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TIME DECAY OF MATERNAL ANTIBODIES IN INFANTS DEMONSTRATE HIV ANTIBODIES ARE NON-SPECIFIC

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Published data on the decay of maternal antibodies raise questions in regard to the specificity of the HIV antibody tests. In 1973 the evidence was that transplacental antibody did not persist beyond nine months,¹ a view also asserted by a foremost paediatric text, "Maternal IgG gradually disappears during the first 6-8 months of life".² In 1987, without citing evidence of a serological procedure that distinguishes maternal from infant antibody, the CDC stated "Most of the consultants believed that passively transferred maternal HIV antibody could sometimes persist for up to 15 months". In 1991, again without evidence, the CDC extended the time to 18 months and by 1995 "...the range of WB [Western blot] seroconversions might eventually extend beyond 30 months".³

In 1993, researchers from the CDC HIV Serology Laboratory developed "a human immunodeficiency virus type 1 (HIV-1)-specific IgG-Fc capture enzyme immunoassay (IgG-CEIA) to decide the dynamics of HIV-1 maternal antibody decay and de novo synthesis of HIV-1 antibodies in infants". They reported "a rapid decay" of maternal HIV antibody "with decline to background levels by 6 months (T1/2 [half-life] = 28-30 days)",⁴ a result equal to that reported two decades earlier for the "immune globulins".⁵ In other words, any child seropositive beyond 9 months must be generating his own antibodies and thus should remain HIV positive for the remainder of his life.

In the Ariel study of mother to child transmission, in which no child was breastfed, 42% of children seroreverted between 12 and 18 months of age.⁶ In the European Collaborative Study⁷ data on 268 children provide the only published detailed analysis of *post partum* loss of HIV antibody. In this cohort "5% were breastfed for periods from 1 to 30 weeks (median 2.5)". Approximately 23% of infants seroreverted between birth and 9 months and 59% became seronegative between 9 and 22 months. Since seroreversions beyond 9 months cannot be due to loss of maternal antibody, the only explanation is either: (i) the antibody test is non-specific or; (ii) the children managed to clear HIV infection without treatment, which, according to HIV experts, cannot be the case. If 23% of children serorevert because of loss of maternal antibody and in 59% because the antibodies are non-specific, how is it possible to know that the remaining 18% of

children will not also serorevert after 22 months? And if they do not serorevert, the test is specific and proof of HIV infection?

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Addendum for the reader.

This letter addresses why the time rate of decay and time of disappearance of maternal antibody is critical to the notion of specificity of HIV antibodies. Unless one has a method that can distinguish between antibodies of maternal and infant origin (as Parekh developed⁴), it is impossible to state categorically, what proportion of each antibody, if any, is responsible for a positive test. It is also unlikely that the infant's catabolic pathways are able to distinguish HIV antibody from all other transplacental antibodies, which disappear by nine months of age. Nor can the claimed high sensitivity of the tests provide an explanation since sensitivity is not specificity. In fact, despite repeated research of the literature and communication with test manufacturers, we have not been able to determine the dilutional sensitivity of the HIV ELISA or Western blot antibody tests.