Open University animal research

THERE were substantial delays to the release of the OU Biology Department's 2001 report to the Animal Ethical Committee due to the Animal Ethical Committee's request for several changes.

Unless otherwise stated, the information below relates to the year 2001; despite requests since October 2003 the OU has failed to provide us with the 2002 report by March 2004.

The general covering statement in the Research and Graduate Teaching section of the report includes the following:

> *"The number and species of animals used varies from year to year, depending on the investigations* (experiments) *performed and the number of researchers involved...All experimental animals are killed at the end of the experiment."*

The second sentence represents a change from the statement used from at least 1991-2000, which included the phrase

> "*with or without killing them at the end of the experimental period*."

The change suggests that the previously-used wording was inaccurate. Another claim, made in 2000, that

> "*no vivisection is carried out at the OU*"

has been dropped, apparently as a result of pressure from SES. Our lawyer deems this claim an example of 'semantic sophistry'.

A substantial proportion of OU animal research is on the brain, purporting to have potential benefits for the much-feared Alzheimer's disease.

Professor Steven Rose continues his apparently interminable research on chicks, and Head of Biology Professor Mike Stewart is now studying rats,

both claiming that their work has relevance for human memory. One of Stewart's published papers refers to the proliferation of neurons (nerve cells) in 'trained' chicks. This does not occur in humans after birth.

Under principal researcher Steven Rose, chicks have been 'trained' on a passive avoidance task or a bead-floor visual discrimination task for many years. This involves them pecking at a bitter bead or at chick crumbs on the cage floor. Various substances have been injected into their brains and the effects of these on their performance noted. (After 'training' in 1994, chicks were given electric shocks to cause amnesia. It is not clear whether or not these procedures were followed in 2001, as the amount and type of detail in the report to the Animal Ethical Committee varies from year to year.) 300 chicks were being used **per week** by Rose's team in 1991, since when figures have been withheld; however, Rose admitted on the BBC Radio 4 programme 'The Moral Maze' in 2002 that his research kills ten thousand chicks a year.

The chicks are killed by decapitation and their brains are examined for biochemical changes resulting from the 'training'. Rose has abandoned the implication, included since 1997, that his work has relevance to

> "*deep philosophical questions of the relationship between mind and brain and the question of consciousness*"

and

> "*understanding of the prevention and treatment of...diseases such as Alzheimer's*".

He has dropped another claim, also included since 1997, that studies of mechanisms in chick brains point directly to how memory loss occurs in human dementia. This has been replaced with

> "*although the impetus for the work over many years has been that of basic science, work over the past couple of years has increasingly turned to exploiting our findings to explore potential treatments for memory loss in Alzheimer's disease*".

Professor Rose and colleagues published 5 articles based on the research described above in 2001.

Professor Mike Stewart heads other teams researching cellular mechanisms of learning and memory, this time using rats. One of Stewart's experiments, the results of which were published in 2000, involved immobilising 6 anaesthetised rats in stereotaxic holders in recording chambers, drilling holes in their skulls and stimulating parts of their brains with electrodes.

From 1998 to 2000 Professor Stewart was also involved in US-based experiments on mice 'modified' to possess a human gene which is involved in Alzheimer's. There is no mention of this project in the 2001 report.

The claim made in 1999-2000 for Stewart's work that it

> "*provides additional insights into the cellular mechanisms of neural plasticity in rats and mice*"

has been replaced with

> "*the overall goal of this research is to provide…*"

(Note – mice are still mentioned here.)

Stewart's report states that

> "*following stimulation of nerve tissue, there is a marked alteration in neural circuitry in the key region of the brain involved in learning, the hippocampus.*"

As the normal learning process involves natural electrical stimulation of learning-related brain regions, all this seems to prove is that we can simulate this, and the implications are unclear. In any case, rodents have far superior regenerative capabilities to humans. Furthermore, even in primates, particular brain functions do not always occur in the same part of the brain as in humans, and animal experiments have consequently produced misleading, even useless, results. This clearly *hinders* scientific progress.

Rose and Stewart's research is excessively reductionist, and is unlikely to translate into benefits for human sufferers. A report (Langley *et al*., 2000) from a workshop including the OU's own Dean of Science Dr Stephen Swithenby states:

> *"the precision of animal studies may be superfluous if the results are not directly transferable to humans"*

and

> *"human studies of disease evolution...particularly with dementia...are revealing the limitations of some traditional animal methods".*

Langley and colleagues' report describes scanning techniques which can be used to study the effect of Alzheimer's on <u>human</u> neurons. These plus clinical, epidemiological, *in vitro* and post-mortem studies are the preferred methods of many scientists studying dementia in humans. Please contact Vivien for details of bequeathing your body/brain for humane research.

There are also ample data from human studies on the benefits of nutritional supplementation, and other lifestyle changes, for the prevention and treatment of dementia (Pomfrey, 2002). Most illness in the industrialised world is a result of diet and other lifestyle factors, stress and pollution.

Even if an animal 'model' can be induced to develop illness similar to that seen in humans, and a drug is found that prevents or cures it in the animal, the following problems will still exist:

1. Humans and other animals do not react to drugs in the same way (*see* Animal Aid, 2002; Students for Ethical Science, 2004).

2. t is likely to be impossible to correctly identify human sufferers on whom a clinical trial could be carried out, as diagnostic criteria are extremely inconsistent and unreliable, even when it comes to deciding whether or not someone is demented (Erkinjuntti *et al*., 1997), before using further inconsistent and unreliable protocols to attempt to determine which kind of dementia they might have (Pohjasvaara *et al*., 2000).

3. If a drug were to be approved following such a deeply flawed trial, the same problems would pertain in identifying patients 'suitable' for more widespread use of the drug.

4. Even post-mortem, pathologists cannot agree on whether brains come from demented or non-demented patients, or whether they had Alzheimer's or vascular dementia (Ince, 2001).

5. (This applies to all reductionist molecular research.) We are still discovering new roles for the body's own chemicals - for example vitamin D is now believed to be produced in the brain and to have a neuroprotective role, in addition to its production in the liver and kidney and role in bone maintenance. Yet we use foreign chemicals to disrupt tiny sections of biological pathways in the hope that they have just one specific effect. Poor specificity means that few animal-research derived drugs are without side-effects, and hundreds of thousands of people die every year from prescribed drugs.

The third project, conducted under principal researcher Dr Caroline Pond, relates to the relationships between adipose tissue (fat) and the immune system. In one experiment, guinea pigs bred at the OU were fed on different diets for a number of weeks, injected in the leg daily for 4 days, and then killed. The injections induce a full immune response in a lymph node. Rats were given similar treatments before being killed. There is no mention in 2001 of the mouse experiments detailed in 1999 and 2000, so presumably these have ceased.

Pond's third experiment used bought-in guinea pigs, which were fed on various diets, injected in both hind legs 3 times a week for 2 weeks to simulate HIV-like chronic, low-level immune activation, then killed. Their cells were then tested with human HIV drugs.

Pond's report claims that her experiments are relevant to HIV-associated fat redistribution syndrome, whilst conceding that this was a completely unexpected side-effect of anti-viral drugs – which, of course, had been tested on non-human animals! Even our closest relatives - chimpanzees - do not develop AIDS, so it is difficult to see how experiments on rodents can provide any useful knowledge about this disease.

4 primary journal articles, 4 review articles and one book or popular article, based on the above experiments, are listed in the 2001 report.

Pond's animal experiments on adipose tissue have been ongoing since at least 1982. Her work used 70 rats, 60 guinea pigs and 50 dwarf hamsters in 1991, since when figures have been withheld.

It is disingenuous for animal researchers to keep restating the facts that their work meets the requirements of the Home Office. There are just 21 Home Office inspectors for 16,000 animal experiment licence-holders. They failed to stop gross cruelty at Huntingdon Life Sciences and Cambridge University, which was eventually exposed by undercover animal rights activists.

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