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Early Mouse Models of Human Diseases

The News Focus article "The rise of the mouse, biomedicine's model mammal" by David Malakoff (14 Apr., p. 248) highlights the mouse as a mammalian model for human diseases, with the emphasis being on the provision of mouse strains and mutants by suppliers. In this otherwise fine survey of the role of the mouse as a prototype of human genetics and physiology, there is, however, little mention of the numerous mouse mutations as counterparts of human diseases that were already discovered in the early part of the 20th century. For instance, mouse pituitary dwarfism was discovered by Snell in 1929 (*1*). A^y, the top-dominant allele of the agouti coat color series, was discovered by the French geneticist Cuénot in 1905 (*2*). Many animals with the A^y gene become remarkably obese and thus offered an early model for human obesity (*3*). At least six genes were known early in the century to produce neurological and labyrinthine disturbances, the prototype being the Japanese waltzing mouse (*4*).

An outstanding resource that would have been valuable to mention is the volume by Hans Grüneberg titled *The Genetics of the Mouse* (*5*), a scholarly treatise about the mouse, including detailed descriptions of mouse mutants, gene action, genetic strains, and cancer genetics. It was first published in 1943 and enlarged greatly for the second edition in 1952, which includes more than 1700 references. Modern physiologists and geneticists should be aware of this gold mine of information about mouse mutants, many of which can still be obtained and studied using modern biochemical and molecular techniques. These mutants exhibit many abnormalities, including those in the central nervous system, the eye, blood and blood-forming organs, skin, and skeleton.

My criticisms should not detract from the thrust of the article, which is that the mouse should be recognized as the prime mammalian model of human genetics and physiology.

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Antipsychotics and Working Memory in Schizophrenia

Patients with schizophrenia suffer from substantial cognitive deficits, notably in the realm of memory functioning (*1*), which warrants considerable research efforts aimed at developing pharmacological treatment strategies. S. A. Castner, G. V. Williams, and P. S. Goldman-Rakic describe in their Report (17 Mar., p. 2020) the reversal of antipsychotic-induced working memory deficits in monkeys by stimulation of short-term dopamine D1 receptors. They suggest that the results of their study may have therapeutic implications for schizophrenia. However, the putative implications of their results for treatment of cognitive dysfunction in schizophrenia may be somewhat overstated.

Castner and colleagues propose that chronic haloperidol treatment should induce