Editorial

The discovery of a new virus is always exciting. In this issue of *The Journal,* O'Sullivan et al. (1) describe a new simian parvovirus (SPV), thus adding to a family of successful parasites that are widespread in a range of mammalian species and associated with significant disease in some, including humans. The similarities between SPV and human parvovirus B19 are striking in both molecular and pathogenetic terms. Thus, the discovery of SPV provides an opportunity for a better understanding of both simian and human disease.

In humans B19 infection is a common childhood event with 60% of adult populations exhibiting serological evidence of past infection. In normal children infection is most frequently asymptomatic; however, an erythematous-rash illness, erythema infectiosum, is the most common distinct disease due to B19 infection. The more dramatic clinical manifestation, however, is a profound anemia in individuals who have an underlying chronic hemolytic state making them sensitive to temporary interruptions of erythropoiesis or in individuals who have difficulty in clearing the virus due to lack of a fully competent immune system, e.g., acute lymphatic leukemia, HIV positivity, or infection of the fetus (O'Sullivan et al. references 21 and 22). This predilection for early cells of the erythroid lineage is explained by the fact that the blood group P antigen is the cellular receptor for B19 (2) and that parvoviruses will only replicate in relatively rapidly dividing cells.

SPV was discovered during an investigation of severe anemia occurring in 1992 in four of five cynomolgus monkeys initially housed together and two of their subsequent contacts. All five of the original group had active infections with type D simian retrovirus, and this may be the cause of immunosuppression in the monkeys allowing persistent SPV to cause anemia. Certainly all affected monkeys were viremic at the time of investigation (tested with or without DNA amplication), and this is entirely consistent with the findings in B19 infection in humans. Serological tests of infection are now required to determine whether cynomolgus monkeys are the natural host of SPV and to study the epidemiology and natural history of the infection. It is interesting that the virus was also found in the serum of one of a small group of cynomolgus monkeys with anemia in the same animal facility in 1991.

The characteristics of the anemia and the features of the bone marrow aspirates from SPV-infected monkeys are strikingly similar to those found in humans infected with B19 virus. However O'Sullivan et al. (1) comment particularly on the morphologically abnormal erythroid cells in the simian bone marrow aspirates showing evidence of regeneration. They remark that the dyserythropoiesis is similar to that found in cases of congenital dyserythropoietic anemias in humans and speculate on the possible link with fetal B19 infection. This inevitably leads to a recognition of the potential importance of SPV infection in monkeys as a model for studying one of the major remaining questions in B19-related disease, namely what is the full range of consequences of intrauterine infection.

Viremia is a consistent feature of B19 infection; should this occur in pregnancy there is ample opportunity for infection of the placenta and fetus. There is no doubt that the fetus does become infected since the virus can be identified in tissues from cases of hydrops fetalis (e.g., reference 3) which is the most dramatic manifestation of intrauterine B19 infection. It should be emphasized that most pregnancies complicated by B19 result in the full-term delivery of normal infants, but about 10% end in fetal loss, most frequently in the second trimester (4) and sometimes in association with fetal hydrops. This situation in which pregnancies complicated by infection either proceed normally or, in a minority of cases, are lost means that difficult decisions about termination of affected pregnancies are not usually required. Certainly intrauterine B19 infection is not frequently associated with congenital abnormalities (5). However, the question as to whether functional or anatomical abnormalities occur at a rate of less than 1% remains unanswered. The paper on SPV in this issue alludes to the unpublished observations of Neal Young and colleagues at the National Institutes of Health of an association between intrauterine B19 infection and some human congenital dyserythropoietic anemias or conditions such as Diamond-Blackfan anemia. Moreover, Larry Anderson and colleagues at the Center for Disease Control (Atlanta) have unpublished data possibly indicating a low incidence of central nervous system abnormalities after intrauterine infection. It would take a very large series of human cases to prove or disprove these associations, and it is in this context that the new potential for experimental infection with SPV in monkeys is particularly important.

There is already a genetically engineered vaccine against B19 under development. Nevertheless, it is notable that SPV shows about 65% homology with B19 in the region of the major capsid protein. Assuming rapid progress towards the expression of the structural proteins of SPV, then there is a potentially important series of experiments to be done on the vaccination of monkeys against SPV that would inform the process of the prevention of B19 by vaccination.

As is true of most new viruses, we see that the discovery of SPV is likely to confirm certain general principles about the genus as a whole and add significantly to our knowledge of animal viruses and the diseases they cause. The authors are to be congratulated on their original findings.

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