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Editorial

Exploring Experimental Animal Models in HIV/AIDS Research

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INTRODUCTION

Animal models serve as important tools for preclinical testing of therapeutic regimens against human immunodeficiency virus (HIV-1), the primary etiologic agent that causes acquired immunodeficiency syndrome (AIDS). Infection and treatment of patients often cannot be controlled in clinical studies. In addition, performing certain procedures and sampling cannot be routinely performed in humans with ease and may be unethical. There are many different primate and murine models of HIV/AIDS, each with their advantages and disadvantages. Some models are appropriate in certain contexts but not others. Knowing how the different models work and their limitations will help guide the researcher to select the appropriate model to answer a specific question. Information gained from the use of preclinical testing of antiretroviral therapies will help identify and improve preventive, therapeutic, and eradication strategies against HIV/AIDS in humans.

HIV-1 INFECTION OF NONHUMAN PRIMATES OR HUMANIZED MICE

Which is the better model to use? An animal model for human disease should mimic the infection of humans as closely as possible. The disease course in the model should be similar to or more accelerated than in humans. In the case of HIV-1, an animal model that progresses to AIDS over the period of many years will cost time and money in preclinical studies. The use of animals instead of humans usually means certain procedures can be performed more easily and/or ethically. For example, removing vital organs to study pathogenesis, drug penetration, immunity, or virology cannot be performed in humans but can be done after necropsy of an animal. Moreover, unlike in humans, the exact virus, timing of infection, and timing of treatment can be controlled in a model.

THERAPY FOR HIV PREVENTION IN ANIMAL MODELS

As there is no cure for HIV-1 yet, efforts have been made to develop and evaluate compounds that would prevent HIV-1 infection prior to or immediately after exposure to the virus. These differ from vaccines in that they are not designed to elicit antiviral immunity in advance of exposure, but rather would inhibit the virus before, during, or just after exposure to HIV-1 to avoid systemic infection. Pre-exposure prophylaxis would be initiated in high-risk individuals likely to be exposed to HIV-1, whereas post-exposure prophylaxis would be used in individuals who were believed to be recently exposed to the virus. Animal models have been used rather extensively over the past decade in this area of research with generally positive results. Unlike in clinical trials, the timing and adherence of treatment and the timing of virus challenge can be controlled in the model.

New delivery methods one complication of HIV-1 infection is the presence of large numbers of infected cells within different tissues. Although discussed more in the next section, CD4+ target cells are present within lymphoid tissues, the brain, and multiple mucosal tissues. Drug penetration into these tissues is likely to be lower than in the blood, allowing viral replication to continue within tissues. Research on better drug delivery methods into different tissues has been ongoing. This will be especially important for methods to target drugs into mucosal tissues during pre-exposure prophylaxis and also persisting viral reservoirs. Transfer Inhibitors (INSTIs), Nucleoside/Nucleotide Reverse Transcriptase.

CONCLUSION

The use of macaque and mouse models for antiretroviral therapies. These animal models have improved greatly since the development of SHIVs and the ability to reconstitute a functional human immune system in mice. Refinement of these models is ongoing to make them more closely resemble humans with regards to infection, pathogenesis, and antiretroviral immunity. For example, macaque models can be improved by increased understanding of macaque cell proteins that inhibit HIV-1 infection use of Animal Models for Anti-HIV Drug Development 85 the genetic differences between HIV-1 and SIV, particularly the accessory proteins that appear to significantly contribute to pathogenesis.

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