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Preface

Special issue: Animal models of disease

Animal models are a vital resource for modern biomedical research. The variety and complexity of animal models available to researchers have increased remarkably over the past several decades, and now encompass a wide range of species, both vertebrate and invertebrate. Although there have been some modest advances in non-animal alternatives in recent years, we still rely on animal models as a means to obtain answers to difficult biological questions that cannot (as yet) be found in other ways. Given our incomplete understanding of whole organism physiology, it is likely that this situation will continue for the foreseeable future.

When we think of animal models of human disease, we often think of genetically modified mice. Introducing a genetic change into a mouse line, either by generating a transgenic mouse or through gene targeted knock-in or knock-out, has become a standard method of studying the mechanisms that underlie many human diseases. Further, many human diseases have no ready animal counterpart, and introducing a genetic modification into a suitable organism can often at least partially mimic the disease. This has the added advantage of creating a system that might theoretically be used to develop and test potential therapeutics. Although mice are the most widely used mammalian models, this technology is now more common in rats, and in some larger animal species as well. Zebrafish are only recently being tapped as important vertebrate models, and their true potential will likely be realized in the very near future.

There is no denying that we can do a better job of refining our model systems. This can be thought of not only as a means of obtaining a better understanding of disease, but also from the perspective of improving animal welfare. Understanding diet and metabolism, and the role of modifier genes, will be an important part of this process. As our models grow in both ambition and complexity, this area represents the greatest potential for innovation and growth. This special issue only scratches the surface of where we can go in the future.

Dr. Murphy has worked as a researcher in the areas of aging and neurodegenerative disease for more than 20 years (starting with his M.A. degree, in psychology, at the University of Toronto, followed by a Ph.D. in neuroscience). He began to focus on Alzheimer's disease (AD) while a post-doctoral fellow at the Mayo Clinic in Jacksonville, FL, in the lab of Todd Golde. He moved to the University of Kentucky in 2005. His lab studies the production of the amyloid-β peptide (Aβ), the regulation of these processes, and how the peptide ultimately causes AD pathology in the brain. His lab has a strong focus on models of disease, mainly genetically modified mice, but also in higher mammals and in cultured cells. Dr. Murphy has been engaged throughout his career in basic research, preclinical translational research, and in the early stages of human clinical trials. Dr. Murphy has authored or co-authored almost 90 journal articles, including influential articles in Science, Nature, Nature Medicine, and recently (this past year) in the Annals of Neurology and the American Journal of Pathology. In 2010, Dr. Murphy was honored by the receipt of the Thomas Maciag Award from NIH for exemplifying research excellence and innovation, and for the mentorship of new scientists. Dr. Murphy is probably best known for the study and development of different models of AD and related pathology. Several of the mouse models that he was involved with from the development stage onwards are well known and widely used in the AD field, and he has several notable papers exploring AD pathology in elderly cats, dogs, and people. Dr. Murphy's recent research, includes understanding how life style choices, particularly those that lead to obesity and type 2 diabetes, can eventually lead to neurologic disease.

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