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EDITORIAL

A new pathogenic human parvovirus

The first parvovirus to be detected in the human population was found in 1975 during routine screening of blood donors [1,2]. Working in Australia, Yvonne Cossart found a parvovirus-like antigen in the sera of nine healthy blood donors and two patients. The spherical particles were 21–23 nm in diameter and a corresponding antibody was found to be widely distributed in the population. Because the original sample in which the parvovirus-like particles were seen was coded 'B19' this became the name of the human parvovirus. Following this chance discovery, a few years elapsed before a survey of serum samples archived at King's College Hospital, London revealed an association between B19 virus infection and aplastic crisis in children with sickle cell anaemia [3]. Later in 1981 it was reported that 24 of 28 young children in Jamaica with sickle cell anaemia who had an aplastic crisis were infected with a parvovirus [4] and similar findings were reported concerning adult patients with sickle cell anaemia in Chicago where aplastic crisis was associated with acute infection with a human parvovirus [5]. In 1983, the virus was shown to be the cause of a childhood rash called erythema infectiosum (fifth disease) [6], and this was confirmed 2 years later by experimentally infecting human volunteers with the virus [7]. The final step in characterising the virus, which appears to infect and damage early erythroid progenitor cells, was to characterise, clone and sequence the genome of the virus, and this established definitively that it was a parvovirus [8,9]. However when the sequence was analysed in detail, it was phylogenetically distinct from the existing genera (*Parvovirus* and *Dependovirus*), and so was placed in a new genus named *Erythrovirus*.

In 2001, Allander *et al*. working at the NIH reported a new method for virus 'discovery' in serum or plasma based on treatment of the sample with DNase followed by restriction enzyme digestion and sequence-independent single primer amplification of the fragments [10]. Whilst evaluating the method, they detected two previously unknown bovine parvoviruses in the bovine serum used as diluent. When sequenced, the two viruses were distinct, but different from all other known parvoviruses except a virus of dogs, Canine minute virus. So it was decided by the ICTV to include the bovine and canine viruses in a new genus to be named *Bocavirus*, of the subfamily *Parvovirinae* within the family *Parvoviridae* [11]. When Allander *et al.* now working in Sweden, applied the virus discovery method to screening of pooled human respiratory tract samples, they found a novel human coronavirus (which is related by sequence to the newly described human coronavirus HKU1 [12]), and a novel human parvovirus, which is related by sequence to the genus *Bocavirus*, and so has been provisionally named human bocavirus [13].

Several laboratories worldwide have now confirmed the existence of human bocavirus in 1.5–5.7% of respiratory samples tested, and it appears to be associated with lower respiratory tract disease, though in many cases there is co-infection with another respiratory virus, so the exact role of human bocavirus in pathogenesis remains the subject of further studies. So far, reports have been published from Australia [14], Canada [15], France [16], Japan [17] and Korea [18]. More are in press.

In the past, the majority of viruses infecting humans were found because of the disease symptoms they caused. It is, therefore, worth noting that both human parvoviruses, B19 virus and human bocavirus, were initially discovered without an obvious association with disease. This suggests that a systematic search of human samples for viruses of other families might be profitable, as suggested by Allander *et al*. [13]. For example, could there be a human arterivirus waiting to be discovered?

BWJ Mahy

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