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SCIENTIFIC CORRESPONDENCE

Borna again, starting from the beginning

SIR – Recently, *Molecular Psychiatry* published an article discussing the issue of neutropic viruses in neuropsychiatry.[1] We agree with the author of this article that neutrotropic viruses are an important, active and interesting area of study in neurodevelopmental injury research and appreciate the interest expressed in animal models of human psychiatric disease. However, some of the statements regarding the 'new' neonatal rat infection with the Borna disease virus (BDV) animal model were misleading for your readers, who may not be aware of almost two decades of previous publications in this model system and its previous application to human disease pathogenesis.

The editorial referred to the development of a 'new' animal model of human psychiatric disease by Hornig *et al*,[2] citing a publication in *Proceedings of the National Academy of Sciences of the USA* (*PNAS*), wherein the authors present data from studies of neonatally BDV-infected rats. Unfortunately, Dr Collier failed to note the extensive work in this model system previously published by other investigators prior to the *PNAS* article, many of which are cited in the *PNAS* article's bibliography. The work published by Hornig *et al* in *PNAS* provides some new data (eg, apoptosis) and further elaboration of many previously published features of this model system (eg, cerebellar deficits, hippocampal degeneration, and increased spontaneous locomotor activity). However, the data presented in *PNAS* follow the development of this model system by the work of multiple laboratories over the past 20 years.

For the edification of your readership with an interest in animal models of human psychiatric disease, we summarize the development of the neonatal rat BDV infection model as follows. In 1983, two independent groups, Narayan *et al*[3] and Hirano *et al*[4] were the first to characterize the non-inflammatory brain infection and note some intriguing behavioral abnormalities in rats following neonatal infection with BDV. In 1989, Dittrich *et al*[5] published the first formal evaluation of select behavioral abnormalities in the neonatal BDV infection rat model, presenting data on abnormal learning and hyperactivity. These initial studies sparked our interest, almost 10 years ago, in this model system and resulted in our laboratory publishing a series of studies characterizing this model and citing its value for human psychiatric disease pathogenesis studies, some of which are cited in the bibliography below.[6–13] Over the past decade, our studies defined and expanded upon the core constellation of abnormalities that led to the recognition of the neonatally BDV-infected rat model as a valuable animal model system of developmental neurobehavioral disease. Those reports included data on neuroanatomical deficits (eg, abnormal cerebellar and hippocampus development), neurochemical abnormalities (eg, serotonin neurotransmitter system), behavioral deficits (eg, hyperactivity, social behavior, chronic anxiety), and physiological deficits (eg, stunted body growth, abnormal taste preferences). Finally, one of our recent publications, in print at the time of submission of the *PNAS* publication, clearly stated that the neonatal BDV infection in rats was a valuable animal model for autism.[10]

This correspondence is in no way intended to criticize the valuable scientific data presented by Dr Hornig *et al* in their first publication on this model system, and, indeed, by several other research groups working on this model in the US, Japan and Europe. We are delighted at the recent recognition of the value of the neonatally BDV-infected rat model system, as evidenced by the increasing number of publications resulting from studies of this model, for their contributions to the scientific knowledge of virus-induced developmental neurobehavioral disease. Rather, it is our intent to inform your readers that *Molecular Psychiatry* editorial's attribution of development of this 'new' model is incorrect, since the author failed to note that this model was a previously characterized animal model for human psychiatric disease pathogenesis study, including the study of autism.

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