 to £10 per £1,000 sum assured. Life cover will only be available if the hepatitis C and HIV have been successfully treated/controlled with drugs and will invariably not be available to those who are co-infected.

7.9 This indicates that it is quite difficult to disentangle the premium loadings associated with haemophilia, or any other underlying condition, from the premium loadings associated with hepatitis C or HIV infection. The review noted that the decision to take out life insurance is, and should remain, a personal choice.

Mortgage payment protection insurance

7.10 Mortgage payment protection insurance is not required as a condition for obtaining a mortgage. Those who do choose such a policy will usually find benefits are available for a maximum period of 12 months, and some do not pay out for illnesses related to pre-existing medical conditions, or for redundancy that could have been foreseen when the policy was taken out.

Travel insurance

7.11 The review identified that travel insurance is usually available to infected individuals from specialist providers. If individuals wish to travel with full cover, cost will be a factor. Premium loadings may vary depending on the overall health of the applicant as well as the destination and duration of the holiday. However, some holiday providers utilise group or multi-people plans and it is unlikely that infected individuals would be disadvantaged or refused in such circumstances. The review noted that the decision to travel overseas is, and should remain, a personal choice.

Replicating the insurance scheme that operates in the Republic of Ireland

7.12 The insurance scheme in the Republic of Ireland is cited by campaigners as a potential model to create a similar scheme in the UK. The review considered the option of setting up a state run insurance scheme akin to the Irish scheme that would provide life cover, travel insurance and mortgage payment protection insurance (Option 4.2).

7.13 However, the Irish insurance scheme[4] takes no account of haemophilia because it treats the applicants as if they have no underlying health condition(s). It also has high administration costs – in excess of €137,000 in 2009 for only €570,000 paid out in actual policies – and has had a relatively low take-up:

- Take-up of life assurance – 17% of those eligible
- Take-up of mortgage protection insurance – 0.5% of those eligible
- Take-up of travel insurance – 17% of those eligible.

7.14 The Irish scheme does not therefore represent good value for money as the administrative costs are disproportionately high for the amount of cover provided, due to the low take-up. It is considered unlikely that a similar scheme would represent value for money in the UK. Therefore this option was not taken forward as a recommendation.

[4] Information on the Republic of Ireland's insurance scheme was taken from the 2009 Annual Report for the Hepatitis C & HIV Insurance Scheme, as laid before both houses of the Oireachtas

Increasing *ex-gratia* payments

7.15 Instead, the review considered focusing resources on *ex-gratia* payments to provide financial security in life (Option 4.3).

7.16 Where commercial insurance products are available, the increased *ex-gratia* payments, including the discretionary elements, proposed in other sections of this report will help individuals pay for premiums if they wish. This option was taken forward as a recommendation (see Section 11 of this report).

Table 4 – Options considered for access to insurance

Option	**Description**	**Estimated additional cost**
4.1	Keep as is currently - do nothing additional.	£0
4.2	Introduce a state run insurance scheme akin to the scheme available in the Republic of Ireland, including life cover, travel and mortgage payment protection insurance	Not known – would depend on take-up

Section 8 – Prescription charges

8.1 The review considered whether individuals who were infected with HIV and/or hepatitis C by NHS-supplied blood transfusions or blood products should be exempted from prescription charges.

8.2 A large proportion of this patient group will already receive free prescriptions because they fall into one of the existing prescription charge exemption categories (primarily age related) but there are a number of individuals who are not exempt from charges.

8.3 It would be administratively complex and require changes to secondary legislation to tie this small group of individuals into a new exemption category under the existing exemption arrangements. Instead, payments administered by the *ex-gratia* payment schemes could be made to those infected individuals who are not otherwise exempt from charging. All beneficiaries of the Macfarlane and Eileen Trusts and the Skipton Fund who still pay prescription charges could apply for a payment that would cover the cost of an annual prescription pre-payment certificate (currently £104 a year) subject to making a declaration that they are liable to pay prescription charges.

8.4 This option was taken forward as a recommendation (see Section 11 of this report).

Section 9 – Access to nursing and care services

9.1 Evidence gathered for the review has shown that there are a number of issues related to the current provision for social care services and home nursing, that are of concern to individuals who have been infected with HIV and/or hepatitis C through NHS-supplied blood transfusions or blood products, for example:

- individuals cannot access sufficient nursing or social care, and that they often have to pay for it;
- individuals who develop terminal disease will require continuous nursing care in the period leading up to this stage, and that some have encountered problems accessing continuous home nursing care, forcing them to rely on unpaid informal carers;
- the level of knowledge about HIV and hepatitis C among nursing and care providers;
- individuals wish to ensure that they have access to appropriate nursing and/or social care at the point in their illness when they consider it would be most valuable;
- infected individuals may need both social and/or community nursing care and help in maintaining their quality of life and their independence. Their families may also require help in providing physical and emotional care, to support them in their caring role.

<u>Current provision</u>

9.1 NHS nursing care is provided free, irrespective of whether it is provided in a care home or the individual's own home. It is not means tested. Community nursing services have a key role with individuals, families and carers, to encourage and support infected individuals, to develop strategies to meet individual needs in collaboration with their family and other health and social care professionals and in providing direct treatment and care. Nurses are required by the Nursing and Midwifery Council code of conduct to ensure that they have the competence to provide high quality care. Provider organisations must ensure that any additional training required to provide care for this group of individuals is available to clinical staff. Community nurses are part of the primary health care team (PHCT, and the PHCT needs to work closely together and with other professionals to ensure seamless care is provided

9.2 Provision of social care services, including residential social care, is made through local authorities. Everyone is entitled to have their social care needs assessed free of charge by their local authority, but provision of social care is means tested, and those with a low level of financial resources can seek local authority support. There are different charging systems for residential and non-residential social care.

9.3 For residential social care, all residents must pay something towards their care. Local Authorities use the National Assistance (Assessment of Resources) Regulations 1992 and the Charging for Residential Accommodation Guide (CRAG) to assess how much a person is able to contribute. The assessment is based on the income and capital assets of the individual who needs care, not that of the household in which they live. In general, local authorities do not arrange care for anyone who has more than £23,250 in capital.

9.4 For non-residential social care, including home care, local authorities have discretionary powers to charge for arranging services. Statutory guidance, *Fairer Charging Policies for Home Care and other non-residential Social Services*, sets out the framework within which each authority designs its own charging arrangements. Whilst local authorities are free to decide not to charge for services, they must have regard to this guidance if they do. As with charging for residential care, the assessment should be based on the income and capital assets of the individual who needs care, not of the household in which they live. The guidance means that local authorities should not be less generous in their treatment of service users' assets and savings than is set out in CRAG.

9.5 There is a clear linkage between the way in which the Department of Work and Pensions (DWP) treats certain ex-gratia payments for the purposes of assessing a person's eligibility for benefits, and the treatment of those same payments by local authorities when assessing that person's means for the purposes of arranging social care provision. All current ex-gratia payments in regard of hepatitis C and HIV are already disregarded in regulations for the purpose of means testing for social care[5]. DWP has

[5] All these payments are disregarded by DWP for the purposes of benefits assessment, having been included in amendments to the relevant DWP regulation (Income Support (General) Regulations 1987). The DH National Assistance (Assessment of Resources) Regulations 1992 refer to the relevant part of the Income Support Regulations. Therefore, an amendment to the Income Support Regulations automatically amends the Assessment of Resources Regulations. This change should be reflected in CRAG, and the

agreed that any new ex-gratia payments will be disregarded for the purposes of assessing an individual's eligibility for benefits. It is intended that the same disregards will apply to local authorities when assessing capital assets in any means testing for social care services.

9.6 An alternative option briefly considered was for Secretary of State for Health to ask local authorities to use their discretionary powers to exempt ex-gratia payments to individuals infected with HIV and/or hepatitis C by NHS-supplied blood transfusions and blood products from means testing for non-residential social care services. However, local authorities would be under no obligation to comply, and as DWP agreed to disregard any new payments for the purposes of benefits assessment, there was no need to consider this further.

9.7 A further concern identified by the review is the wish for improved access to counselling, for both individuals and their families. There have been many complaints that individuals and families have received no counselling through the NHS. Additional provision for counselling would meet the specific request made in many letters that the Department has received, and could be delivered with a minimum additional administrative burden through relevant third sector organisations.

9.8 A summary of the options considered in relation to social care and counselling is presented in Table 5.

Table 5 – Options for access to care services

Option	**Description**	**Additional estimated cost***
5.1	Keep as is currently, do nothing additional.	£0
5.2	Make any new ex-gratia payments (and any new payments arising from this review) exempt from means testing for residential social care services.	No additional cost to local authorities, as there is no loss of funding.

CRAG guidance on treatment of capital then feeds through to the Fairer Charging guidance for non-residential social care.

5.3	Make additional provision for counselling	£100,000 per annum for 3 years

* All costs are as of 2010.

9.9 Options 5.2, and 5.3 were taken forward as recommendations (see Section 11 of this report).

Section 10 - Additional issues outside the Terms of Reference of the review

Ex-gratia payments for individuals infected with HIV and their dependants

10.1 Currently, HIV infected individuals receive a flat-rate payment of £12,800 per annum, with access to needs-based discretionary payments. This gave an average of £17,400 per infected individual in 2009/10.

10.2 In light of some of the key proposals for hepatitis C-infected individuals, as outlined in Tables 1-3, and the aim to avoid the creation of new anomalies between the HIV and hepatitis C patient groups, options were considered that mirror these proposals for HIV.

10.3 Option 6.2 mirrors the proposal for a higher flat rate payment in Option 1.3b, in both cases with the caveat that increasing the flat-rate payment will decrease the amount of discretionary funding available.

10.4 Option 6.3 mirrors the proposal for a payment to the bereaved dependant of a hepatitis C infected individual who has died (Option 2.3).

Table 6 – Options for payments to individuals infected with HIV and their dependants

Option	Description	Estimated additional cost*
6.1	Keep existing arrangements, but uprate the annual payment of £12,800 to infected individuals annually by the CPI, to keep pace with living costs.	Approximately £94,000 in 2011/12, rising to approximately £364,000 in 2014/15, and continuing to rise thereafter depending upon changes in the CPI.

6.2	Introduce a higher flat-rate recurrent payment for individuals infected with HIV. As an example, an increase of £4,600 for the higher rate was costed.	£2 million per annum, decreasing slightly over subsequent years
6.3	A lump sum payment of £50,000 for the dependant on the death of the infectee (hepatitis C and/or HIV). To be applied prospectively and retrospectively for the dependant of all infected individuals	See Option 2.3

* All costs are as of 2010 and rounded to the nearest million.

10.5 Option 6.1 is taken forward as a recommendation. For the reasons set out for hepatitis C in paragraphs 5.9, 5.10 and 5.20, options 6.2 and 6.3 were not taken forward as recommendations.

<u>Exemption from the DWP's Work Capability Assessments.</u>

10.6 Some campaigners have asked that individuals infected with HIV and/or hepatitis C by NHS-supplied blood transfusion or blood products should be exempted from having to undergo the Government's new Work Capability Assessment (WCA). The Department of Health has not received any reports that individuals in this patient group who are unable to work for health reasons are now being assessed as being capable of work.

10.7 The WCA is based on the principle that a health condition or disability should not automatically be regarded as a barrier to work, as a health condition will affect different people in different ways. By focusing on the functional effects of an individual's condition, rather than the condition itself, the WCA provides an assessment of what an individual can do, taking into account the requirements of the modern workplace.

10.8 The Government is committed to supporting those who cannot work because of a health condition or disability, and recognises that asking people to attend a face-to-face assessment unnecessarily is in no-one's interests. Therefore the WCA does not always include a face-to-face

assessment. Where possible, decision makers may use the paper-based evidence available, specifically information that the customer provides on their ESA50 questionnaire and information from their GP or consultant. However, in order to assess people fairly and accurately it is often necessary to assess them face-to-face, in order to understand how their condition affects them.

10.9 The review considered exemption from the WCA for this patient group with the DWP. However, the DWP have confirmed that individuals infected with HIV and/or hepatitis C by NHS-supplied blood transfusion or blood products will not be granted exemption from the WCA, given that the WCA is based on the principle that a health condition should not automatically be regarded as a barrier to work.

Access to dentistry for individuals infected with hepatitis C

10.10 During the backbench debate on 14 October, an MP suggested that hepatitis C sufferers often have significant problems with gum disease, and that there are issues around access to dentistry for that patient group. However, we are unaware of any evidence to support either of these statements. This issue has not been raised in correspondence, or in any of the submissions received during the course of the review, and we are unaware of this being a specific problem encountered by those with hepatitis C. The question is therefore whether there is a systemic problem around access to dentistry, or whether the issue that was highlighted was simply an example of local difficulties with access.

10.11 Department of Health advice to the dental profession provides for individuals infected with hepatitis C and HIV to be treated safely in general (high street) dental practices. However, dentists have discretion over which individuals to treat, and might refer an individual to the salaried service or a dental hospital. If infected individuals have been denied access to treatment for whatever reason, they have recourse to the NHS complaints system.

10.12 In-patient specialist/secondary dental care is free of charge; however, patients may have to pay for dental appliances, including crowns and dentures, for NHS Hospital Dental Service outpatient care. In respect of charges for high street dental care, it has been the policy of successive governments to base support for dental charges on income

rather than medical conditions. However, people in the following groups are exempt from charges:

- Aged under 18;
- Aged under 19 in full time education;
- Expectant mothers;
- Women who have had a baby in the last 12 months.

10.13 In addition, the following groups have their dental charges remitted:

a. Those receiving income support, and their partners;
b. Holders of an NHS low income scheme HC2 certificate;
c. Holders of an NHS low income scheme HC3 certificate (partially remitted only);
d. Those receiving job seekers allowance, and their partners;
e. Those receiving Income-related Employment and Support Allowance and their partners;
f. Those receiving Pension Credit Guarantee Credit; and
g. Those named on a valid NHS tax credit exemption certificate, or entitled to an NHS tax credit exemption certificate.

10.14 Many recipients of disability living allowance, incapacity benefit, and other benefits would receive full or partial exemption on application for help from the NHS low income scheme.

10.15 In conclusion, there does not appear to be a systemic problem with access to dentistry for hepatitis C patients. Individual patients might experience problems locally, and they need to be resolved locally via the local complaints procedures.

Section 11 – Recommendations and conclusions

Recommendations

11.1 In deciding on an appropriate package of measures, the representations made by those affected have been considered together with the expert scientific evidence on the spectrum and impact of disease associated with hepatitis C infection, as well as the cost of the various options under consideration, and the affordability in the financial context of the current spending review, given the current fiscal context.

11.2 Based on these considerations, and the principles underpinning the review identified at paragraph 3.6, the following package of measures has been developed:

i. Introduce a recurrent flat-rate annual payment of £12,800 for all living Skipton Fund stage 2 payment recipients.

   Set up access to additional discretionary payments for those infected with hepatitis C by NHS-supplied blood transfusions and blood products, and for the dependants of infected individuals, including of those who have died, targeted at those in greatest need.

   These measures will reduce anomalies with HIV payments. Those who have already received a stage 2 payment will start to receive recurrent payments as soon as the necessary arrangements can be put in place, backdated to the date of the announcement of this review report. New individuals who meet the Skipton Fund stage 2 eligibility criteria in future will receive the lump sum stage 2 payment and the first of the recurrent payments prospectively from the date of that payment.

   Individuals who have been infected with HIV, and who have severe liver disease as a result of their hepatitis C infection, will receive two flat-rate annual payments of £12,800, one in respect of each infection.

ii. Uprate levels of this new flat-rate recurrent payment for hepatitis C, as described in (i), and the existing payment for HIV, in line with the CPI annually, to keep pace with living costs.

Officials in HMRC and DWP have confirmed that continued exemption of all payments from tax and benefit calculations can be secured. It may take a little time to set up the necessary payment arrangements but all flat-rate recurrent payments will be backdated to the date of the announcement of this review report or the date of the making of a Stage 2 payment as appropriate.

iii. Extend eligibility for stage 1, or both stage 1 and stage 2, payments, based on the eligibility criteria of the Skipton Fund, in respect of an individual who was infected with hepatitis C through NHS-supplied blood transfusions or blood products, but who died prior to 29 August 2003. Claims are to be registered by the end of March 2011.

iv. Make a further payment of £25,000 to those who receive a Stage 2 payment from the Skipton Fund.

Patients who have developed hepatitis C-related B cell non-hodgkins lymphoma, will become eligible for a Skipton Fund stage 2 payment, as well as the additional lump sum of £25,000 for Skipton Fund stage 2 recipients.

This payment should also be applied in respect of individuals infected with hepatitis C through NHS-supplied blood or blood products who died before 29 August 2003.

v. Introduce application for a payment to cover the cost of an annual prescription season ticket for individuals infected with HIV and/or hepatitis C by NHS-supplied blood transfusions and blood products, so that those who are not otherwise exempt from charges will not have to pay for their prescriptions.

vi. Update the Social Care guidance to reflect regulations which exempt ex-gratia payments from means-testing for social care.

vii. Provide £100,000 per annum to selected third sector organisations over the next three years to provide additional access to counselling for individuals infected with HIV and/or hepatitis C by NHS-supplied blood transfusions and blood products.

11.3 There are no changes proposed to the mechanisms by which the *ex-gratia* payments are made. It is appropriate that the *ex-gratia* payment schemes should continue to be administered by the Department of Health, as the Department that works most closely with the NHS.

11.4 There is not a systemic issue with respect to access to dentistry for this patient group, so no changes are proposed.

11.5 The DWP has confirmed that those infected with HIV and hepatitis C by NHS-supplied blood transfusions and blood products are not exempt from the Work Capability Assessment.

11.6 Whilst it is recognised that some infected individuals might be uninsurable for some risks, a state run insurance scheme is not considered to represent value for money. Individuals for whom insurance is available have freedom to use the *ex-gratia* payments that they receive to help pay for the premiums.

11.7 The increased provision in the *ex-gratia* schemes could go some way to giving infected individuals and their families choice about how to tailor any nursing and social care needs over and above those freely available.

Conclusion

11.8 This package of measures, coupled with the support that is currently available to these infected individuals and DWP benefits that they are eligible for, is consistent with the evidence for greater support for those with hepatitis C, and their dependants. Importantly, it also removes the anomaly that prevents payments in respect of hepatitis C infection being unavailable in respect of those who died before 29 August 2003. Overall it is considered a balanced package of support, is within the range of the representations made by those affected during the course of the review, and given the current fiscal context, meets the principles set out for conduct of the review.

Annex 1

WRITTEN MINISTERIAL STATEMENT

DEPARTMENT OF HEALTH

Support for those affected by contaminated blood

Thursday 14 October 2010

The Parliamentary Under Secretary of State, Department of Health (Anne Milton): On 16 April 2010 Judgement was handed down on a Judicial Review of a decision made by the previous Government not to accept a recommendation made in the report of Lord Archer of Sandwell's independent inquiry into infections transmitted some decades ago through contaminated blood products. The recommendation in question, 6(h), which concerned payments to those affected by this tragedy, stated that:

> "We suggest that payments should be at least the equivalent of those payable under the Scheme which applies at any time in [the Republic of] Ireland."

The Judgement found against the Government, therefore I am now required to look again at this recommendation, and decide whether or not to accept it.

Having carefully compared the circumstances pertaining here and in the Republic of Ireland during the period when most of the infections occurred, and having taken account of the fact that this tragedy similarly affected many other countries; I do not consider there is a case for accepting Lord Archer's recommendation 6(h) that levels of payment here should match those made in Ireland. Every country must make its own decisions on financial support for those affected, taking account of its own particular circumstances, and affordability. The scheme in Ireland was set up on that basis, and has not been replicated in any other country, as far as we know. However, our ex-gratia payment schemes for HIV compare well with those of other countries.

In addition, it is estimated that implementing a similar scheme to Ireland's here in the UK, would cost in excess of £3 billion.

I recognise that this decision will disappoint those who are living with serious health problems as a result of their infections, as well as their families and the families of those who have already died. During the summer I met representatives of those affected, and heard first hand about the hardships that they have to face on a daily basis.

I believe that to a large extent the recommendations are already in place. The previous Government increased the level of payments to those affected with HIV to a minimum of £12,800 per annum, and has increased the discretionary funding available to their dependents. I do not intend to revisit that decision, but I am persuaded that there are some aspects of Lord Archer's recommendations that should be looked at afresh. These include:

- the level of ex-gratia payments made to those affected by hepatitis C, including financial support for their spouses and dependants, and taking account of the level of payments made to those infected with HIV in the UK and via schemes in other countries;

- the mechanisms by which all ex-gratia payments are made;

- access to insurance;

- prescription charges;

- access to nursing and other care services in the community.

I am initiating a review of the issues raised by these recommendations, which will take place in the context of the current financial climate and results of the Spending Review. Terms of reference have been placed in the Library. I expect to be able to report the outcome of this work and my intentions by the end of 2010. I will be speaking to the other UK Health Ministers to seek their confirmation whether they wish to participate in reviewing the UK-wide aspects within this timescale or whether I will proceed on an England only basis.

Annex 2

Estimate of total number of hepatitis C-infected individuals in UK infected over the period 1970-1991

Year	All HCV	Chronic HCV[2]	Cirrhosis[3]
Blood transfusions			
All	28,043[1]	21,032	4,206
Those alive in 1995	9,785[1]	7,339	1,468
Those alive in 2003	5,609[4]	4,207	841
Clotting factor products (bleeding disorder patients)			
All	4,675[5]	3,506	701
Those alive in 1995	3,500[6]	2,625	525
Those alive in 2003	3,000[6]	2,250	450
All infected individuals (i.e. combines those infected from blood transfusions and clotting factor products)			
All	32,718	24,539	4,907
Those alive in 1995	13,285	9,964	1,993
Those alive in 2003	8,609	6,457	1,291
Deaths (1995 to 2003)	4,676	**3,507**	**702**

[1] Source: Soldan, Ramsay, Robinson et al. The contribution of transfusion to HCV infection in England. Epidemiology and Infection 2002. 128, 587-591 (corrected to UK)

[2] Assumes that 75% of those infected with acute HCV will develop chronic infection (see Chen SL, Morgan TR. The Natural History of Hepatitis C Virus (HCV) Infection. Int J Med Sci 2006; 3(2):47-52 and Micallef JM, Kaldor JM, Dore GJ, Spontaneous Viral Clearance Following Acute Hepatitis C Infection: A Systematic Review of Longitudinal Studies. J Viral Hepat. 2006;13(1):34-41)

[3] Assumes that 20% of those chronically infected will develop cirrhosis (see Seeff LB, Buskell-Bales Z, Wright EC et al. Long term mortality after transfusion associated NANB hepatitis. N Engl J Med 1992; 327: 1906-1911, Seeff LB, Hollinger B, Alter AJ et al. Long-term morbidity of post-transfusion hepatitis C. Hepatology 1998; 28:407A, Pagliaro L, Peri V, Linea C et al. Natural history of chronic hepatitis C: a systematic review. It J Gastroenterol 1999; 31(1): 28-44 and Seeff L. The history of the "natural history" of hepatitis C (1968-2009). Liver International 2009; 29(s1):89-99)

[4] We have assumed that 20% of those infected were still alive by 2003, in line with data from the UK HCV National Register

[5] From the UKHCDO Annual Report 2010

[6] UKHCDO Annual Report suggests that 2,775 HCV infectees with bleeding disorders were alive in 2010; this provides some broad indication as to the proportion of infectees who may have been alive in 1995 and 2003.

A2.2: Estimated number of infected beneficiaries of ex-gratia payments for Hepatitis C in UK

	Stage 1	Stage 2
Total number of infected beneficiaries to end of 2010/11 [7]	4,310	901
Number of infected beneficiaries still alive at end of 2010/11 [8]	3,050	736
Number of infected beneficiaries still alive at end of 2011/12 [9]	3,393	783

[7] Provided by Skipton fund (assuming the same number of new recipients in the last 4 months of 2010/11 as the previous 4 months).

[8] Estimated assuming that 3% and 5% of Skipton Stage 1 and Stage 2 infected beneficiaries have died per year since the beginning of the scheme.

[9] This is estimated from the number of infected beneficiaries still alive at the end of 2010/11 minus the expected number of deaths in 2011/12 plus the additional number expected to join in 2011/12 plus a number of extra claimants due to demand effects.

Annex 3						
	Scheme	**Date established**	**Who is eligible for payment?[6]**	**Payment type and mechanism**	**Range of payment received by primary infected individual**	**Who pays?**
HIV	Macfarlane Trust (MFT) for haemophilia patients Eileen Trust (ET) for non-haemophilia patients	1987 1993	Infected individuals and Dependants (Payments to bereaved dependants have been limited by the size of discretionary funds available)	Charitable Trusts Lump sum payments from the late 1980s to those infected, and annual discretionary payments thereafter From 20 May 2009, flat-rate recurrent payment of £12,800[7], with continuing scope for discretionary payments Also, discretionary payments to dependants	For MFT: Between £43,500[7] (where death occurred before or upon establishment of scheme) to around £150,000 - £180,000 to date to those still living For ET: £43,500[7] (where death occurred before or upon establishment of scheme) to around £80,000 - £150,000 to date to those still living	UK Government on behalf of all
Hep C	Skipton Fund (no distinction between haemophilia and non-haemophilia patients)	2003 (became operational in 2004)	Only infected individuals who were living on 29 August 2003 (date scheme was announced) No payments to dependants	Company limited by guarantee Lump sum of £20,000 for chronic infection (stage 1) Further lump sum of £25,000 for cirrhosis and its complications (stage 2)	£20,000 for chronic infection (stage 1) Further £25,000 for cirrhosis and its complications (stage 2)	England administers. Each UK country meets the cost of their Stage 1 and 2 payments made to victims infected in their country. DAs also make a contribution towards the running costs of the scheme as agreed in SLA agreement

[6] Dependants: spouses (including partners), parents, children and other dependants

[7] Non-discretionary payments were made through non-charitable mechanisms – MFET Ltd and MSPT2

Annex 4

REVIEWING THE NATURAL HISTORY OF HEPATITIS C INFECTION

1. INTRODUCTION

Hepatitis C virus (HCV) is a blood-borne virus that is an important cause of chronic liver disease and liver cancer in the UK. Data from the Health Protection Agency (HPA) estimate that 142 000 individuals aged 15-59 years are living with chronic hepatitis C infection in England and Wales.[1] Routes of transmission vary worldwide and include exposure to infected blood products, injecting drug use, vertical transmission and rarely through sexual transmission. In the UK, the introduction of blood donor screening for HCV antibodies in September 1991 has had a major impact on the acquisition of HCV, and injecting drug use is now the most common reported route of transmission.

Characterising the natural history of HCV infection is important to understand its impact on an individual, but also to determine the population burden for health service planning. However, the changing epidemiological pattern and a greater understanding of the impact of co-factors on disease progression have added to the complexity of developing a general model that describes the natural history of HCV.[2]

Infection with HCV causes acute and chronic liver disease with differing severity and outcomes, and is associated with extra-hepatic manifestations that are related to chronic stimulation of the immune system and to virus-induced autoimmunity.[2] This paper reviews published evidence on the different stages of HCV infection, rates of progression and impact of disease in each of these stages. The evidence was then reviewed by an expert working group set up by the Department of Health (see the end of the document for list of members).

2. NATURAL HISTORY

a. Clinical course of acute hepatitis C infection

Acute hepatitis C refers to the period immediately following incubation. Exposure to the virus is normally followed at around 6-8 weeks by a rise in alanine aminotransferase (ALT) with or without mildly raised bilirubin.[3] Infection may be asymptomatic, but can be accompanied by a short-lived acute hepatitis (including malaise, anorexia and jaundice).

Acute HCV infection is asymptomatic or mild in 70-80% cases [4] and a fulminant course is extremely rare in the absence of co-infection or other modifiable co-factors.[2] Therefore, acute infection is infrequently diagnosed and the majority of acutely infected individuals are unaware of their diagnosis.[5] In a prospective study of 117 patients with post-transfusion hepatitis, sustained clearance of serum HCV RNA was observed in 15%, 12% had normalised alanine transaminase or ALT (but remained viraemic) and 73% progressed to chronic hepatitis.[6] In a systematic review of 31 longitudinal studies (n=675), the proportion with viral clearance ranged from 0.0-0.8 with a weighted mean of 0.26 (95%CI: 0.22-0.29).[7] In addition, where acute infection is detected and treated promptly, high response rates to standard or abbreviated treatment courses (up to 98%) have been reported.[8,9] The impact of acute HCV infection on an individual therefore appears to be limited unless those individuals progress to chronic infection.

Chronic hepatitis C is marked by the persistence of HCV RNA in the blood for at least 6 months after the onset of acute infection. Approximately 75-85% of infected patients do not clear the virus by 6 months and chronic hepatitis C infection develops.[5,7] The proportion of patients who develop chronic HCV infection may be determined by many factors. These include age at time of infection, gender, ethnicity, presence of symptoms during the acute infection, genotype, immuno-suppression and HIV infection.[5,10]

Chronic HCV infection may develop with or without ALT abnormalities and with persistent or intermittent viraemia.[2] Prospective studies suggest that around 60-90% of acutely infected individuals have ALT abnormalities and progress to chronic infection (see below) while the remaining 10-40% have persistently normal ALT levels and progress more slowly.[2]

b. Progression of chronic hepatitis C infection

Determining the natural history of HCV infection and rate of progression to cirrhosis is challenging. Estimates of progression have largely been derived from cross-sectional studies based in secondary or tertiary care. Long-term prospective follow-up of large cohorts are required to provide accurate estimates of progression to cirrhosis and hepatocellular carcinoma. Such cohorts are difficult to identify and data from one cohort may not be generalisable to another that differs with respect to their route of acquisition, their age and gender profile and the presence of other factors important to progression.

A number of studies have assessed the long-term clinical and histological outcomes of HCV in different cohorts of patients with a well-defined time point of contamination.[11,12,13,14] In one of the most extensive studies, Seeff *et al.* followed a large number of cases who acquired post-transfusion hepatitis between 1968-1980.[11] Assessment at twenty years after infection found 26% cases had cleared infection and 15% had developed cirrhosis.[12] Follow up of 376 (n= 390) Irish women infected with HCV during 1977-78 from contaminated anti-D immunoglobulin found that only 2% had cirrhosis 17 years after infection.[15] This low rate of significant fibrosis persisted at the 27 year follow-up study.[16] In a multi-centre cohort study of 847 haemophiliac patients with hepatitis C, the cumulative incidence of end stage liver disease was 11.5% in HIV-negative patients after 35 years.[17] Overall mortality was reported at 24% with 6% of patients dying of liver disease, although the proportion of deaths amongst co-infected individuals was not reported. Risk factors for rapid progression included alcohol abuse, HIV co-infection, older age at infection and presence of HCV genotype 1.

A number of other studies have attempted to measure the time interval from infection to cirrhosis and hepatocellular carcinoma in different population groups. Although the mean time to cirrhosis is estimated at 20 years, only 10-20% patients will actually develop cirrhosis within this time period.[18] In a European study by Castells *et al.* the mean time to development of cirrhosis and HCC was 24 years and 27 years, respectively.[19] The prognosis for patients infected for longer than three decades remains uncertain but there are increasing data suggesting that disease progression increases with age, probably leading to higher rates of cirrhosis in patients infected for more than 30 years.

In a systematic review of 111 published studies, the estimated prevalence of cirrhosis at 20 years was 16% (95%CI: 14-19%) for all studies, but only 7% (95% CI: 4-12%) for studies conducted in non-clinical settings.[20] Data on 987 HCV-infected patients from three UK observational cohorts from different referral sources demonstrated different progression rates.[21] The estimated 20 year probability of progression to cirrhosis was 12% (95% CI: 6-22) in a hospital based cohort, 6% (95%CI: 3-13) in a post-transfusion cohort and 23% (95%CI: 14-37) in a cohort recruited from a tertiary referral centre.[21] These studies suggest that observed progression rates appear to be higher in cohorts presenting for clinical care; in contrast, individuals recruited prior to the development of symptoms have a more favourable course. Despite this potential bias in many published studies, factors that have been associated with progression include gender, age at acquisition, duration of infection, ALT levels, genotype,

smoking, alcohol consumption and presence of co-morbidities (including co-infection with HIV or HBV).[22, 23] Observational prospective studies and modelling predictions suggest that the risk of progression to severe fibrosis/cirrhosis is minimal in those with persistently normal ALT levels.[24]

c. Mortality

In a UK study of HCV-infected transfusion recipients, all-cause mortality during the first decade of infection was 1.4 times greater than that observed in a similarly traced group of transfusion recipients negative for HCV, and after 16 years all-cause mortality was 1.2 times greater.[25,26] However, this did not reach statistical significance. During the first ten years, the risk of dying directly from liver disease was almost 6 times higher for people infected with HCV, but this difference was not significant.[25] Excess alcohol consumption was implicated in 40% of the deaths from liver disease among patients. Other studies have quoted mortality rates between 2.5-14%, which may be due to differences in follow-up and inclusion of patients at different stages of HCV-related disease.[11,24] In a multi-centre cohort study of 847 haemophiliac patients, overall mortality after 35 years was reported at 24% with 6% of patients dying of end stage liver disease. [17]

3. SPECTRUM OF CLINICAL OUTCOMES IN HCV INFECTION

HCV infection can lead to a wide spectrum of clinical outcomes ranging from acute asymptomatic infection with spontaneous resolution to decompensated liver cirrhosis and hepatocellular carcinoma (Table 1). The severity and stage of compensated chronic HCV infection can be defined according to histological criteria on the basis of the extent of necroinflammation and fibrosis.[27]

a. Mild chronic hepatitis C

Many patients with chronic hepatitis C infection are found to have a mild form of liver disease. This includes individuals who are asymptomatic with persistently normal or nearly normal ALT levels or those with abnormal ALT who have minimal/mild liver histological lesions.[24]

Population-based studies have demonstrated that approximately 50% of chronically infected individuals have persistently normal ALT levels and around two thirds have mild histological liver lesions.[24] Studies on the natural history of mild disease indicate that the short-term outcome is always benign. However, progression of liver fibrosis can be observed in the longer term, particularly in those with elevated and/or fluctuating

ALT levels.[24] Patients with mild liver damage associated with hepatitis C may report symptoms such as fatigue, malaise, bodily pain and joint symptoms.[28] Reduced health-related quality of life (HRQoL) is commonly reported in these individuals.[29] In a trial of therapy, the mean baseline HRQoL score for patients with mild disease was reported as 0.77 (where HRQoL score of 0= death; score of 1 is perfect health)[28] (Table 2). This is slightly lower than a UK general population where the mean score was 0.825, although HRQoL is associated with a range of demographic factors, including age, sex and social class.[30] Furthermore, reductions in HRQoL score may not simply be a function of health compromises from HCV infection, but due to the patient's awareness of having a serious disease.[31] A diagnosis of HCV infection alone can affect patient quality of life.[29] Studies have also investigated the impact of HCV infection amongst haemophiliac patients. In a Dutch cross sectional study of registered haemophiliac patients, patients with HCV infection demonstrated a decrease in HRQoL domains of general health and vitality compared with non-infected haemophiliacs.[32]

The impact of HCV infection on employment and absenteeism remains unclear. Although there are no published UK data, one large US study (n=339,456) which compared absenteeism between employees with HCV infection (but not stratified by stage of infection), reported that HCV-infected workers had 4.15 more days of absence per employee per year than those without HCV infection.[33] This suggests that some loss of productivity does occur with HCV infection but that it is not clear at what stage of disease this would become significant.

b. Moderate and severe chronic hepatitis C

Moderate chronic hepatitis C infection is characterised by portal and periportal fibrosis, while severe chronic HCV infection is a pre-cirrhotic stage with histological evidence of binding fibrosis and incomplete regenerative nodules.[24]

Studies have demonstrated an association between impairments in HRQoL in patients with HCV and severity of liver disease.[34] In one trial of combination therapy, over 60% of patients with moderate disease (prior to therapy) reported problems with either pain and discomfort or anxiety and depression, and the HRQoL associated with moderate disease was 0.66 (Table 2).[28] In studies amongst haemophiliacs, patients with HCV infection demonstrated a decrease in HRQoL domains of general health and vitality compared with non-infected haemophiliacs.[32]

Table 1: Hepatic manifestations of HCV infection

STAGES OF CHRONIC HEPATITIS C INFECTION	DESCRIPTION
Presence of HCV RNA persisting for more than 6 months.	
A. **Mild**[24]	A. **Mild**: asymptomatic HCV carriers with persistently normal or nearly normal ALT **OR** HCV carriers showing minimal/mild liver histological lesions (no/minimal fibrosis) independent of ALT profiles
B. **Moderate**[24]	B. **Moderate**: evidence of portal and peri-portal fibrosis
C. **Severe (pre-cirrhotic)**[24]	C. **Severe**: Pre-cirrhotic stage with bridging fibrosis and incomplete regenerative nodules
Compensated cirrhosis	Defined histologically as a diffuse hepatic process characterized by fibrosis and the conversion of normal liver architecture into structurally abnormal nodules. Modified HAI (Ishak) score = 6
Decompensated cirrhosis	Functional deterioration of the liver. Evidence of cirrhosis with development of any of the following complications: • Variceal haemorrhage • Ascites **OR** • Encephalopathy
Malignancy associated with hepatitis C infection	Hepatocellular carcinoma
Liver transplant	Transplant as a result of decompensated cirrhosis / HCC from HCV

Table 2: Mean HRQoL for each disease stage

Mild disease 0.77	**Treatment for mild disease** 0.65	**SVR after mild disease** 0.82
Moderate disease 0.66	**Treatment for moderate disease** 0.55	**SVR after moderate disease** 0.72
Cirrhosis 0.55	**Decompensated cirrhosis** 0.45	
Hepatocellular carcinoma 0.45	**Post liver transplant** 0.67	

(Source: Wright *et al*. 2006[28])

c. Cirrhosis

The progression to cirrhosis is often clinically silent and some patients are not known to have hepatitis C until they present with the complications of end stage liver disease or hepatocellular carcinoma (HCC). Although studies have demonstrated reduced quality of life measures in patients with HCV infection (Table 2), the impact of cirrhosis on quality of life (QoL) is not straightforward. In a study to evaluate the quality of life in cirrhotic and non-cirrhotic patients (n=271), no significant association was found between mean utility and disease stage.[35] Changes in HCV disease stage appeared to explain only small changes in QoL and with factors such as underlying co-morbidities, income and marital status having a greater effect on QoL than disease stage.[35]

The morbidity and mortality associated with severe liver fibrosis can be severe. Of patients with cirrhosis, approximately 75% remain stable and do not develop decompensation during 5 years or longer.[36] Approximately 80% of patients with stable cirrhosis and no previous episodes of decompensation will survive the next 10 years.[37] Conversely after a patient with chronic HCV infection develops a major complication of cirrhosis, their survival significantly declines: 50% after 5 years and 30% during the next 10 years.[36] The rate at which patients with stable cirrhosis develop complications is approximately 3-5% per year.[37] In a UK study of 150 HCV-infected patients with severe liver fibrosis, 25% of the 131 patients with no prior history of decompensation died or were transplanted, when assessed after a median interval of 42 months.[38] The

probability of survival without liver transplantation was 97%, 88% and 78% at 1, 3 and 5 years, respectively. In non-UK studies, mortality amongst individuals with compensated cirrhosis has been reported at 9% during a mean follow-up of 5 years (with HCC and liver failure being the main causes of death).[36] The probability of survival after diagnosis of compensated cirrhosis was 96%, 91% and 79% at 3, 5 and 10 years respectively.[36]

d. Decompensated cirrhosis

Studies have estimated that the annual incidence of developing decompensated cirrhosis is 3.9% during the first 5 years.[36] The features of decompensated cirrhosis include the development of ascites, upper gastrointestinal bleeding secondary to varices or portal hypertensive gastropathy, hepatorenal syndrome and hepatic encephalopathy.[5] In patients with HCV-induced cirrhosis, decompensation (functional deterioration of the liver) or liver cancer occurs at a rate of approximately 5-6% per year. The 5 year mortality following decompensation has been reported at 13%.[36] HRQoL for decompensated cirrhosis is significant and has been reported at 0.45 (Table 2).[28]

e. Hepatocellular carcinoma

Hepatocellular carcinoma (HCC) is a major complication of chronic HCV infection. However, unlike individuals with chronic hepatitis B infection, HCC appears to almost always develop in HCV-infected patients with cirrhosis.[39] In a European study of 384 patients with compensated cirrhosis due to HCV (different routes of transmission), the 5 year risk of HCC was 7% and the annual incidence was 1.4%.[36] During a 5 year follow-up of HCV-infected cirrhotic patients, complications relating to HCC accounted for 33% of deaths.[36] Genetic factors, alcohol consumption and gender are known to influence the risk of developing HCC. The impact of HCC on quality of life is very significant with the mean HRQoL score estimated at 0.45 (Table 2).[28]

f. Liver transplant

Patients transplanted for HCV have survival rates of 80% and 70% at 1 and 5 years, respectively.[40] Re-infection in the graft occurs in nearly all patients.[41] Re-infection with HCV then leads to cirrhosis in 25-33% of patients in 5 years, and 1-5% develop rapidly progressive fibrosing cholestatic hepatitis leading to hepatic failure in 1-2 years.[42]

4. ANTI-VIRAL THERAPY FOR CHRONIC HEPATITIS C

Drug therapy for hepatitis C has been the subject of technology appraisals by NICE and involves a 6 or 12 month course of therapy with a combination of pegylated interferon and ribavirin.[28,43] Combination anti-viral therapy (pegylated interferon-alpha and ribavirin) is now recommended for individuals with mild, moderate and severe chronic hepatitis C. A complete (sustained) virological response (SVR) is defined as the sustained loss of HCV RNA with normalisation of transaminase values, 6 months after discontinuing treatment.[44]

Therapy for chronic HCV infection eliminates the infection in the majority of individuals and viral elimination is associated with reduced disease progression and a marked reduction in the incidence of disease sequelae. For patients infected with either genotype 2 or 3, response rates after a 24 week course of therapy approach 80%, although there may be some differences.[45] For patients with genotype 1 infection sustained virological response occurs in up to 50% after a 48 week course of therapy. Sustained viral response rates are also affected by a range of factors including age, viral load, certain host genetic polymorphisms and co-infections. [40,46,47,48,49]

The APRICOT and RIBAVIC studies investigated the effects of interferon and ribavirin in HIV co-infected patients. In those studies the highest SVR rate was 62%, lower than published rates of SVR for monoinfected individuals.[50,51] The use of low doses of ribavirin has been identified as a contributing factor to lower rates of SVR, due to the susceptibility of patients with HIV to haemolytic anaemia, a side-effect associated with ribavirin use. However, rates of SVR published are generally lower in HIV co-infected patients treated for HCV even when full doses of ribavirin are used, rather than reduced doses.[52]

Response rates are higher in patients with mild and moderate disease than in those with cirrhosis. Patients with compensated cirrhosis have SVR rates of 41-43% for pegylated interferon-ribavirin compared to 51% for those without fibrosis.[53,54] There is also evidence that combination therapy significantly reduces the rate of fibrosis progression in patients with chronic hepatitis C and in some cases, reverses the degree of fibrosis.[41] There is evidence that interferon treatment reduces the incidence of HCC in treated patients, particularly those who showed an SVR.[41,55,56] Antiviral therapy can also be used to prevent re-infection in transplanted patients.[42]

Although therapy with interferon-alpha is generally well tolerated, studies report approximately 10% of patients fail to complete a therapy course.[57,58] Symptoms such as depression, myalgia, lethargy, influenza-type symptoms and biochemical and haematological abnormalities are common on treatment and account for much of the drop-out in the trials. Symptoms are more frequently reported in patients with cirrhosis, and very poorly tolerated in those with decompensated cirrhosis. Neutropaenia and thrombocytopaenia are more common than in non-cirrhotic patients, particularly with pegylated interferon regimes.[40]

Studies assessing the impact of treatment with interferon-alpha on HRQoL show that following successful treatment patients have significant improvement in their total HRQoL score and in individual categories including work and sleep.[59,60] A fall in HRQoL while on treatment followed by return to baseline after cessation (and improvement in those who achieve an SVR) is well-documented,[60] although persistent impairments in QoL despite viral clearance have been reported.[61] In the 2006 Health Technology Assessment of combination therapy for mild chronic HCV infection, the HRQoL score during treatment for mild disease fell to 0.65 but increased to 0.82 in individuals who achieved an SVR following therapy for mild disease (Table 2). There did not appear to be any gains in HRQoL following treatment for those who did not have an SVR. A similar pattern was observed for those with moderate disease; reported HRQoL for patients with moderate disease on treatment is lower (0.55) than baseline values but increased above pre-treatment levels (0.72) in individuals with an SVR.[28] Studies have also shown that maintenance pegylated interferon therapy was associated with statistically and clinically significant declines in sexual health that did not rebound after cessation of treatment, despite minimal worsening of fatigue and well-being.[62]

Future therapies for HCV infection (e.g. telaprevir and boceprevir) appear to increase SVR rates and have potential to lower side-effect profiles and lower therapy induced HRQoL impairment.[63,64,65]

5. HIV AND HCV CO-INFECTION

Studies have found that, compared directly to HCV-monoinfected patients, HIV-HCV co-infected patients appear to develop cirrhosis 9-12 years earlier on average.[66,67] In addition, the incidence of cirrhosis in the first 10 years of HCV infection is greatly increased in HIV positive patients.[68] A study of men with haemophilia demonstrated a cumulative risk for liver-related mortality of 6.5% in HIV-HCV co-infection versus

1.4% in HCV monoinfection.[69] Extrapolating to the current rate of progression for co-infected patients needs to be done with some caution, as many of these individuals would not have been on optimal fully suppressive highly active antiretroviral therapy (HAART) for a considerable length of time.

The effect of antiretroviral therapy on the natural history of HCV infection has been investigated by a number of clinical groups. Some studies found no association between the use of antiretroviral therapy and the progression of liver fibrosis[70,71] with HAART not fully correcting the adverse effect of HIV infection on HCV prognosis.[72] In other studies, there is evidence that HAART slows the rate of fibrosis progression and reduces long-term liver-related mortality in those co-infected with HIV and HCV.[73,74,75] Brau *et al* additionally reported that the fibrosis progression rate in co-infected patients with undetectable HIV RNA through HAART was similar to HCV-monoinfected individuals.[75]

6. EXTRA-HEPATIC MANIFESTATIONS

It is increasingly clear that chronic HCV infection may have an impact on patients beyond liver damage. These extra-hepatic manifestations can involve multiple organ systems, including renal, dermatological, haematological and rheumatological systems. Approximately 1-2% of HCV-infected individuals will develop extra-hepatic manifestations.[5] Based on available data in 2007, one review determined that the only clearly linked extra-hepatic condition was mixed cryoglobulinaemia. The authors concluded that the link between the virus and many other extra-hepatic manifestations needed further confirmation (Table 3).

Cryoglobulins are found in 50% of patients with chronic HCV infection.[5] Only 25-30% of HCV patients with mixed cryoglobulinaemia develop clinical symptoms, ranging from fatigue, skin rashes, purpura, arthralgias, Raynaud's phenomenon, vasculitis, renal disease and peripheral neuropathy.[76] The clinical manifestations are thought to be caused by immune complex deposition in various organs. Severe symptoms from cryoglobulinaemia appear to respond to interferon treatment, but relapse can occur once treatment is discontinued.[77]

The existence of an association between HCV infection and B-cell non-Hodgkin's Lymphoma (NHL) has been a matter of debate.[78] A statistically significant association between NHL and HCV infection in Italian subjects was initially reported[79] and then subsequently confirmed

by a number of national and international studies.[80,81] However this was not consistent with findings from Northern European and North American data which may reflect a geographic variation in prevalence.[82,83]

A strong association between the sporadic form of Porphyria cutanea tarda (PCT) and HCV was suggested by the high prevalence (>50%) of HCV markers in these patients, mainly in studies from Southern Europe.[84] However, in HCV-positive patients without PCT, no significant alteration in porphyrin metabolism was shown, suggesting an indirect role of infection, probably acting as a triggering factor in genetically predisposed individuals.[85]

In addition to these more specific clinical syndromes, chronic HCV infection has also been associated with more common conditions, where attribution of an individual's condition to the infection is more difficult. In several studies, a high prevalence of diabetes mellitus type 2 has been observed in patients with chronic HCV infection.[86,87,88] Another commonly reported association was with impaired cognitive function; this latter association is supported by evidence of CNS involvement.[89,90,91, 92]

Improvements in some extra-hepatic manifestations including cognitive function and insulin resistance have been demonstrated with anti-viral therapy.[9394]

Table 3: Classification of extra-hepatic manifestations of HCV infection

A. Association on the basis of high prevalence and pathogenesis
Mixed cryoglobulinaemia
B. Association defined on the basis of higher prevalence than controls
B-cell Non Hodgkins Lymphoma
Monoclonal gammopathies
Porphyria cutanea tarda
Lichen planus
C. Association to be confirmed/characterised
Autoimmune thyroiditis
Thyroid cancer

Sicca syndrome
Alveolitis – lung fibrosis
Diabetes mellitus type 2
Non-cryoglobulinaemic nephropathies
Aortic atherosclerosis
Impaired cognitive function

(Adapted from: Zignego et al. 2007[78])

7. SUMMARY AND RECOMMENDATIONS

Hepatitis C is an important cause of chronic liver disease in England. It is a disease characterised by onset that is largely silent due to the paucity of clinical symptoms during the acute infection. The majority of individuals who develop acute HCV infection will progress to chronic infection (detectable HCV RNA for more than 6 months). Given the lack of morbidity associated with acute infection, individuals who are acutely infected and clear infection within six months should not normally require hardship payments.

For those who progress to chronic infection, which is associated with demonstrable reductions in quality of life, a hardship payment is warranted. Mild, moderate and severe chronic HCV infection are all associated with a range of non-specific symptoms and some loss in quality of life, although the latter is not clearly linked to stage of liver disease. In addition, chronic infection has been associated with a range of extra-hepatic symptoms including neurocognitive effects that impact on daily life. Prior to the development of cirrhosis, current therapy is able to achieve sustained virological response (effective viral clearance) in the majority of recipients, although therapy itself is associated with a range of side-effects. Following successful treatment, the prognosis for disease progression and quality of life largely improves. Although a range of extra-hepatic manifestations have been associated with chronic HCV, many of these are difficult to attribute to HCV infection in an individual patient, and many more specific syndromes should subside with effective treatment. Within the next few years more successful and better tolerated therapies are likely to become available. The hardship payment for individuals in this stage is designed to take account of the range of symptoms caused by HCV infection, or the treatment of HCV infection, including specific and non-specific symptoms (such as depression and

fatigue), as well as the risk of extra-hepatic manifestations (such as diabetes).

The progression to cirrhosis, decompensated cirrhosis, or hepatocellular carcinoma will have a substantial impact on life expectancy. Quality of life is also substantially reduced and liable to deteriorate over time. Current anti-viral therapies are poorly tolerated and have a low chance of achieving viral response in this patient group. Even if an SVR can be achieved in cirrhotic patients, liver fibrosis is not completely reversed and the risk of decompensation or of developing liver cancer is retained. Some patients will be eligible for liver transplantation, but this in itself involves considerable morbidity and re-infection occurs in nearly all patients. These individuals therefore would warrant an ongoing payment to reflect the long term hardship encountered during these later stages of chronic HCV infection. Patients who develop life threatening hepatitis C-related tumours, specifically B-cell non-Hodgkin's lymphoma, will experience ongoing hardship of a similar level to those who develop cirrhosis and severe liver disease and should also be eligible for this on-going payment.

Individuals who acquired both HIV and HCV infection from blood or blood products will continue to be in receipt of separate hardship payments for their HIV infection. Co-infection with HIV can increase the rate of progression to chronic HCV infection and cirrhosis. The advent of more effective antiretroviral therapy has improved the quality of life of individuals with HIV, and is likely to markedly improve the prognosis for their HCV infection. It seems reasonable, therefore, that the criteria for and level of HCV related-payment for these individuals should be the same as for HIV negative individuals and that the separate hardship payment for HIV should not be affected.

MEMBERSHIP OF EXPERT WORKING GROUP

Advisory Group on Hepatitis
Professor Maggie Bassendine
Professor Graham Foster
Dr Peter Moss

Expert Advisory Group on AIDS
Professor Brian Gazzard – Chair
Dr Chris Conlon
Dr Keith Radcliffe

UK Haemophilia Centre Doctors' Organisation
Dr Charles Hay
Dr Mike Makris

Health Protection Agency
Dr Mary Ramsay
Dr Gayatri Manikkavasagan

The Hepatitis C Trust
Mr Charles Gore

REFERENCES

1. Health Protection Agency. Hepatitis C 2008 Annual Report.
2. Alberti A, Chemello L, Benvegnu L. Natural History of hepatitis C. Journal of Hepatology 1999; 31 (Suppl 1):17-24
3. Hwang SJ, Lee SD, Lu RH et al. Hepatitis C viral genotype incluences the clinical outcome of patients with acute post transfusion hepatitis C. J Med Virol 2001; 65: 505-9.
4. McCaughan GW, et al. Clinical assessment and incidence of hepatitis C RNA in 50 consecutive RIBA-positive volunteer blood donors. Med J Aust, 1992; 157(4): 231-3.
5. Chen SL, Morgan TR. The Natural History of Hepatitis C Virus (HCV) Infection. Int J Med Sci 2006; 3(2):47-52
6. Tremolada F, Csarin C, Alberti A et al. Long-term follow-up of NANB (type C) post-transfusion hepatitis. J Hepatol 1992; 16:273-281
7. Micallef JM, Kaldor JM, Dore GJ, Spontaneous Viral Clearance Following Acute Hepatitis C Infection: A Systematic Review of Longitudinal Studies. J Viral Hepat. 2006;13(1):34-41.
8. Jaeckel E, Cornberg M, Wedemeyer H et al. Treatment of acute hepatitis C with interferon alpha-2b. New Eng J Med 2001; 345 (20): 1452-1457.
9. Kamal SM. Acute hepatitis C: a systematic review. Am J Gastroenterol. 2008 May;103(5):1283-97.
10. Harris HE, Eldridge KP, Harbour S, et al; HCV National Register Steering Group. Does the clinical outcome of hepatitis C infection vary with the infecting hepatitis C virus type? J Viral Hepat. 2007 Mar;14(3):213-20.
11. Seeff LB, Buskell-Bales Z, Wright EC et al. Long term mortality after transfusion associated NANB hepatitis. N Engl J Med 1992; 327: 1906-1911.
12. Seeff LB, Hollinger B, Alter AJ et al. Long-term morbidity of post-transfusion hepatitis C. Hepatology 1998; 28:407A.
13. Pagliaro L, Peri V, Linea C et al. Natural history of chronic hepatitis C: a systematic review. It J Gastroenterol 1999; 31(1): 28-44
14. Seeff L. The history of the "natural history" of hepatitis C (1968-2009). Liver International 2009; 29(s1):89-99.
15. Kenny-Walsh E. Clinical outcomes after hepatitis C infection from contaminated anti-D immune globulin. Irish Hepatology Research Group. N Engl J Med 1999; 340: 1228-1233.
16. Levine RA, Sanderson SO, Ploutz-Snyder R, et al. Assessment of fibrosis progression in untreated Irish women with chronic hepatitis C contracted from immunoglobulin anti-D. Clin Gatroenterol Hepatol 2006; 4:1271-7.

[17]. Posthouwer D, Makris M, Yee TT, et al. Progression to end-stage liver disease in patients with inherited bleeding disorders and hepatitis C: an international, multicenter cohort study. Blood. 2007 May 1;109(9):3667-71

[18]. Seeff LB. Natural history of chronic hepatitis C. Hepatology 2002; 36(5 Suppl 1):S356-46.

[19]. Castells L et al. Long interval between HCV infection and development of hepatocellular carcinoma. Liver 1995; 15(3): 159-163.

[20]. Thein HH, Yi Q, Dore GJ, Krahn MD. Estimation of stage-specific fibrosis progression rates in chronic hepatitis C virus infection: a meta-analysis and meta-regression. *Hepatology. 2008 Aug;48(2):418-31*

[21]. Sweeting MJ, De Angelis D, Neal K et al. Estimated progression rates in the United Kingdom hepatitis C cohorts differed according to method of recruitment. J Clin Epid 2006; 59:144-152.

[22]. Missiha SB, Ostrowski M, Heathcote EJ. Disease progression in chronic hepatitis C: modifiable and non-modifiable factors. Gastroenterology 2008; 341: 1699-1741.

[23]. Poynard T, Yuen MF, Ratziu V, Lai CL. Viral hepatitis C *Lancet 2003; 362: 2095-2100*

[24]. Tong MJ, El-Farra NS, Reikes AR, CoRL. Clinical outcomes after transfusion associated hepatitis C. N Eng J Med 1995; 332:1463-1466.

[25]. Harris HE, Ramsay M, Anderws N, Eldridge KP. Clinical course of hepatitis C virus during the first decade of infection: cohort study. BMJ 2002; 324:1-6.

[26]. Harris HE, Ramsay ME, Andrews NJ. Survival of a national cohort of hepatitis C virus infected patients, 16 years after exposure. Epidemiol Infect 2006; 134:472-477.

[27]. Alberti A, Benvegnu L, Boccato S, et al. Natural history of initially mild chronic hepatitis C. Digestive and Liver Disease 2004. 36: 646-654.

[28]. Wright M, Grieve R, Roberts J, Main, Thomas HC. Health benefits of antiviral therapy for mild chronic hepatitis C: randomised controlled trial and economic evaluation. Health Technology Assessment. 2006; 10:21.

[29]. Rodger AJ, Jolley D, Thompson SC et al. The impact of diagnosis of hepatitis C virus on quality of life. Hepatology 1999; 30:1299-301.

30. Kind P, Dolan P, Gudex C, Williams A. Variations in population health status: results from a United Kingdom national questionnaire survey BMJ 1998; 316 : 736-41.

[31]. Foster GR, Goldin RD, Thomas HC. Chronic hepatitis C virus infection causes a significant reduction in quality of life in the absence of cirrhosis. Hepatology 1998; 27:209-212.

[32]. Posthouwer D, Plug I, van der Born J et al. Hepatitis C and health-related quality of life among patients with haemophilia. Haematologica 2005; 90: 846-850.

[33]. Su J, Brook R, Kleinman N, Corey-Lisle P. The impact of Hepatitis C virus infection on work absence, productivity and healthcare benefit costs. Hepatology 2010; 52(2):436-442.

[34]. Bjornsson E, Verbaan H, Oksanen A et al. Health-related quality of life in patients with different stages of liver disease induced by hepatitis C. Scand J Gastroenterol 2009; 44:878-887.

[35]. Hsu P, Krajden M, Yoshida E et al. Does cirrhosis affect quality of life in hepatitis C virus-infected patients? Liver International 2009; ISSN 1478-3223: 449-458

[36]. Fattovich G, Giuliano G et al. Morbidity and mortality in Compensated Cirrhosis Type C: A retrospective follow-up study of 384 patients. Gastroenterology 1997; 112:463-472.

[37]. Shiffman ML. Natural history and risk factors for progression of hepatitis c virus disease and development of hepatocellular cancer before liver transplantation. Liver Transplantation 2003; 9(11):Suppl 3 S14-S20

[38]. Lawson A, Hagan S, Rye K, et al. The natural history of hepatitis C with severe hepatic fibrosis. Journal of Hepatology 2007; 47: 37-45.

[39]. El-Serag HB. Global epidemiology of hepatocellular carcinoma. Liver Clin N Amer 2001; 5:87-107.

[40]. Wright TL. Treatment of patients with hepatitis C and cirrhosis. Hepatology 2002;36:S185-S194.

[41]. Teo M, Hayes P. Management of hepatitis C. Br Med Bulletin 2004; 70:51-69.

[42]. Shiffman ML, Vargas HE, Everson GT. Controversies in the management of hepatitis C virus infection after liver transplantation. Liver Transplant 2003; 9:1129-1144.

[43]. National Institute for Health and Clinical Excellence (NHS) Interferon alpha (pegylated and non-pegylated) and ribavarin for the treatment of chronic hepatitis C. Technology Appraisal 75. London: NICE. Available on-line at www.nice.org.uk

[44]. Reichard O, Glaumann H, Freyden A et al. Two year biochemical, virological and histological follow-up in patients with chronic hepatitis C responding in a sustained fashion to interferon alpha-2b treatment. Hepatology 1995; 21:918-922

[45]. Mauss S, Hueppe D, John C et al. Estimating the likelihood of sustained virological response in chronic hepatitis C therapy. J Viral Hepatitis 2010; Epub (ahead of print)

[46]. Yoshida H, Shiraori Y, Moriyana M et al. Interferon therapy reduces the risk for hepatocellular carcinoma, national surveillance programme of cirrhotic and non-cirrhotic patients with chronic hepatitis C in Japan. Ann Intern Med 1999; 131:174-181.

[47]. Brau N, Rodriguez-Torres M, Prokupek D. et al. Treatment of chronic hepatitis C in HIV/HCV co-infected with interferon-alpha-2b +full course vs 16 week delayed ribavarin. Hepatology 2004; 39(4):989-998.

48. Mangia A, Thompson AJ, Santoro R et al. An IL28B polymorphism determines treatment response of hepatitis C virus genotype 2 or 3 patients who do not achieve a rapid virologic response. Gastroenterology. 2010 Sep;139(3):821-7, 827.e1. Epub 2010 Jun 2.

49. Thompson AJ, Muir AJ, Sulkowski MS, et al. Interleukin-28B polymorphism improves viral kinetics and is the strongest pretreatment predictor of sustained virologic response in genotype 1 hepatitis C virus. Gastroenterology. 2010 Jul;139(1):120-9.e18. Epub 2010 Apr 24.

[50]. Torriani FJ, Rodriguez-Torres M, Rockstroh M et al. Peginterferon Alfa-2a plus ribavirin for chronic hepatitis C virus infection in HIV-infected patients. New Eng J Med 2004; 351(5):438-450.

[51]. Carrat F, Bani-Sadr F, Pol S, et al; ANRS HCO2 RIBAVIC Study Team. Pegylated interferon alfa-2b vs standard interferon alfa-2b, plus ribavirin, for chronic hepatitis C in HIV-infected patients: a randomized controlled trial. JAMA. 2004; 292(23):2839-48.

[52]. Laguno M, Murillas J, Blanco JL et al. Peginterferon alfa-2b plus ribavirin compared with interferon alfa-2b plus ribavirin for treatment of HIV/HCV co-infected patients. AIDS. 2004 Sep 3;18(13):F27-36

[53]. Fried MW, Shiffman ML, Redd R. et al. Peginterferon alpha-2a plus ribavarin for chronic hepatitis C virus infection. N Eng J Med 2002; 347: 975-982.

[54]. Manns MP, McHutchinson JG, Gordon SC et al. Peginteferon alpha-2b plus ribavarin compared with interferon alpha 2b plus ribavarin for initial treatment of chronic hepatitis C, a randomised trial. Lancet 2001; 358: 958-965.

[55]. Camma C, Giunta M, Andreone P, Craxi A. Interferon and prevention of hepatocellular carcinoma in viral cirrhosis: an evidence based approach. J Hepatol 2001; 34:593-602.

[56]. Papatheodoridis GV, Papadimitropoulos VC, Hadziyannis SJ. Effect of interferon therapy on the development of hepatocellular carcinoma in patients with hepatitis C virus related cirrhosis : a meta-analysis. Alimnet Pharmacol Ther 2001; 15: 689-698.

[57]. McHutchinson JG, Gordon SC, Schiff ER et al. Interferon alpha-2b alone or in combination with ribavarin as initial treatment for chronic hepatitis C. Hepatitis Interventional Therapy Group. N Eng J Med 1998; 339:1485-92.

[58]. Foster GR, Goldin RD, Main J et al. Management of chronic hepatitis C: clinical audit of biopsy based management algorithm. BMJ 1997; 315:453-8.

[59]. Ware JEJ, Bayliss MS, Mannocchia M, Davis GL. Health-related quality of life in chronic hepatitis C: impact of disease and treatment response. The Interventional Therapy Group. Hepatology 1999; 30:550-5.

[60]. Bonkovsky HI, Woolley JM. Reduction of health-related quality of life in chronic hepatitis C and improvement with interferon therapy. The Consensus Interferon Study Group. Hepatology 1999; 29:264-70.

[61]. Tillmann HL, Wiese M, Weigand J, et al. Quality of life in patients with various liver diseases. J Viral Hepatitis 2010; Mar 8 [Epub ahead of print].

[62]. Snow KK, Bonkovsky HL, Fontana RJ et al. Changes in quality of life and sexual health are associated with low dose pegylated interferon therapy and disease progression in patients with chronic hepatitis C. Alimentary Pharmacology and Therapeutics 2010; 31:719-734.

[63]. Kronenberger B, Zeuzem S. Current and Future Treatment Options for HCV. Ann Hepatol 2009 8(2): 103-112.

[64]. Shiffman ML. Treatment of Hepatitis C in 2011: What can we expect? Curr Gastroenterol Rep 2010; 12(1): 70-75.

[65]. Asselah T, Estrabaud E, Bieche I et al. Hepatitis C: viral and host factors associated with non-response to pegylated interferon plus ribavirin. Liver Int. 2010 Jul 14. [Epub ahead of print]

[66]. Benhamou Y, Bochet M, Di Martino V et al. Liver fibrosis progression in human immunodeficiency virus and hepatitis C virus coinfected patients. The Multivirc Group. Hepatology. 1999 Oct;30(4):1054-8.

[67]. Mohsen AH, Easterbrook PJ, Taylor C et al. Impact of human immunodeficiency virus (HIV) infection on the progression of liver fibrosis in hepatitis C virus infected patients. Gut. 2003 Jul;52(7):1035-40.

[68]. Soto B, Sánchez-Quijano A, Rodrigo L et al. Human immunodeficiency virus infection modifies the natural history of chronic parenterally-acquired hepatitis C with an unusually rapid progression to cirrhosis. J Hepatol. 1997 Jan;26(1):1-5.

[69]. Darby SC, Ewart DW, Giangrande PL et al. Mortality from liver cancer and liver disease in haemophilic men and boys in UK given blood products contaminated with hepatitis C. UK Haemophilia Centre Directors' Organisation. Lancet. 1997 Nov 15;350(9089):1425-31.

[70]. Martinez-Sierra C, Arizcorreta A, Díaz F et al. Progression of chronic hepatitis C to liver fibrosis and cirrhosis in patients coinfected with hepatitis C virus and human immunodeficiency virus. Clin Infect Dis. 2003 Feb 15;36(4):491-8.

[71]. Martín-Carbonero L, Benhamou Y, Puoti M, et al. Incidence and predictors of severe liver fibrosis in human immunodeficiency virus-infected patients with chronic hepatitis C: a European collaborative study. Clin Infect Dis. 2004 Jan 1;38(1):128-33.

[72]. Thein HH, Yi Q, Dore GJ, Krahn MD. Natural history of hepatitis C virus infection in HIV-infected individuals and the impact of HIV in the era of highly active anti-retroviral therapy: a meta-analysis. AIDS 2008; 22(15):1979-91.

[73]. Benhamou Y, Di Martino V, Bochet M, et al; MultivirC Group. Factors affecting liver fibrosis in human immunodeficiency virus-and hepatitis C virus-coinfected patients: impact of protease inhibitor therapy. Hepatology. 2001 Aug;34(2):283-7.

[74]. Qurishi N, Kreuzberg C, Lüchters G, et al. Effect of antiretroviral therapy on liver-related mortality in patients with HIV and hepatitis C virus coinfection. Lancet. 2003 Nov 22;362(9397):1708-13.

[75]. Bräu N, Salvatore M, Ríos-Bedoya CF, et al. Slower fibrosis progression in HIV/HCV-coinfected patients with successful HIV suppression using antiretroviral therapy. J Hepatol. 2006 Jan;44(1):47-55.

[76]. Cacoub P. et al. Extrahepatic manifestations associated with hepatitis C virus infection. A prospective multicenter study of 321 patients. The GERMIVIC. Groupe d'Etude et de Recherche en Medecine Interne et Maladies Infectieuses sur le Virus de l'Hepatite C. Medicine (Baltimore) 2000;79(1):47–56.

[77]. Lunel F, Cacoub P. Treatment of autoimmune and extrahepatic manifestations of hepatitis C virus infection. J Hepatol. 1999;31(Suppl 1):210–6.

[78]. Zignego AL, Ferri C, Pileri SA, et al. Extra-hepatic manifestations of Hepatitis C virus infection : A general overview and guidelines for clinical approach. Digestive and Liver Disease 2007; 39:2-17.

[79]. Mazzaro C, Zagonel V, Monfardini S, Tulissi P et al. Hepatitis C virus and non-Hodgkin's lymphomas. Br J Haematol 1996; 94:544-550.

[80]. Zuckerman E, Zuckerman T, Levine AM et al. Hepatitis C virus infection in patients with B cell Non-Hodgkins Lymphoma. Ann Intern Med 1997; 127:423-8.

[81]. Monti G, Pioltelli P et al. Incidence and characteristics of non-Hodkins lymphomas in a multicenter case file of patients with hepatitis C virus related symptomatic mixec cryoglobulinaemia. Arch Intern Med 2005; 165:101-105.

[82]. Collier JD, Zanke B, Moore M, Kessler G, Krajden M, Shepherd F et al. No association between hepatitis C and B-cell lymphoma. Hepatology 1999; 9:1259-1261.

[83]. McColl MD, Singer IO, Tait RC, et al. The role of hepatitis C virus in the aetiology of non-Hodkins lymphoma – a regional variation? Leuk Lymphoma 1997; 26:127-130.

[84]. Ferri C, Baicchi U et al. Hepatitis C virus-related autoimmunity in patients with porphyria cutanea tarda : evidence of a strong association. Hepatology 1992; 16: 1322-6.

[85]. Hussain I, Hepburn NC, JJones A et al. The association of hepatitis C viral infection with Porphyria cutanea tarda in the Lothian region of Scotland. Clin Exp Dermatol 1996; 21: 283-285.

[86]. Negro F, Alaei M. Hepatitis C virus and type 2 diabetes. World J Gatroenterol 2009; 15(13): 1537-1547.

[87]. Lonardo A, Adinolfi LE, Petta S et al. Hepatitis C and diabetes: the inevitable coincidence. Expert Rev Anti Infect Ther.; 2009 7(3):293-308.

[88]. Mehta SH, Brancati FL et al. Prevalence of type 2 diabetes mellitus among persons with hepatitis C virus infection in the United States. Ann Intern Med 2000; 133: 592-9.

[89]. Forton DM, Thomas HC, Murphy CA et al. Hepatitis C and cognitive impairment in a cohort of patients with mild liver disease. Hepatology 2002; 35:433-9.

[90]. Forton DM, Allsop JM, Main J et al. Evidence for a cerebral effect of the hepatitis C virus. Lancet 2001; 358:38-9.

[91]. Forton DM, Taylor-Robinson SD, Thomas HC. Cerebral dysfunction in chronic hepatitis C infection. J Viral Hepatol 2003; 10:81-86.

[92]. Lowry, D, Coughlan B, McCarthy O, Crowe J. Investigating health-related quality of life, mood and neuropsychological test performance in a homogeneous cohort of Irish female hepatitis C patients. Journal of Viral Hepatitis 2010; 17:352-359.

[93]. Jacobson IM, Cacoub P, Dal Maso L, Harrison SA, Younossi ZM. Manifestations of chronic hepatitis C virus infection beyond the liver. Clin Gastroenterol Hepatol 2010; 8(12):1017-1029.

[94]. Conjeevaram HS, Wahed AS, Afdhal N, Howell CD, Everhart JE, Hoofnagle JH. Changes in Insulin sensitivity and Body weight during and after Peginterferon and Ribavarin therapy for hepatitis C. Gastroenterology 2010; [Epub ahead of print].

Annex 5

SUMMARY OF CAMPAIGNERS' REPRESENTATIONS IN RELATION TO SUPPPORT PACKAGES

The following list reflects the representations that have been received from the campaigners since July 2010. It has been collated from the written submissions of the campaigners, comments made in their meetings with the Parliamentary Under Secretary of State for Public Health, Anne Milton MP, and correspondence received by the Department of Health. Any that go beyond the terms of reference of the review have not been considered in the report.

Compensation

- Evidence gathered during the course of the review shows a fairly wide range of views on the level of payments that this patient group should receive, in respect of both HIV and hepatitis C infection:
  - the minimum wage, - c£11k pa gross (£5.75 per hour, 40hr week);
  - lump sum of £100k-£150k, followed by recurrent annual payments of £3,600-£6,000 for Skipton Fund Stage 1 patient, followed by lump sum of £300k for Stage 2 payment;
  - HIV and hepatitis C stage 2 patients to receive £18k pa; hepatitis C stage 1 patients to receive £5-7k pa; widows and orphans eligible for unspecified discretionary payments.
  - a lump sum of £200k-£300k;
  - individual assessment of need – one campaigner estimated this at a lump sum of c£400,000 plus (unspecified) regular payments for each type of infection.
  - individual assessments of loss – two of those affected cited figures of c£500k - c£800k.
  - payments equivalent to those in Ireland – estimated average lump sum of c£750k for an infected individual.

- Compensation on a par with Ireland. Lump sum payment followed by regular payments. Other submissions suggest applicants should have choice on how they receive payments
- Regular payments for those infected with hepatitis C, on a par with those received by HIV patients
- Skipton Fund stage 2 payments are only made when patients are close to death –the trigger for stage 2 payments needs to be improved

- DH should pay interim lump sum payments while the scheme is being set up
- On-going payments should rise in line with the RPI
- Payments should not be means tested
- Payments should be based on individual assessments
- Payments should be made to the widows/dependents of those who died before August 2003
- On-going payments to widows of those who either have died since August 2003, or will die. (NB: they make no distinction about what the patient dies of, i.e the implication is that they do not need to die of hep C to qualify)
- Compensation for carers. Backdated. Some suggest this should be a lump sum
- Payment should be made through DWP
- Payments should not be means tested, or taxable, or taken into account in calculating benefits
- The Macfarlane and Eileen Trusts and Skipton Fund should remain in existence to provide on-going support
- Payments should be made to those who clear the virus. Implicit that this should include those who clear in the acute phase

Treatment/Care

- Free prescriptions
- Free NHS care for all health needs
- Extend patient representation in all health care decision making
- Make home nursing free of charge (is currently charged for, and DLA/Carers allowance does not pay for 24/7 care)
- Priority access to counselling (within 1 week). Or make provision in the financial settlement to cover this cost privately
- Give GPs the ability to apply for additional funding to enable them to meet their patients needs
- Commissioners for Trusts should be able to access additional funds for haemophilia patients
- Put haemophilia treatment and ethics on the curriculum of medical schools

Other

- Government to establish a comprehensive insurance scheme

Annex 6

Life cover for individuals infected with hepatitis C and/or HIV by NHS supplied blood transfusions or blood products

The following tables have been provided by the ABI as an indicative example only that does not purport to represent the industry, considers the availability of life cover for this patient group. It shows that people with haemophilia who are not infected with HIV and/or hepatitis C are likely to have access to life cover albeit with increased premium loadings of up to 300% for those with severe haemophilia. Those who are additionally infected with hepatitis C are likely to subject to an additional premium loading that might be within the range of 75% to 250%. People who are infected with HIV may be able to obtain life insurance, for example for a period of 10 years, up to the age of 60, with an additional premium that might be within the range of £3-10 per £1000 of sum assured. Life cover will only be available if the hepatitis C and/or HIV have been successfully treated/controlled with drugs, and will invariably not be available to those who are co-infected.

Life cover for Non Haemophilia patients **Indicative example that does not represent an industry standard. Customers need to confirm details with own insurer.**		
HIV	**Hepatitis C**	**HIV + Hep C**
The fact that the disease was acquired following treatment with contaminated blood will have no effect on whether terms will be offered. Life cover may be available if the HIV is controlled by antiviral medication (HAART) and certain other eligibility criteria are met for example: undetectable viral load, good CD4 count, good compliance with treatment and no significant co-morbidities. In this case, life cover may be offered for no more than 10 years with a maximum expiry age of 60 with a premium loading. Example: Additional premium of £3 per £1000 of sum assured to additional £10 per £1000 of sum assured.	If there are indications of continued chronic infection, changes on biopsy or raised enzyme levels the loadings for Hepatitis C would be in the region of: Under age 30 Additional +200% to+250% Age 30-40 Additional +100% to +200% Age 40 to 50 Additional +75% to +150% Over age 50 Additional + 75% to +100% If there has been effective treatment with a sustained viral clearance and normal biopsy and enzymes the loadings will be +50% to +100%	Life cover unlikely to be available

Life cover for Haemophilia Patients **Indicative example that does not represent an industry standard. Customers need to confirm details with own insurer.**			
Haemophilia	**Haemophilia +** **Hepatitis C**	**Haemophilia +** **HIV**	**Haemophilia +** **HIV & Hepatitis C**
Mild haemophilia (Factor VIII concentration over 15%): • Cover available with no exclusions • Premium loading – range from 0% to +75% **Moderate** haemophilia (Factor VIII between 5-15%) • Cover available with no exclusions • Premium loading – range from +50% to +200% **Marked** haemophilia (Factor VIII between 1-5%): • Cover available with no exclusions • Premium loading – range from +100% to +300%	Life cover is unlikely to be available unless the Hepatitis C has been treated effectively with interferon. If there are no indications of chronic liver changes then life cover may be offered in the same ranges as shown for haemophilia alone. If there are indications of continued chronic infection, changes on biopsy or raised enzyme levels, than an extra loading for Hepatitis C would be imposed in addition to the loading for haemophilia. Additional loadings for Hepatitis C would be in the region of: Under age 30 Additional +200% to+250% Age 30-40 Additional +100% to +200% Age 40 to 50 Additional +75% to +150% Over age 50 Additional + 75% to +100% Example: 35yr old with moderate haemophilia and Hepatitis C – premium loading +150% to + 400%	Life cover is unlikely to be available unless the HIV is controlled by antiviral medication and the haemophilia is mild. In this case, life cover may be available for no more than 10 years with a maximum expiry age of 60 with a premium loading. Example: Additional premium of £3 per £1000 of sum assured to additional £10 per £1000 of sum assured.	Life cover unlikely to be available

The additional premium will be specific to each application.

Indicative example that does not represent an industry standard. Customers need to confirm details with their own insurer:

Male / age next birthday 35 years / sum assured £100,000 / non-smoker / term 20 years/ level term assurance	
Monthly premiums e.g. Basic premium = £9.01	Premium loading for marked haemophilia e.g. Rated premium = £14.88 Premium loading for mild haemophilia and Chronic Hepatitis C e.g. Rated premium = £21.06 Premium loading for mild haemophilia with HIV e.g. Rated premium = £70.80 with maximum term 10 years

Insurers

This is a new market – only recently has robust data from longitudinal studies been available for insurers to assess the risk.