A review of hepatitis C virus (HCV) vertical transmission: risks of transmission to infants born to mothers with and without HCV viraemia or human immunodeficiency virus infection

SL Thomas, a M-L Newell, a CS Peckham, a AE Ades a and AJ Hall b

Background Hepatitis C virus (HCV) vertical transmission studies have reported conflicting findings, possibly due to differences in HCV transmission risk factors among maternal populations, or to methodological differences.

Methods Systematic review of worldwide published and unpublished HCV vertical transmission studies. Standardized diagnostic criteria were applied to minimize methodological differences, and transmission rates recalculated according to maternal HCV viraemic and human immunodeficiency virus (HIV) infection status.

Results In all, 976 eligible infants from 28 studies were followed up sufficiently for recalculation of transmission rates. Overall transmission rates were less than 10% in 8/12 studies of HIV negative mothers, compared with 2/7 studies comprising at least 50% HIV-coinfected mothers. Rates from 409 viraemic mothers in 15 studies ranged from 0% to 41%, being less than 10% from HIV negative mothers in 6/13 studies and from HIV positive mothers in 1/6 studies. Nine studies measured maternal viraemia levels, with only 2/30 transmitting mothers having <10^6 copies/ml of HCV RNA. Eight transmissions were identified overall from non-viraemic mothers. Significant transmission rate variation remained after accounting for maternal viraemia and HIV coinfection, possibly due to differences in other vertical transmission risk factors, in frequencies of postnatal transmission, or residual differences in study methodologies.

Conclusions Overall, HCV transmission is largely restricted to infants born to HCV viraemic mothers, and low risks among most HIV negative mothers may be due to lower HCV viraemia levels. International agreement on standardized diagnostic criteria for HCV vertical transmission would facilitate pooling of individual findings, to allow more precise transmission estimates and further investigation of risk factors.

Keywords Hepatitis C virus, vertical transmission, review, HIV

Accepted 13 June 1997
Concern about the public health importance of HCV has led to debate about the desirability of introducing HCV screening for antenatal or other populations, but further information is needed on the frequency of HCV vertical transmission before widespread HCV antenatal testing is initiated. A systematic review of worldwide published and unpublished research on HCV vertical transmission was therefore carried out to clarify the risks of vertical transmission from mothers with or without HCV viraemia or HIV infection and to highlight issues which might explain differences in transmission rates.

Methods

Data identification

Articles published between January 1990 and July 1996 were identified by computerized literature searches of Medline and Embase, using combinations of key and free text terms. No language restrictions were employed. Thirty journal titles were hand searched to identify recently published or unlisted articles, and references from all articles, reviews and relevant books were checked. Conference abstracts were identified by searching the British Library’s Boston Spa Conferences database and the Index of Scientific and Technical Proceedings, supplemented by hand searching printed proceedings from relevant meetings. Unpublished information was sought by sending structured questionnaires to published authors, to six European antenatal centres previously identified as screening pregnant women for HCV infection, and to four other centres known to be undertaking studies. Researchers were asked for unpublished details of previously published work, for updated or new findings, for clarification of duplicated data, and whether they were aware of any other unpublished research.

Data extraction/exclusion

Information extracted from each study included the numbers of mothers and infants studied, the diagnostic criteria used for maternal and infant HCV infection, maternal HCV viraemic status and HIV serostatus, duration and frequency of infant follow-up, and infant HCV RNA polymerase chain reaction (PCR) and antibody test results. Data excluded from further analyses comprised case reports, data duplicated in updated reports or collaborative studies, PCR test results on cord blood or on unspecified samples taken at birth, and mother-infant pairs with possible misclassified HCV infection status. The latter comprised individuals diagnosed solely by first generation, unsupplemented second generation or unspecified antibody assays, and mothers whose infection status was determined after delivery. Studies with some ineligible mother-infant pairs were included if enough information was available to recalculate transmission rates to remaining infants.

Data analysis

Standardized diagnostic criteria were developed to reduce study heterogeneity due to the variety of definitions used for infant HCV infection and non-infection. Existing data on patterns of HCV infection markers in vertically exposed infants were analysed. Infants were then categorized as infected, uninfected, probably uninfected or of indeterminate status. Infected infants (\( n_I \)) were defined as any of the following: PCR positive on two or more separate occasions; antibody positive at 18 months or later; evidence of seroconversion (seroconversion followed by renewed seropositivity, or change in RIBA reactivity). Uninfected infants (\( n_U \)) were persistently PCR negative, but negative results before 6 months were considered insufficient to classify infants as uninfected. If there was no evidence of seroconversion, two negative PCR results at least 6 months apart (to exclude intermittent viraemia) from the age of 6 months were required: if antibody tests were used, seroconversion or loss of RIBA-reactivity by 18 months, and if seroconversion occurred before 12 months confirmation of antibody negativity at least 3 months later or at least one PCR negative result from 6 months was also required. Probably uninfected infants (\( n_P \)) had a single PCR negative result from the age of 6 months without evidence of seroconversion, or two negative results less than 6 months apart without evidence of seroconversion, or had no PCR results and seroverted before 12 months without a confirmatory negative sample 3 months later. Infants with indeterminate status (\( n_O \)) had a single PCR positive result at any stage of follow-up (as false positive results may be common), or were less than 18 months old with no evidence of seroconversion and either no PCR results or negative PCR test results before 6 months only. Transmission rates were recalculated for studies with sufficient information, obtaining up to three estimates. The best estimate was calculated from the number of infected, uninfected and probably uninfected infants, as \( \frac{n_I + n_U + n_P}{n_I + n_U + n_P + n_O} \). Minimum and maximum estimates were calculated by considering infants with indeterminate or probable infection status to be all uninfected for the minimum estimate, \( \frac{n_I}{n_I + n_U + n_P + n_O} \), or all infected for the maximum estimate, \( \frac{n_I + n_U + n_O}{n_I + n_U + n_P + n_O} \).

Consideration was also given to duration of infant follow-up. Exclusion from analyses of infants followed up for short periods may limit any over- or underestimation of transmission rates resulting from earlier identification of infected infants (in studies using PCR) or uninfected infants (in studies using antibody assays). However, use of a minimum follow-up period may introduce bias if follow up in any study related to HCV infection risk, for example if uninfected infants were followed only until seroconversion, but infants born to HIV-coinfected mothers (who may be more likely to transmit HCV) were followed more intensely. Studies with sufficient information were therefore categorized into three groups. Group One comprised studies with follow-up protocols of at least 12 months, and mostly reported findings only for infants followed up for this minimum period; transmission rates were calculated after excluding infants with shorter follow-up. Group Two comprised studies where the follow-up protocol was unclear, and some infants were followed for less than 12 months. Where PCR was used, analyses were restricted to infants followed for at least 6 months, to minimize potential biases. Where antibody assays were used alone, minimum follow-up of 18 months was used, but consideration was given to findings for infants with shorter follow-up. Longer-term risks of persistent infection were estimated in Group Three studies, which initiated follow-up of most infants after the first 12 months.

Exact 95% confidence intervals (CI) for vertical transmission rates were derived using the binomial distribution. Transmission rates were calculated separately according to maternal HCV viraemic status and HIV serostatus for those studies with available information and a total of 10 or more infants, and differences
Table 1: Hepatitis C (HCV) vertical transmission rates restricted to mother-infant pairs with minimum 12-month follow-up (Group One studies)

<table>
<thead>
<tr>
<th>Author</th>
<th>Mothers</th>
<th>Infants &gt;12m</th>
<th>Vertical transmission % (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>% PCR+ /ive</td>
<td>% HIV+ /ive</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Di Domenico</td>
<td>25</td>
<td>40%</td>
<td>0%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Giacchino</td>
<td>31</td>
<td>61%</td>
<td>0%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ruiz-Extegerma</td>
<td>31</td>
<td>65%</td>
<td>0%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pipan</td>
<td>24c</td>
<td>75%</td>
<td>0%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sabatino</td>
<td>30</td>
<td>33%</td>
<td>0%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chen</td>
<td>28</td>
<td>100%</td>
<td>0%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Resti</td>
<td>22</td>
<td>54%</td>
<td>0%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fischler</td>
<td>55</td>
<td>73-75% (40/54)</td>
<td>4%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Zanettl</td>
<td>116</td>
<td>55%</td>
<td>19%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Manzini</td>
<td>43</td>
<td>63%</td>
<td>37%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Zuccotti</td>
<td>37</td>
<td>57%</td>
<td>54%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Macabruni</td>
<td>44c</td>
<td>100%</td>
<td>73%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quinti</td>
<td>68</td>
<td>16-72% (11/30)</td>
<td>100%</td>
</tr>
</tbody>
</table>

a Recalculated using standardized criteria (see text).
b Polymerase chain reaction
c Human immunodeficiency virus.
d Numbers of HCV infected / uninfected / probably uninfected / indeterminate infants (see text).
e Restricted to mothers conforming to inclusion criteria (see text).
f Not all mothers tested.
g Assumes two excluded mothers had one infant each

in these rates within individual studies were assessed using Yates’ corrected χ² and Fischer’s exact tests. Heterogeneity in study findings was assessed with exact likelihood ratio tests and Monte Carlo inference, using the StatXact software package (1989, 1991, Cytel Software Corporation).

References to unpublished data
Unpublished data are referenced in the text as ‘up’ and a number which corresponds to that in the list of contributors (see Acknowledgements).

Results
A total of 116 published and unpublished reports were identified. Data were clarified or updated for 24 previously published studies or conference abstracts;sup1,2,4-10,12-16,18-20,23 (including one updated summarysup1 which replaced five earlier reports of ongoing research20-24), and newly provided for three studiesup3,11,17 A total of 65 studies were excluded from transmission risk analyses because of one or more exclusion criterion (Table available on request). Numbers in the 51 remaining studies ranged from 4 to 116 infants, with reported transmission rates of 0-100%. In 18 studies there was insufficient detail on individual mothers or infants to recalculate transmission rates of 0-100%. In 18 studies there was insufficient detail on individual mothers or infants to recalculate transmission rates (Table available on request).4,25-38sup9-20 Standardized diagnostic criteria were applied to 33 studies, four of which followed less than two eligible infants for the minimum period.19,42 and one of which used an unspecified ELISA assay for infants who were PCR tested at 3 months only.43 The remaining 28 studies comprised 13 Group One studies,3,11,36,44-53sup1-4,12 Group Two studies,54-65sup5-7 and three Group Three studies.12,66,67sup8 (a total of 976 eligible infants sufficiently followed up).

Overall transmission rates
Table 1 and Table 2 summarize Group One and Group Two studies, with details of maternal HIV infection and HCV
Table 2 Hepatitis C virus (HCV) vertical transmission rates restricted to mother-infant pairs with minimum 6 month follow-up (using polymerase chain reaction [PCR]) or 18-month follow-up (using antibody assays alone) (Group Two studies)

<table>
<thead>
<tr>
<th>Author*</th>
<th>Mothers</th>
<th>Infants &gt;6m</th>
<th>Vertical transmission % (95% CI)*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>% PCR+ive</td>
<td>% HIV+ive</td>
<td>N</td>
</tr>
<tr>
<td><strong>Using PCR</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Uehara</td>
<td>7</td>
<td>100</td>
<td>N/D</td>
</tr>
<tr>
<td>Kojima</td>
<td>18</td>
<td>100</td>
<td>N/D</td>
</tr>
<tr>
<td>Roudot-Thoraval</td>
<td>12</td>
<td>67</td>
<td>0</td>
</tr>
<tr>
<td>Matsubara</td>
<td>23</td>
<td>83</td>
<td>0</td>
</tr>
<tr>
<td>Wejstal</td>
<td>14</td>
<td>100</td>
<td>0</td>
</tr>
<tr>
<td>Moriya</td>
<td>84</td>
<td>100</td>
<td>0</td>
</tr>
<tr>
<td>Koseki</td>
<td>14</td>
<td>100</td>
<td>7</td>
</tr>
<tr>
<td>Kudesia</td>
<td>12</td>
<td>N/D</td>
<td>8</td>
</tr>
<tr>
<td>Thaler</td>
<td>8</td>
<td>100</td>
<td>38</td>
</tr>
<tr>
<td>Papaevangelou</td>
<td>35</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Novati</td>
<td>8</td>
<td>63</td>
<td>100</td>
</tr>
<tr>
<td><strong>Using antibody assays only</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Buffolano</td>
<td>29</td>
<td>N/D</td>
<td>3</td>
</tr>
</tbody>
</table>

* Recalculated using standardized criteria (see text)
* Restricted to mothers conforming to inclusion criteria (see text).
* Numbers of HCV infected / uninfected / probably uninfected / indeterminate infants (see text)
* 11 older children excluded (unclear whether maternal HCV infection status was determined before delivery).
* Transmission estimates calculated using infants followed up for 18 months (see text).

N/D = no details.

Vertically transmitted, the number of infants assigned to each diagnostic category, and transmission rate estimates. A total of 81 mother-infant pairs were ineligible because mothers were classified as infected solely on the basis of second generation ELISA positivity, or because it was unclear whether maternal serostatus was determined before delivery. These excluded pairs included seven mothers who transmitted infection to their infants. Only three of the 11 studies of HIV negative mothers had 'best' transmission rates of greater than 10%, one of which selected only HCV viraemic women. The one relatively small study of HIV negative mothers documenting more than 20% transmission gave no details of selection criteria. In the six Group One studies which included HIV infected mothers, there was a near consistent trend to increasing HCV transmission with rising prevalence of maternal HIV infection (\(P < 0.0001, \chi^2\) trend), from 0% in a maternal population with 4% HIV coinfection to an estimated 40% where all mothers were HIV infected. There was less evidence of a trend among the Group Two studies (although sample sizes were small), and one study documented 100% transmission, with PCR positivity documented at least once in all five infants included in analyses, and in all three excluded infants with shorter follow-up. One Group Two study used antibody serostatus alone to determine infant infection (Table 2). Only 63% (15/24) of infants were eligible using a minimum follow-up of 18 months, and 7/9 excluded infants with shorter follow-up also seroreverted.

Infection rates in Group Three studies were all less than 10%, despite high prevalence of maternal HIV coinfection in two studies (Table 3).

Viraemic mothers

Figure 1 summarizes 'best' transmission estimates from HCV viraemic mothers with and without HIV infection, from the 15 larger Group One/Two studies with sufficient information. Maternal viraemic status was assessed at delivery in most studies which reported timing of tests. Transmission estimates ranged from 0% to 42%, with less than 10% transmission from HIV infection.
negative mothers in 6/13 studies and from HIV positive mothers in 1/6 studies. There was significant variation in study findings in both maternal HIV groups (P < 0.001 and P = 0.01 respectively), and summary estimates were not calculated. Within three of the four studies which comprised both HIV positive and negative viraemic mothers, 'best' transmission estimates were higher among the HIV coinfection women (Figure 1), although this difference only reached statistical significance in the largest study (P<0.001).

**Level of maternal viraemia**

Nine studies (six from Groups One/Two and three others) were identified which measured levels of maternal HCV viraemia, using a variety of techniques (Table 4). Only two of the 30 viraemic woman who transmitted infection (by researchers' own diagnostic criteria) had viraemia levels of <10^6 copies/mL. 23,31,35 Transmitting mothers in two studies were those with the highest levels of viraemia, 23,31 and four studies reported higher average viraemia levels in transmitting versus non-transmitting mothers, 3,4,35,37 although a significant difference was only reported in one study. 4 Two studies investigated levels of HCV viraemia according to maternal HIV serostatus, one of which reported significantly higher viraemia levels among 18 HIV coinfected mothers (7-8 of whom transmitted HCV) compared with 10 HIV negative mothers, 3 whereas the other (using a semi-quantitative assay) found no significant association between maternal HIV serostatus and viraemia levels in 27 non-transmitting mothers. 6 One study reported lower CD4 levels of maternal viraemia
Table 4 Levels of hepatitis C virus (HCV)-RNA among mothers who transmitted and did not transmit HCV

<table>
<thead>
<tr>
<th>Author</th>
<th>HCV-RNA Quantitative assay</th>
<th>Mothers transmitting HCV</th>
<th>Mothers not transmitting HCV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lin</td>
<td>competitive rt-PCR</td>
<td>1</td>
<td>10^10</td>
</tr>
<tr>
<td>Nagata</td>
<td>limited dilution</td>
<td>1</td>
<td>10^7</td>
</tr>
<tr>
<td>Ohio</td>
<td>limited dilution</td>
<td>7f</td>
<td>mean: 10^6.4±0.3</td>
</tr>
<tr>
<td>Matsumura</td>
<td>limited dilution</td>
<td>3</td>
<td>mean: 10^3.3±0.3</td>
</tr>
<tr>
<td>Moriya</td>
<td>branched DNA</td>
<td>4c</td>
<td>8.6 x 10^6-9.6 x 10^7</td>
</tr>
<tr>
<td>Zanetti</td>
<td>branched DNA</td>
<td>8d</td>
<td>median: 10^7</td>
</tr>
<tr>
<td>Fujisawa</td>
<td>multicyclic rt-PCR</td>
<td>5</td>
<td>10^6-10^7</td>
</tr>
<tr>
<td>Manzini</td>
<td>limited dilution</td>
<td>1</td>
<td>N/D</td>
</tr>
<tr>
<td>Pipan</td>
<td>branched DNA</td>
<td>0</td>
<td>-</td>
</tr>
</tbody>
</table>

**a** By researchers' own diagnostic criteria.

**b** Not all mothers were tested.

**c** Includes mothers not included in transmission rate calculations.

**d** Restricted to HTV+ve mothers.

N/D = no details.

### Figure 2 HCV vertical transmission rates among infants born to HCV non-viraemic mothers with and without HTV infection, calculated from data in refs 3, 6, 45, 46, 48-50, 54, 55, 56, 67 using standardized diagnostic criteria (see Methods): x: vertical transmission point estimates, bars: exact 95% confidence intervals of transmission rates

### Discussion

After adopting standardized diagnostic criteria, HCV transmission was (1) less than 10% overall in most unselected HIV negative pregnant populations, (2) higher in a number of studies which included HIV coinfected mothers, and (3) largely restricted to women with demonstrable HCV viraemia during pregnancy or delivery. Findings of a limited number of studies suggest that transmission risks increase with increasing maternal viral load, with transmissions almost entirely confined to those with viraemia levels of >10^6 copies/ml. Coinfection with HIV may result in increased maternal HCV viraemia, possibly operating via HIV-induced immunosuppression, although not all studies have demonstrated an association between levels of HCV viraemia and CD4 counts. The higher levels of viraemia
and/or higher HCV transmission rates among HIV coinfected mothers rarely reached statistical significance within individual studies, but sample sizes were often small. Variation between studies in overall transmission rates may therefore be partly attributable to differences in the proportion of mothers selected with HCV viraemia or HIV infection.

Transmission from non-viraemic mothers appears to be very uncommon. The eight non-viraemic mothers who transmitted infection may have been misclassified as a result of HCV RNA degradation in stored maternal serum samples. However, HCV infected pregnant women may have intermittent viraemia, and one of the eight non-viraemic mothers was viraemic earlier in pregnancy. These mothers may therefore have transmitted infection whilst temporarily viraemic.

Significant differences in transmission rates remained after accounting for maternal viraemia and HIV infection status, and summary estimates were not carried out. This heterogeneity may have been partly due to varying prevalence of other potential risk factors for HCV transmission, such as viral genotype, degree of liver disease or mode of delivery. Transmission of various HCV genotypes have been reported, and although preferential transmission of specific genotypes has been suggested, individual studies are too small to detect significant associations. Findings are also conflicting with respect to any protective effect of Caesarean delivery.

Secondly, varying numbers of infants may have been at additional risk of postnatal infection. Intrafamilial transmission of HCV is probably uncommon, but the role of breastfeeding is unclear and if maternal HIV serostatus is determined before delivery this may reduce the probability of breastfeeding and thus obscure any association between breastfeeding and infant HCV infection. Although HCV has been isolated from breastmilk, individual studies of HIV negative mothers have reported no transmissions among breastfed infants, or no significant difference in transmission rates between breastfed and non-breastfed infants (Table 5). Ohno et al. found that infected infants were breastfed for longer than non-infected infants, but this difference did not reach statistical significance. Group Three studies may have included a higher proportion of postnatally infected children, although they may also have missed some early resolving infections. The findings of these studies, and of two large studies of children born to women infected via contaminated anti-D immunoglobulin (excluded from analyses due to uncertainty about maternal HCV serostatus before delivery) suggest that the overall postnatal infection risk for children born to HCV infected mothers is low.

Thirdly, there may have been remaining variation in study methodologies after applying standardized diagnostic criteria and minimum follow-up periods. Reliability of PCR can be poor, which may be particularly relevant in studies which tested infants infrequently. Most Group One studies gave little information on children lost to follow-up, and some losses may have related to infant HCV infection. Exclusion of infants with less than 6 months follow-up in Group Two studies was a compromise between two conflicting sources of bias.

Comparability of studies could be increased by standardizing PCR methodologies. Further information is also needed on the temporal patterns of serum HCV infection markers in vertically exposed infants, to inform development of diagnostic criteria. The definitions used in these analyses were adopted to facilitate comparison of study findings, but were not intended to be definitive. For example, women diagnosed solely with ELISA assays may be truly infected and transmit infection to their infants, and the predictive value of a PCR negative result at 3 months may be sufficient to classify infants as ‘uninfected’.
in low risk populations (Thomas et al., submitted for publication). Some infants classified as ‘probably uninfected’ could easily be classified as ‘uninfected’. However, these areas of uncertainty would have made little difference to ‘best’ transmission risk estimates for most studies.

If international consensus were reached on diagnosis of HCV vertical transmission, standardized definitions could be adopted. This would facilitate pooling of individual study findings, to allow more precise estimates of transmission risks and clarify the role of a number of risk factors (including breastfeeding). However, other research is needed to inform the debate on HCV antenatal testing, such as the seroprevalence of HCV infection among antenatal populations (including frequency of HCV viraemia and HIV co-infection), the potential benefits for pregnant women in knowing their HCV serostatus, and the natural history of HCV infection and the efficacy of treatments in perinatally infected children. Widespread antenatal HCV testing can only be considered once all these factors have been taken into account.

Acknowledgements

We are grateful to the following researchers who contributed unpublished data to this review:

1. Professor Ding-Shinn Chen, National Taiwan University Hospital, Taiwan.
2. Dr Angela Ruiz Extremera, University of Granada, Spain.
3. Drs Isabella Quinti & Giulia Scaravelli, Universita La Sapienza, Rome, Italy.
4. Dr Bjorn Fischer, Huddinge University Hospital, Sweden.
5. Dr Takashi Moriya, Hiroshima University School of Medicine, Japan.
6. Dr Vana Papaevangelou, Athens, Greece.
7. Dr Satoshi Koseki, Yokohama City University, Japan.
8. Dr James Goedert, National Institutes of Health, Bethesda, USA.
9. Dr Toomo Fujisawa, National Defense Medical College, Tokorozawa City, Japan.
10. Dr Sachiko Ogawara, Kurume University, Japan.
11. Dr Tatsuo Miyamura, National Institute of Health, Tokyo, Japan (data submitted for publication).
12. Dr Ikuo Nagata, Tottori University, Japan.
13. Dr Adolfo Suarez Gonzalez, Cabuenes Hospital, Gijon, Spain.
14. Dr Susanne Polywka, Universitätskrankenhaus Eppendorf, Hamburg, Germany (data submitted for publication).
15. Dr Antonio Sarrion, Hospital Universitario La Fe, Valencia, Spain.
16. Professor Alessandro Zanetti, University of Milan, Italy.
17. Drs Brunella Guerra, Sonia Bianchi, Paola dalla Casa & Isabella Bosi, Clinica Osteristica e Ginecologica e Pediatrica, Bologna, Italy.
18. Dr Osamu Kurauraichi, Nagoya University, Japan.
19. Professor Raffaella Giacchino, Istituto G Gaslini, Genova, Italy.
20. Dr Rune Wejstal, Goteborgs Universitet, Sweden.
21. Dr Lluís Roura, Hospital Universitari Materno-infantil Valle Hebron, Barcelona, Spain.
22. Dr Wilma Buffolano, Universita degli Studi di Napoli Federico II, Italy.

References


Vertically transmitted hepatitis C virus


