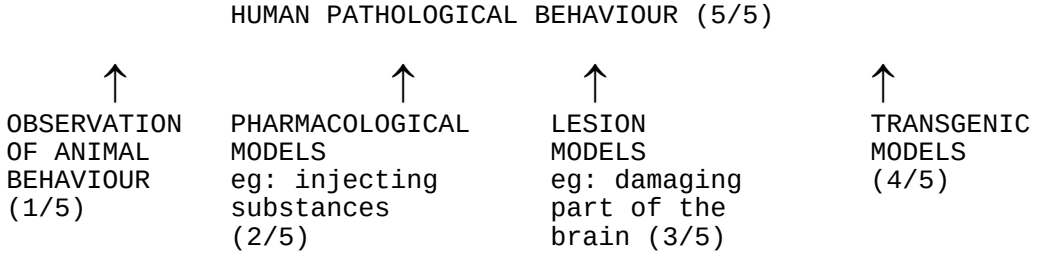
TRANSGENIC STUDIES IN PSYCHOLOGY: A NEW ISSUE FOR ANIMAL RESEARCH ETHICS

INTRODUCTION

Non-human animals have been studied throughout the history of psychology in order to understand human behaviour. In recent years, animals have been used in a different way in psychology (and science generally). This is by genetic modification of the animals including using human genes (as in transgenic studies).

These types of studies, which change specific genes in the embryo, are most useful for understanding pathology. Genes can be manipulated to create the pathology, and thus isolate the genes involved, in a way that is not possible with humans. In other words, to go from healthy to unhealthy. Any use of genetic manipulation in humans would be the other way around.

For researchers using these types of studies, genetic manipulation is the best of the methods available to understand a pathology. In figure 1, each method of studying non-human animals gives varying degrees of detail for understanding human pathological behaviour. Transgenic models are the closest yet (scoring four out of five), but they are not necessarily perfect.



(After Codita et al 2006)

Figure 1 - Nearness of different animals models to human pathological behaviour.

There are concerns about transgenic studies including the patenting of new genetic versions as if the animals were products. The correct reference number allows ordering of particular types of genetically manipulated animals from a mail-order catalogue. Debate about the right and wrongs of using hon-human animals in psychology experiments can be outdated compared to the new areas like this where human genes are placed into mice, for example.

Table 1 summarises the main arguments for and against transgenic studies.

ARGUMENTS FOR	ARGUMENTS AGAINST
- Able to manipulate genes in way that unacceptable with humans	- Moral argument of treating animals in such a way; eg: adding human genes; patenting as if products. These animals live just to show the pathology
- Change genes and isolate those that cause problems	- Transgenic studies found different results depending on the process of genetic engineering or the breeding strain of animal
- Help find a cure for human conditions caused by genes	- Applicability of findings to humans. Do human genes behaviour the same way when placed in other species?
- Better method than other types of animal studies	- Ignores the role of the environment: eg dementia in humans affected by nutrition and physical exercise

Table 1 - Main arguments for and against transgenic studies.

MICE AND DEMENTIA

Cordita et al (2006) saw transgenic models "allowing ethically approved manipulations that cannot be performed on humans" as "a natural approach for Alzheimers disease researchers". Mice are easier to handle and less expensive than primates to study in this way.

The goal is "of modelling an animal so that it could resemble, as much as possible, the features of humans suffering from Alzheimers disease" (Cordita et al 2006 p560).

The mouse model of dementia has been developed by changing certain genes:

i) Changing a single gene

Amyloid precursor protein (APP) is linked to the build up of plaque in the brain as a cause of dementia Alzheimers type (DAT). Ultimately this plaque leads to nerve cells dying.

Games et al (1995) produced a mouse with a specific mutation to the APP gene which had amyloid deposits by eight months old. These mice, known as PDAPP, had

problems learning.

Studies involving this mouse and other transgenic ones related to APP sometimes find differing results. The breeding strain of mouse and the promoter are important (Cordita et al 2006). The promoter is the means by which a gene is "driven" (ie: how the human gene part into the mouse).

ii) Changing two genes

For example, a mutant known as Tg CRND8 combined two different APP mutations (eg: Janus et al 2000). This mouse showed accelerated amyloid deposits. Other combinations include two genes that are involved in different processes (eg: APP and presenilin genes). Presenilin genes code for certain cell proteins (Brindle and St.George-Hyslop 1998).

iii) Changing three genes

Oddo et al (2003) co-microinjected two independent genes into the single cell of a mouse embryo, which already had a mutation. This 3xTg-AD mouse, thus, had three genetic changes.

Researchers are happy that transgenic mice show aspects of DAT in terms of the physiology of the brain. This type of research, say the advocates, could help in finding a cure for DAT: these studies "can take us further towards completing the circle of knowledge in the human pathology of Alzheimers disease" (Codita et al 2006).

But findings from mice do not necessarily apply to humans. For example, clinical trials of a vaccine to combat amyloid deposits showed severe side effects in humans, which had not been found with mice (Orgogozo et al 2003).

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