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it is well established that blood plasma viral load is lower in hiv-2 compared with hiv-1 infection [34]. it would therefore make sense that viral replication could largely explain the difference in pathogenicity between the two viruses. studies of natural disease progression caused by hiv-1 have indicated large variations in viral loads between individuals and the difference in plasma viral load may not fully explain the difference in rate of disease progression between the two virus infections. interestingly, a recent study showed that cd4+ t-cell levels during the asymptomatic stage of infection was stronger associated with hiv-2 disease progression rate than with cd4+ t-cell decline [35]. further studies are therefore needed to determine the causative effects and predictive values of viral load and cd4+ t-cell levels in natural disease progression of both hiv-1 and hiv-2 infection [36, 37]. both potent and broadly neutralizing antibodies have been detected at different disease stages in hiv-2 infection [51, 76, 77, 78, 79]. moreover, although susceptibility of hiv-2 to neutralizing antibodies seem to vary according to the infecting virus strain, it is in general significantly elevated compared to hiv-1 [77, 78, 79, 80]. fc-mediated effector functions, such as the effect of complement on antibody antiviral activity, have also been reported to be potent in hiv-2 infection [76]. in addition, antibodies with a broad ability to mediate antibody dependent cellular cytotoxicity (adcc), and even cross-react with hiv-1, are found in hiv-2 infected individuals [83, 84]. thus, despite low-level viraemia, strong antibody responses in hiv-2 infection are sustained during both asymptomatic and progressive hiv-2 infections and do not distinguish between patient groups in different disease stages [79]. the impact of antibodies on disease progression during hiv-2 chronic infection is, therefore, not entirely clear.

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although the term "hiv-2" is often used to refer to a number of different strains of hiv-2, we only considered hiv-2 clades b and d in our analysis. the most likely explanation for the slower disease progression observed in hiv-2 is that it possesses a smaller reservoir size, which is consistent with reports of lower viral load at hiv-2 disease progression [21]. the difference in reservoir size may also be due to the specific

mechanisms by which hiv-1 and hiv-2 replicate in infected individuals. there is evidence that hiv-1 and hiv-2 use similar mechanisms to replicate in infected individuals [22, 23]. hiv-2 may therefore use a more efficient and/or robust mechanism to replicate in host cells than hiv-1, and this may explain the lower viral load at disease progression. as hiv-2 infection is very rare, little is known about the pathogenesis of hiv-2 disease progression and how and why the

virus may persist in the host. this review outlines some of the key differences between hiv-1 and hiv-2 infections and the immune responses that affect the host during hiv-2 infection.

furthermore, it highlights the possible contribution of hiv-2 infection to hiv-1 control, and the possible implications of hiv-2 control and remission in hiv-1 cure efforts. it may therefore be important to consider the possible differences in hiv-1 and hiv-2 infections when developing

therapies and approaches for hiv cure. although there are only a few reports of hiv-2 pathogenesis in humans, these reports have clearly defined the characteristics of the infection in vivo. these findings could guide the design of appropriate animal models for the study of hiv-2 pathogenesis and latency reversal strategies. finally, more studies are needed to determine whether hiv-2 infected individuals have a lower risk of developing aids than hiv-1 infected individuals, and whether

the characteristics of hiv-2 infection contribute to hiv-1 control. with the advent of exciting new technologies such as crispr/cas9, ngs, and long-lived lymphocyte repopulation assays, it will be important to assess the role of these new methods in the control of hiv-2 infection. we hope that this review will help to raise awareness of the characteristics of this infection, and the importance of hiv-2 infection in the hiv-1 cure and vaccine effort. 5ec8ef588b

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