The Commonsense Case against Animal Experimentation

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As this volume illustrates, most arguments for the immorality of animal experimentation take one of two forms. Either they follow Peter Singer’s lead and maintain that most animal experiments are morally unjustifiable on utilitarian grounds; or they follow Tom Regan’s deontological rights-based approach and insist that virtually all of the animals experimented on in research facilities around the country possess the very same properties that confer rights on humans, and therefore, experimenting on these animals is wrong because it violates their rights. When confronted with Singer’s and Regan’s arguments opposing animal experimentation, proponents of animal experimentation tend to casually dismiss these arguments by rejecting the ethical theories on which they are predicated. These dismissals take roughly the following form: “Singer’s preference utilitarianism is irremediably flawed, as is Regan’s theory of moral rights. Since Singer’s and Regan’s arguments against animal experimentation are predicated on flawed ethical theories, their arguments are also flawed. Until someone can provide me with clear moral reasons for not experimenting on animals, I will continue to experiment on animals as I see fit.”

Consider two examples. In an effort to defend animal experimentation, Carl Cohen (2001a and 2001b) goes to great lengths to try to show, contra Regan, that nonhuman animals lack rights and that, therefore, it is permissible to experiment on them (as if the latter followed from the former). In an article published in the Journal of the American Medical Association, Richard Vance admits that: “Both [Tom Regan and Peter Singer] are exceptionally good philosophers in the analytical tradition. They provide sophisticated defenses of their positions” (1992, 1715). However, Vance rejects their defenses of the immorality of animal experimentation because he rejects the analytical ethical tradition on which they are based. As Vance
sees it, Singer's and Regan's arguments against animal experimentation ultimately fail because of the
limited nature of the philosophical tools they use. Their ultimate theoretical weaknesses are extremely common among analytical ethicists. Unlike more substantive ethical traditions (for example religious or ethnic traditions), analytical ethics cannot draw on a rich array of sources—canonical texts, authoritative readings, overlapping (even contradictory) platiitudes, interpretative communities, and the like. In comparison with such traditions, analytical ethics is abstract and thin. Despite claims of rational consistency, no analytical model has been able to claim adequacy. (1715)

A moment's reflection reveals the self-serving sophistry of such a reply. Since no ethical theory to date is immune to objection, one could fashion a similar reply to "justify" or rationalize virtually any behavior. One could "justify" slavery as follows: An opponent of slavery might appeal to utilitarian, Kantian, or contractarian grounds to establish the immorality of slavery. Our fictitious slavery proponent could then point out that all of these ethical theories are flawed and, ipso facto, so too are all the arguments against slavery. Our slavery proponent might then assert: "Until someone can provide me with clear moral reasons for abolishing slavery, I will continue to own and exploit slaves."

The speciousness of such a "justification" of slavery should be obvious. No one who seriously considered the brutality and inhumanity of slavery could think that it is somehow permissible simply because all current ethical theories are flawed. But such specious reasoning is often used to "justify" the equally brutal and inhumane breeding, confining, infecting, injuring, mutilating, maiming, blinding, torturing, and killing of animals in animal experiments. My aim in the present chapter is to block this spurious reply by providing an argument for the immorality of animal experimentation that does not rest on any particular highly contentious ethical theory. Rather, it rests on commonsense moral beliefs that we all share.

Before turning to these beliefs, a few prefatory observations are in order. First, unlike other arguments for the immorality of animal experimentation, my argument is not predicated on the wrongness of speciessism, nor does it depend on your believing that all animals are equal or that all animals have a right to life; rather, it is predicated on several commonsense moral principles which you no doubt believe. The significance of this argumentative strategy is two-fold: First and most important, all effective argumentation must start with premises one's interlocutor accepts. The reason Singer's and Regan's arguments sometimes fall on deaf ears is that their arguments do not start with premises their readers share. In contrast,

my argument starts with premises the reader already accepts and traces out the moral implications of those premises. Consequently, the reader is already rationally committed to the truth of the resulting conclusion, on pain of inconsistency.

Second, some philosophers remain unmoved by Singer's and Regan's arguments for a different reason than the ones just cited. These philosophers find that the nonspeciessistic implications of Singer's and Regan's arguments just feel wrong to them. They sincerely feel that humans are more important than nonhumans. Perhaps, these feelings are irrational in light of evolutionary theory and our biological kinship with other species, but these feelings are nonetheless real. My argument is neutral with respect to such sentiments. It is compatible with both an anthropocentric and a biocentric worldview. In short, my argument is designed to show that even those steadfastly committed to valuing humans over nonhumans are nevertheless committed to the immorality of animal experimentation, given their other beliefs.

Finally, although animals are used in all sorts of scientific research, including cosmetic testing, household product testing, and psychological experimentation, in what follows, I will focus primarily on the use of animals in biomedical research, since if it is wrong to use animals in experiments aimed at the development of vitally important, potentially lifesaving drugs, then it is a fortiori wrong to use animals to test unnecessary trivial products like a new floor wax or a new shampoo. Having clarified the scope, significance, and rationale for the argumentative strategy that I will be employing, I now turn to the argument itself. In section I, I identify several commonsense moral principles that we all accept. Then, in sections II-IV, I use these principles to develop an ethical consistency argument designed to show that using animals as test subjects in biomedical research is wrong.

I Common Ground

My argument for the immorality of using animals as test subjects in biomedical research is predicated on several widely accepted, commonsense moral principles—principles that you no doubt already believe. These commonsense principles are so central to our conception of morality that any moral theory that conflicted with them would be rejected as unsatisfactory on reflective equilibrium grounds. Since any adequate moral view must cohere with these principles, we can appeal to these principles directly when making moral evaluations. The principles are these:
(P1) It is wrong to intentionally harm conscious sentient animals *for no good reason.*

(P2) It is wrong to cause conscious sentient animals to suffer *for no good reason.*

(P3) It is wrong to kill conscious sentient animals *for no good reason.*

These principles are not in dispute. Even the staunchest defenders of animal experimentation embrace these commonsense principles. For example, Cohen explicitly endorses (P2) and (P3): "If animals feel pain (and certainly mammals do, though we cannot be sure about insects and worms), *we humans surely ought cause no pain to them that cannot be justified. Nor ought we kill them without reason*" (2001a, 46; emphasis mine).

Elsewhere, Cohen reiterates his commitment to (P2) and (P3): "Our obligations to animals arise not from their rights, I believe, but from the fact that they can feel pain and from the fact that we, as moral agents, have a general obligation to avoid imposing needless pain or death" (ibid., 226; emphasis mine). Similarly, Peter Carruthers acknowledges that sentient animals deserve some moral consideration when he explicitly endorses (P2): "It will be useful to have a rough idea at the outset of what our common-sense morality tells us about the status and appropriate treatment of animals. . . . Many people hold that it is wrong to cause animals unnecessary suffering. Opinions will differ as to what counts as necessary. . . . But *all will agree that gratuitous suffering—suffering caused for no good reason—is wrong*" (Carruthers 1992, 8; emphasis mine). These sentiments are not new. In 1813, Le Gallois endorsed (P2): "I own that it would be barbarous to make animals suffer in vain, if the object of the experiment could be obtained without it" (Le Gallois 1813, 20). Thus, even these prominent animal experimentation advocates are on record acknowledging that we owe conscious sentient animals a non-negligible amount of direct moral consideration. How much consideration? At least this much: We cannot harm them, cause them to suffer, or kill them *for no good reason.* If we do harm them, kill them, or cause them to suffer for no good reason, we are doing something morally wrong. We are failing to accord them the moral consideration that they are due. Since we all accept (P1)–(P3), we are all committed to the view that animals deserve at least this much moral consideration.

Principles (P1)–(P3) entail three additional principles directly related to the moral status of animal experimentation. For example, (P1) entails:

(P4) It is wrong to intentionally perform *harmful* experiments on conscious sentient animals *for no good reason.*

Principles (P2) and (P3) respectively entail the following principles:

(P5) It is wrong to perform *painful* experiments on conscious sentient animals *for no good reason.*

(P6) It is wrong to perform *lethal* experiments on conscious sentient animals *for no good reason.*

Anyone committed to (P1)–(P3) is, on pain of inconsistency, also committed to (P4)–(P6), since (P4)–(P6) are simply instantiations of (P1)–(P3), respectively. The relevance of (P4)–(P6) is this: virtually every biomedical experiment performed on animals causes harm to those animals. Many of these *harmful* experiments cause the animal subjects excruciating pain, and virtually all of these experiments are ultimately *lethal,* since the animals are routinely destroyed at the end of the experiment. So, the critical question is this: Is there a good reason to subject animals to these experiments? If not, all of these experiments are wrong and ought to be abolished.

II The Scientific Case against Using Animals in Biomedical Research

There are certain hallmarks of received wisdom. These empirical beliefs have worked their way into mainstream consciousness with no good supporting evidence, and yet, they occupy such a central position in our belief systems that we are loath to give them up. The belief that “milk does a body good” is such a belief. The belief that we need to experiment on animals in order to find cures for human diseases is another. Even people who have become sensitized to the plight of animals needlessly brutalized in factory farms and who have become vegetarians as a result still often think that some animal experimentation can be justified on the basis of its benefits to humanity. Using animals to test new drugs is sacrosanct. It is part of the medical and scientific orthodoxy. Why does almost everyone buy into this orthodoxy? They do so

- in part because the media constantly bombard us with animal research “successes”—for example, the media optimistically report that some drug X tested on rats promises to be a panacea for some horrific human disease, but fail to report when X is pulled from the market on the basis of failed clinical trials.
- in part because of scare tactics advanced by researchers and governmental propaganda, with comments like “If we didn’t test these drugs on animals, we’d have to test these drugs on humans, and wouldn’t that be terrible?” Not wanting to be guinea pigs ourselves, we happily embrace the orthodoxy. What we neglect to realize is that these drugs will be tested
animal subjects are forced to endure in LD50 tests is done for no good reason. Consequently, (P1)–(P6) entail that conducting LD50 tests on non-human animals is wrong and ought to be abolished. Anyone who accepts (P1)–(P6) is committed to the immorality of these tests. But what about other uses of nonhuman animals in scientific and biomedical research? Do we have any good reason to use animals in horrifically painful burn experiments to test potentially therapeutic ointments or drugs? Does medical advancement and treatment of human disease require using nonhuman animals in painful and ultimately lethal research? Do the human benefits of such research justify subjecting animals to painful, lethal biomedical experiments?

Animal experimentation advocates often try to manipulate us into providing affirmative answers to these questions via appeals to emotional intuition pumps like the following: (Q1) “If experimenting on 10,000 rats were the only way to save your child’s life, would you want the experiment to be conducted?” One can, of course, counter one intuition pump with another: (Q2) “If a stranger’s child’s life could be saved by performing a terribly painful experiment on your animal companion, would you allow the experiment to be performed on your beloved cat or dog?” I doubt many people would volunteer their own animal companions for such an experiment, even if a stranger’s child’s life were hanging in the balance.19

Now consider the following more salient questions:

(Q3) If your child would die from cancer because of animal research that caused the suffering and death of 10,000 rats, would you want that research on rats to be conducted?

(Q4) If your child would be born with birth defects directly as a result of research that involved the suffering and death of 10,000 rats, would you want that research on rats to be conducted?

Obviously, no parent would want an animal experiment to be conducted if its being conducted would result in her/his child’s death or deformity. And yet, many animal experiments have had exactly that result. For example, consider the oft-cited thalidomide tragedy. Thalidomide is teratogenic (i.e., causes birth defects) in humans. Moreover, thalidomide’s teratogenic effect in humans was recognized early on, on the basis of clinical observation—mothers who had taken thalidomide gave birth to babies without limbs. However, because thalidomide’s teratogenicity could not be readily reproduced in other animal species, thalidomide continued to be prescribed to pregnant women. Eventually, after testing thalidomide for teratogenicity in countless animal species, scientists were able to demonstrate a teratogenic effect in one breed of rabbit, but only at doses between 25 and 300 times

on humans anyway in phase I, phase II, and phase III clinical trials before they are approved by the FDA.

• in part because some claims made by animal experimentation advocates are true, like the claim: “Some questions can be answered only by animal research.” This claim is true. For example, if you wish to know how much of a given substance X will prove lethal to 50 percent of rats, you must test substance X on rats. Why? Because LD50 (Lethal Dose 50 percent) results vary from species to species. The amount of substance X that will prove lethal to 50 percent of rats won’t be the amount of substance X that will prove lethal to 50 percent of mouse subjects, nor will it be the amount of substance X that will prove lethal to 50 percent of chickens, cats, dogs, or humans. So, if you wish to know how much of substance X will prove lethal to rats, you must use rats. But why should anyone care how much of X proves lethal to 50 percent of rat subjects? This idiosyncratic information has no relevance to human health and well-being. It is no more useful than knowing whether the number of blades of grass in your front yard is odd or even. A real case will illustrate the point. Let X be the substance nicotine. Since some people still smoke tobacco, it is important to know the dose of nicotine that is lethal for humans. The lethal dose of nicotine in rats is 53 milligrams per kilogram of body weight. The lethal dose of nicotine in dogs is 9.2 mg/kg. Neither measure is remotely indicative of the dose of nicotine that is lethal in humans, namely, 0.9 mg/kg. Relying on the LD50 results of nicotine in rats (or dogs for that matter) to estimate the lethal dose of nicotine in humans would have had fatal results in humans. Nicotine is not unique in this regard. The lethal dose of mercury (II) chloride in rats and mice is 1 mg/kg and 6 mg/kg, respectively—six times greater in mice. But the lethal dose of paracetamol in rats and mice is 2400 mg/kg and 340 mg/kg, respectively—seven times greater in rats (PCRM 1999, 2).18 Thirty years ago, two leading toxicologists Zbinden and Flury-Roversi concluded: “For the recognition of the symptomatology of acute poisoning in man and for the determination of the human lethal dose, the LD50-test in animals is of very little value” (1981, 96). They further concluded: “The application of the LD50-test for the solution of pharmacokinetic problems must be regarded as one of the semi-quantitative pilot procedures that have no place in modern pharmaceutical and chemical research. The use of the LD50-test as basis for selection of doses for sub-acute and chronic toxicity tests and other procedures (e.g., teratogenicity, carcinogenicity studies) is obsolete” (ibid.).
that given humans. After still more testing, thalidomide was found to have a teratogenic effect in monkeys, but at ten times the normal human dose (Greek and Greek 2000, 45). The crucial point is this: There was a significant time lag—five years!—between the original reliable human clinical data that clearly demonstrated thalidomide's teratogenic effect in humans and scientists' ability to produce a similar teratogenic effect in some other species, a time lag during which thalidomide continued to be prescribed to pregnant women because it hadn't yet been found to be teratogenic in other species. The result: more than ten thousand babies were born without limbs because researchers depended on unreliable animal tests and ignored the more reliable human clinical data.20 Had thalidomide been pulled from the market on the basis of the human clinical data, these birth defects would not have occurred, but it wasn't pulled, pending "confirmation" in other species.

The thalidomide tragedy is a particularly telling illustration of the human costs of relying on misleading animal experiments. Animal experiments mislead in at least four ways along two distinct vectors—the safety vector and the efficacy vector. Consider first one particularly dangerous way animal experiments can mislead along the safety vector: Frequently, animal experiments mistakenly predict that a drug will be safe in humans (since it was found safe in animal models in preclinical testing), when in fact that drug is unsafe (i.e., toxic, teratogenic, or lethal) in humans. Such misleading results are called "false negatives," because the drugs test "negative" for harmful effects in animals, but are subsequently discovered to have seriously harmful effects in humans. Thalidomide is an example of a false negative, but it is hardly unique in this regard. Here is a partial list of false negatives and their harmful consequences for humans, as identified in Greek and Greek 2000:

- Diethylstilbestrol [DES]—animal models predicted that DES would prevent miscarriages; in humans DES caused spontaneous abortion, premature birth, and neonatal death (61).
- Zimeldine—the first selective serotonin reuptake inhibitor (SSRI), caused the paralyzing illness Guillain-Barré syndrome not predicted by animal tests (62).
- Isuprel—an asthma drug that is highly toxic to humans in the doses that were predicted to be safe for humans based on animal studies. Thirty-five hundred asthmatics died from the drug in Great Britain alone (63).
- Clioquinol—an antidiarrheal drug that tested safe in rats, cats, dogs, and rabbits, but caused blindness and paralysis in humans (67).
- Opren—an arthritis drug that tested safe on monkeys, but killed sixty-one humans (68).

C. Ray Greek, M.D., and Jean Swindle Greek, D.V.M., go on to identify at least forty other examples of false negatives. Several of these "animal-safe" drugs caused liver failure in humans, others caused seizures in humans, still others caused heart attacks in humans, and still others caused kidney failure or strokes or both in humans (Greek and Greek 2000, 61–68).

One other example—Vioxx—is worth highlighting. The Vioxx tragedy poignantly illustrates the magnitude of human harm that can result from relying on animal tests. Nine of 11 studies on mice and rats showed the COX-2 inhibitor Vioxx to be safe for animal hearts and blood vessels. In addition, six different animal studies involving four different species showed Vioxx to be protective against heart attacks and vascular disease (PCRM 2005). This animal-based data proved tragically misleading. In humans, Vioxx caused an estimated 320,000 heart attacks, strokes, and cases of heart failure worldwide—140,000 of them fatal—before being pulled from the market in 2004 (Anderegg 2006, 11).

Drugs that test safe and effective in animal models can also mislead along the efficacy vector. Drugs found effective in animal models often prove ineffective in humans. For example, twenty-two drugs have been shown to be therapeutic for spinal cord injury in animal models, but not a single one of these drugs is effective in humans (Greek and Greek 2004, 18). Call such results "false efficacy predictors." False efficacy predictors don't typically result in direct harm to humans; the drugs simply fail to work in humans. There are, however, indirect harms. The billions of research dollars used annually to develop drugs effective in animal models but ineffective in humans are wastefully being pumped into false leads and dead ends. These dollars could be far better spent on effective prevention campaigns.21

If false negatives and false efficacy predictors were relatively rare, say 1 out of 1,000 or even 1 out of 100, animal experimentation might still be justified on the basis of its benefits to humans, but false negatives and false efficacy predictors are not rare. In prepared remarks presented January 12, 2006, Andrew C. von Eschenbach, acting commissioner of the Food and Drug Administration, had this to say: "Consider just one stark statistic: Today, nine out of 10 compounds developed in the lab fail in human studies. They fail, in large part because they behave differently in people than they did in animal or laboratory tests" (2006, 1). Actually, the current failure rate of drugs that make their way to phase
I human clinical trials on the basis of preclinical animal testing is 92 percent. This clinical failure rate is split roughly equally between drugs that are too toxic and drugs that don't work in humans. Accordingly, of all the drugs that make their way to human clinical trials on the basis of preclinical animal testing, approximately 46 percent prove to be false negatives and roughly 46 percent fall into the category of false efficacy predictors, barely better than flipping a coin along either vector. With its 92 percent clinical failure rate, preclinical animal testing is an extraordinarily unreliable method of establishing the safety and effectiveness of pharmaceutical compounds in humans. With roughly half of these failures being false negatives, relying on animal models to establish the safety of drugs is also a dangerous way of testing drugs. It puts the human subjects in phase I, phase II, and phase III clinical trials at serious risk of grave harm or death.

Preclinical animal testing can mislead along the safety vector in the other direction, as well. Pharmaceutical compounds that are safe in humans can test unsafe (i.e., toxic, teratogenic, or fatal) in animals. Such test results are called "false positives," because the compounds test "positive" for harmful effects in animals, despite being safe in humans. False positives mistakenly suggest that a drug is unsafe for humans, when in fact that drug is perfectly safe in humans. As for the efficacy vector, drugs that are safe and effective in treating disease X in humans can be completely ineffective in treating X in animal models. Call these kinds of misleading results "false inefficacy predictors." By my lights, false positives and false inefficacy predictors are a far greater human cost of animal-based biomedical research than false negatives and false efficacy predictors, because, unlike the latter, false positives and false inefficacy predictors can and do result in drugs that are safe and effective in humans—life-saving panaceas for human diseases—being pulled from development before ever making it to human clinical trials. As long as we test potential drugs using unreliable animal models and table the development of drugs that might have proven safe and effective in humans on the basis of their deleterious effects in these animal models, we will inevitably continue to forego certain cures for human diseases. Your child, now dying of cancer, might very well have been saved by a drug that is both safe and effective in treating cancer in humans, but was shelved before making it to human clinical trials because it made mice and rats sick. Like their false negative counterparts, false positives are not rare. Some safe drugs in common human use that are now known to be examples of false positives include the following:

- Acetaminophen (Tylenol)—causes renal failure and death in cats in low doses.
- Ibuprofen (Advil, Motrin)—causes liver failure in dogs at low doses.
- Acetylsalicylic acid (aspirin)—is teratogenic in mice and rats, and causes blood abnormalities in cats.
- Depo-provera (a widely prescribed birth control pill)—causes cancer in dogs and baboons.
- Digitalis (a heart medication routinely used to treat congestive heart failure)—causes high blood pressure in animals.
- Streptomycin (a commonly prescribed antibiotic)—is teratogenic in rats.
- Prednisone (a widely prescribed corticosteroid)—causes cancer in some rodents.
- Cortisone (another regularly prescribed corticosteroid)—is teratogenic in mice.
- Fluoride (a mineral routinely added to toothpaste and tap water to strengthen teeth and help prevent cavities)—causes cancer in rats. (Greek and Greek 2000, 70–76)

One other example—penicillin—is worth noting because it illustrates so clearly just what is at stake. Isolating penicillin was probably the single greatest medical discovery of the twentieth century. With it, the age of antibiotics was born. Collectively, antibiotics have saved more lives than all other medical advances combined. Sir Alexander Fleming, who observed penicillin killing bacteria in Petri dishes, tested it on rabbits without ill effect. It was serendipitous, indeed, that he tested penicillin on rabbits rather than rodents. Penicillin is teratogenic in rats, and it kills guinea pigs and Syrian hamsters. Had Fleming tested penicillin on these rodent species, it probably would have never been approved for human use and the age of antibiotics might never have come into being.23 Luck—and luck alone—prevented penicillin from being a permanently shelved false positive. It's hard to imagine just how many human lives would have been lost had Fleming tested penicillin on these other species, but surely it's on the order of hundreds of millions of lives. No doubt, we haven't been so lucky with other life-saving pharmaceutical compounds that are now collecting dust in a laboratory somewhere, because they tested unsafe or ineffective in rodent species.

There is no easy way to measure just how many cures for human diseases have been shelved by pharmaceutical companies because the substance in question either proved ineffective in other species or made members of other species sick—partly because that information remains
proprietary and partly because drugs that test highly toxic in animal models rarely make it to human clinical trials—but there are good reasons to think that the number is far from negligible. For example, when the National Cancer Institute tested on mice twelve anticancer drugs currently being successfully used in humans, they found that thirty out of forty-eight times the drugs were ineffective in mice—a false inefficacy predictor rate of 63 percent (Greek and Greek 2004, 17). Animal models also prove terribly unreliable when it comes to testing substances for carcinogenicity. Of twenty compounds known not to cause cancer in humans, nineteen did cause cancer in animals—a false positive rate of 95 percent (ibid.). Couple this information with the fact that 98 percent of the compounds tested in preclinical animal trials are killed by pharmaceutical companies before making their way to human clinical trials, largely on the basis of animal data (Pippin 2007, 2), and one can start to see the magnitude of the risk to human health posed by reliance on unreliable animal models. For every six hundred drugs that enter preclinical testing on animals, only twelve advance to human clinical trials, and only one of these twelve receives FDA approval (ibid.). That means for every one FDA-approved drug, 588 drugs are pulled from development by pharmaceutical companies without ever being tested on humans. Some of these drugs are pulled because of toxicity in animal models. Some of these drugs are pulled because the compounds are ineffective in animals. Of the 98 percent of chemical compounds that are discarded by pharmaceutical companies due to their toxicity or ineffectiveness in animal models, we will never know how many would have proven safe and effective in humans, but again, there are good reasons to think that the number is probably quite large.

Consider another such reason: Occasionally, drugs that test toxic in animals make their way to human clinical trials anyway (cases where the animal data is simply being ignored). In August 2001, Mark Levin, CEO of Millennium Pharmaceuticals, presented data at the Drug Discovery Technology Conference in Boston that suggests just how prevalent false positives are. In the study that Levin presented, twenty-eight potential new drugs were tested in rats for hepatotoxicity (i.e., liver toxicity). Seventeen of these drugs tested nonhepatotoxic in rats, and eleven tested hepatotoxic in rats. Twenty-two of these drugs advanced to human clinical trials anyway—fourteen of the seventeen that tested safe and eight of the eleven that tested positive for hepatotoxicity advanced. Of the eight that had tested hepatotoxic in rats, six were found safe (i.e., nonhepatotoxic) in humans (Greek and Greek 2004, 17–18): a false positive rate of 75 percent! While it's unlikely that 75 percent of the 98 percent of discarded drugs would have proven safe and effective in humans (since the tests reported by Levin focused solely on one form of toxicity), given the large number of false positives of which we are aware, it is quite likely that a significant percentage of the discarded drugs would have proven safe and effective in humans, had we not relied on misleading animal data. We will never have access to those drugs, because we have relied on misleading animal data. All those people whose diseases would have been cured or ameliorated by those drugs unfortunately must continue to suffer from their illnesses because of our reliance on animal-based biomedical research. In these cases, far from making us better, the animal research is keeping us sick.

III What Else Can We Do?

Still, you might wonder, what's the alternative to animal experimentation? We need some way of assessing how a pharmacokinetic agent will behave in humans before we administer it to humans in Phase I clinical trials, and animal testing seems to be the only game in town. Even though animal experimentation is an unreliable—indeed, often malpredictive—way of assessing the likely effects of pharmacological agents in humans, you might think that it's better than nothing.

Two points are in order: First, given how unreliable animal experiments have proven to be at predicting human response to pharmacological compounds, if animal experimentation were the only game in town, we would be wise to stop playing because, as we have just seen, the animal experimentation game is enormously costly to human health. It is directly responsible for the death or deformity of hundreds of thousands of humans and is also directly responsible for our not having all those curative and ameliorative drugs that would have proven safe and effective in humans, but were too toxic or ineffective in animal models to advance to human clinical trials.

Second and more important, animal experimentation is not the only game in town. Scientists are now able to construct, maintain, and analyze complex human-tissue cultures and cell layers in vitro. Testing a pharmacokinetic agent directly on human tissue in vitro provides important information about how that pharmacological compound will be absorbed and metabolized by human tissue. In vitro techniques, which were proven to be superior to animal studies at predicting human response over a decade ago, are now being used to identify disease mechanisms, drug targets, drug efficacy, and drug toxicity in virtually all types of human tissue (Pippin 2005, 17). Human stem cells may also be used to test the toxicities and efficacies
of chemical compounds and pharmacological agents (ibid.), and once again, the results are specific to humans. Pharmacological compounds can also be tested using computer and mathematical models based on existing clinical knowledge. These in silico tests provide important absorption, distribution, metabolism, excretion, and toxicity (ADMET) data that allow us to predict how a new compound is likely to behave in humans. In silico technology provides human ADMET predictions whose accuracy rivals that of in vitro methods (ibid., 16). Unlike animal models, all of the alternatives just mentioned provide information specific to humans.

Animal experimentation advocates may well acknowledge the scientific validity and value of human-tissue in vitro testing, human stem-cell research, and in silico testing, and may even encourage the use of these tests, but they will still likely object as follows: "Sometimes drugs behave differently in intact organisms than they do in isolated tissue cultures. Before a drug can be released to the public, it must be tested on intact organisms. If we did not first test pharmacokinetic agents on animals, we would have to test them directly on humans in order to discover how they will react in intact organisms, and that would be unconscionable."

It is true that if we don’t first test drugs on intact animals, then the first intact organisms in which the drugs will be tested will be human beings in phase 0 trials; but such tests can be performed safely using microdosing technology. As John Pippin, M.D., explains, microdosing technology uses radiolabeled trace doses (1–100 mcg) of candidate drugs to evaluate absorption, distribution, metabolism, and excretion in humans. These doses are less than 1 percent of that required to produce a pharmacological effect, thus there is virtually no risk for adverse effects. The radiation exposure is less than that obtained in a four-hour airplane flight. Positron emission tomography is used to acquire real-time data regarding drug disposition, and accelerator mass spectrometry is used to analyze parent drug and metabolite concentrations in blood, urine, and feces at specific intervals after dosing. (2005, 16)

Microdosing technology can provide us with accurate information as to how a potential drug will be absorbed, distributed, metabolized, and excreted in humans—something that no animal test can do. Is it safe? Yes. Microdosing technology "was endorsed by the European Agency for the Evaluation of Medicinal Products in January 2003 . . . , and has already been used to identify drug candidates for human phase I trials" (Pippin 2005, 16). Thus, not only are there alternatives to testing drugs in animal models, there are better, more accurate alternatives.

The bottom line: the point of preclinical testing is to assess likely human response to a candidate drug before administering therapeutic doses of that drug to humans in phase I clinical trials. Animal tests have little or no relationship to human pharmacology and, thus, cannot provide us with that important information whereas phase 0 microdose testing in humans can. Therefore, we should eliminate animal tests, which are both unhelpful and unnecessary, and replace them with more accurate, more informative microdose tests in humans.

IV Conclusion

Even if one only cared about humans and their well-being, one would still have good reason to oppose using animals in biomedical research. As we have just seen, the cost to human health of relying on unreliable animal models is staggering. First, drugs highly toxic and even lethal in humans routinely test safe in other animal species. The life-threatening problems posed by these false negatives are often exacerbated by the fact that, because of the extensive animal data, human clinical trials are often cursory and brief. The drug is subsequently released to the public at large and because of poor post-release surveillance, it often takes years to recognize the drug’s toxicity in humans, especially when that toxicity can’t easily be demonstrated in other species. By then thousands of humans may have been permanently harmed, if not killed, by the drug. Second, drugs that would be safe and effective in humans routinely test toxic or ineffective in animals, and as a result, never make it to those human populations that so desperately need them. Had we not relied on misleading animal testing, many of these drugs would have advanced to human clinical trials, where their safety and effectiveness would have been demonstrated. Animal-based research is directly responsible for our not having those drugs. And third, there are safe, effective, more accurate alternatives to testing drugs in unreliable, malpredictive animal models, including human tissue in vitro tests, in silico tests, human stem cell research, and microdose tests in humans—alternatives which provide us with reliable information about a candidate drug’s absorption, distribution, metabolism, excretion, and toxicity in humans. Unlike animal experiments, which have virtually no relationship to human pharmacology, these tests specifically address human response to newly developed pharmacokinetic agents.

So, again, if we only cared about humans, we would have excellent reasons for opposing animal-based biomedical research—but we don’t only care about humans. We also care about animals. We accept (P1)–(P6) and think that animals should not be harmed or killed for no good reason. There is no good reason to use a research protocol as notoriously
unreliable as animal-based biomedical research. Indeed, there are good reasons not to use animals as models in biomedical research: (1) People are harmed and even killed because of misleading false negative results in animal models; (2) people are forced to forego curative, even life-saving, drugs because of misleading false positive and false inefficacy results in animal models; and (3) there are safe alternative methods of predicting human response to candidate drugs, the results of which are more accurate and more informative than those of outmoded animal experiments. Since there is no good reason to perform biomedical experiments on animals and good reason not to, (P1)–(P6) entail that these experiments are wrong and ought to be abolished. This conclusion is not predicated on some highly contentious moral theory that one can easily reject, but rather on beliefs central to and constitutive of our conception of morality—beliefs that we all share, namely, (P1)–(P6). Moreover, this conclusion follows, regardless of one’s views on speciesism, animal equality, and animal rights. Even those readers sincerely committed to valuing humans over nonhumans are committed to the immorality of using animals in biomedical experiments, given their commitment to (P1)–(P6). Consequently, consistency with our other beliefs demands that we acknowledge the immorality of these experiments and work to bring them to an end.

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Notes

1. Singer laid the groundwork for his utilitarian case against animal experimentation in his 1974 article, “All Animals Are Equal,” and then developed the argument in much greater detail in his later work (Singer 1993, 2002).

2. Regan’s rights-based argument against animal experimentation is spelled out in his 1987 article, “Ill Gotten Gains,” and then developed much more fully in later works (Regan 2001, 2003, 2004).

3. According to preference utilitarianism, act X is right for agent A if and only if, out of all the actions available to A, act X maximizes the satisfaction of the interests and desires (i.e., the preferences) of all those beings affected by the action. Thus, the preference utilitarian maintains that an action is right if no other action has a better balance of preference satisfaction.

4. In addition to its speciousness and invalidity, Vance’s response suffers from a number of other devastating weaknesses. Consider just two. Weakness 1: Vance instructs us to draw on canonical texts to assess the moral status of animal experimentation, but it is not at all clear what canonical texts Vance has in mind. Perhaps he thinks we should look to the Bible (Isaiah 66:3), which teaches: “He that killeth an ox is as if he slew a man; he that sacrificeth a lamb, as if he cut off a dog’s neck; he that offereth an oblation, as if he offered swine’s blood.” Not much support for animal experimentation there. Perhaps he thinks we should look to other canonical texts. In the Hadith Mishkat (Book 6, Chapter 7, verse 8: 178), Mohammed teaches: “A good deed done to an animal is as meritorious as a good deed done to a human being, while an act of cruelty to an animal is a bad as an act of cruelty to a human being.” The Hindu Bhagavad Gita (verse 5.18) proclaims that a self-realized soul is able to understand the equality of all beings. The First Precept of Buddhist ethical conduct is to not harm sentient beings. Nothing thin and abstract here: all of these passages maintain that we owe animals moral consideration equal to that owed humans and that we should not harm animals. Giving animals equal consideration and refraining from harming them would require bringing an end to almost all animal experimentation. Weakness 2: Since everything follows from a contradiction, drawing on “contradictory platitudes” cannot help us determine anything. It certainly cannot help us determine whether or not animal experimentation is morally permissible. No tool in the analytic philosopher’s toolbox is as dull as Vance’s appeal to contradictory platitudes.

If the reasons Vance offers in support of animal experimentation are the best reasons the animal experimentation advocate can offer, then the animal experimentation advocate’s position is hardly justified.

5. Speciesism is the widespread view that our species is superior to and more valuable than the other species and that, therefore, members of our species have the right to dominate and exploit members of these other species. While the word “speciesism” and its cognates are often used pejoratively in the animal rights literature, I use them only descriptively.

6. For a similar consistency argument in support of ethical vegetarianism, see Engel 2000.

7. If an interlocutor doesn’t initially accept one of the argument’s premises, the arguer must offer an independent argument for the premise in question. If that new argument contains premises that the interlocutor doesn’t accept, these premises will also have to be defended. This process must continue until the arguer and the interlocutor reach a common ground of accepted premises. Until such common ground is reached, the arguer will fail to convince the interlocutor of the truth of
the original conclusion. Consequently, one can argue much more effectively if one
starts with premises one's audience already shares.
8. Bonnie Steinbock's (1978, 255–256) criticism of Singer's view seems to be
rooted in such a sincerely held feeling.
9. Do we need the latest highly caustic oven cleaner when steel wool works just
as well? Must we have the latest "new and improved" soap scum remover, toilet
bowl cleaner, tire cleaner, dashboard preservative, floor wax, or shampoo, when
the market is already flooded with such products? Can we lead meaningful lives
without the latest aftershave, perfume, or feminine deodorant spray? The fact is
that none of these products are necessary for humans to live meaningful, enjoyable
lives, and certainly "new" versions of these products aren't necessary.
10. We also accept the following related principles:
   (P1*) It is wrong to intentionally harm conscious sentient animals unnecessarily.
   (P2*) It is wrong to cause conscious sentient animals to suffer unnecessarily.
   (P3*) It is wrong to kill conscious sentient animals unnecessarily.
Strictly speaking, (P1*)–(P3*) are not equivalent to (P1)–(P3), respectively,
because there might be a good reason to perform a certain action that strictly
speaking isn't necessary for some significant human benefit. Suppose X and Y are
equally effective means to achieving some important end E. Then, strictly speak-
ing, performing X is not necessary to bring about E, since we might perform
Y instead. Still, if performing X costs considerably less than performing Y, we
might have a good reason to perform X to bring about E. Conversely, the fact
performing an action A is necessary for bringing about a certain valuable end E
doesn't always give us a good reason to perform A. Suppose the only way I can
save my son's life is to kill you and harvest your heart and lungs (suppose you are
the only tissue match). In the scenario just imagined, killing you is necessary to
save my son's life, but that doesn't give me a good reason to kill you. I still would
not be justified in killing you. Even though necessity and having good reasons can
pull apart in these ways, they typically go hand in hand. Typically, when perform-
ing an action is necessary to bring about a significant human benefit that will give
us a good reason to perform it; and more important for present purposes, typi-
cally, when there is no good reason to perform an action, performing that action
will not be necessary for some significant human benefit. Accordingly, I will treat
(P1*)–(P3*) as roughly equivalent to (P1)–(P3), respectively, because nothing in
the present chapter will turn on the subtle sorts of situations where necessity and
the having of good reasons pull apart.
11. As I have already noted, these principles are central to our understanding of
morality. Together they specify an important part of the underived conceptual role
of the concept of moral wrongness. By way of illustration, consider the following
much discussed example from Gilbert Harman: "If you round the corner and see
a group of young hoodlums pour gasoline on a cat and ignite it, you do not need to
conclude that what they are doing is wrong; you do not need to figure anything
out; you can see that it is wrong" (1977, 4). Harman offered the example to show
that some moral judgments are direct, as opposed to inferential. What is relevant
about Harman's example for present purposes is this: No one seriously doubts that
burning a cat to death for no good reason is wrong. Treating a cat in such a way,
causes the cat harm, suffering, and death for no good reason, and we all judge
such conduct to be immoral. For a more recent nonfictional example, consider the
public outrage that erupted when it was revealed that professional football player
Michael Vick was guilty of sponsoring dog-fighting rings in which pit-bulls were
forced to rip each other apart in brutal fights to the death. As with Harman's cat,
we are outraged that someone would cause these dogs such harm, suffering, and
death for no good reason, and we view those people who would engage in such
castigate as morally deficient or depraved, or both. These examples illustrate that
principles (P1)–(P3) are partially constitutive of the very concept of moral wrong-
ness, and they confirm that no one seriously doubts (P1)–(P3).
12. To see Cohen's commitment to (P2) here, we need only recognize that justifica-
tion proceeds in terms of reasons. We are justified in causing an animal pain if and
only if we have a good reason for doing so. If there is no good reason to cause an
animal pain, then causing that animal pain cannot be justified.
13. Here, strictly speaking, Cohen commits himself to (P2*) and (P3*). See note
10 for details.
14. For further discussion of the moral ramifications of acknowledging that ani-
mal rights deserve non-negligible direct moral consideration, see Engel 2001.
15. Here is why (P1) entails (P4): First, mere laboratory confinement itself is
so stressful for the animals as to be properly regarded as a psychological harm
(see Balcombe 2004, 6–8). Second, the animals experimented on are virtually
always intentionally harmed in some physical way. Some animals are intention-
ally infected with pathogens. Other animals have diseases their own organs
are intentionally induced, including artificially induced coronary artery disease,
artificially induced strokes, and artificially induced cancers. Still other animals
are irradiated or burned or maimed in other ways, such as intentionally
induced spinal cord injuries and intentional limb amputations. Conducting these
experiments requires, by its very nature, intentionally harming the animal subjects
involved, and the researchers involved are fully aware of that fact. Since, per
(P1), it is wrong to intentionally harm a conscious sentient animal for no good
reason, (P1) entails that it is wrong to intentionally conduct harmful experiments on
conscious sentient animals for no good reason, which is just what (P4) as-
serts. Principles (P5) and (P6) follow from (P2) and (P3), respectively, in equally
straightforward ways.
16. See the previous note for a cataloguing of just some of these harms.
17. Many people unquestioningly believe that drinking cow's milk is healthful
and wholesome despite the fact that (1) no other mammalian species drinks milk
past the age of weaning, (2) no other mammalian species drinks the milk of other
species, (3) the majority of African Americans, Asians, and Hispanics are lactose
intolerant, and (4) less than 40 percent of humans retain the ability to digest
lactose after childhood.
18. Here are a few more examples of the radical differences in LD50 results in rats
and mice. In rats, the lethal dose of carbon tetrachloride is 2350 mg/kg, but in mice
it is 8260 mg/kg—3.5 times greater in mice. The lethal dose of dichloromethane in
rats is 1600 mg/kg; in mice it is 873 mg/kg—1.8 times greater in rats. The lethal dose of dextropropoxyphene HCl in rats and mice is 84 mg/kg and 225 mg/kg, respectively—2.7 times greater in mice; whereas the lethal dose of diphenylhydantoin in rats is 1640 mg/kg; but in mice is only 150 mg/kg—10.9 times greater in rats (PCRM 1999, 2). As these examples further illustrate, one cannot reliably extrapolate information about the lethal dose in rats from LD50 results in mice, or vice versa. With such radical differences in lethal dose between species as similar as rats and mice, extrapolation of human lethal dose from rat and mice LD50s is not only unreliable, it's meaningless.

19. Those who think that most people would volunteer their animal companions for use in a painful, lethal experiment in order to save the life of a stranger's child should consider the following: Each year animal guardians spend hundreds, if not thousands, of dollars on food, litter, basic veterinary care, bedding, and toys for their animal companions. These people could have their animal companions put to sleep and they could then send the hundreds of dollars saved on pet care to OXFAM, UNICEF, or CARE where it would save the lives of numerous children each year; but they would never think of doing so.

20. Greek and Greek give the following poignant description of the thalidomide tragedy:

A German pediatrician named Widikund Lenz was the first to suggest a link between thalidomide and teratogenesis. . . . Mothers who had taken thalidomide gave birth to babies with often shocking deformities. Most lacked developed limbs. The first recorded case of phocomelia secondary to thalidomide occurred on Christmas Day, 1936, but in 1957 the drug was released anyway. A clinician from Australia, W. G. McBride, confirmed thalidomide's dangers. Alarmed, he, Lenz, and others wrote letters to the distinguished medical publication The Lancet, reporting phocomelia in infants of mothers taking thalidomide.

As the incidences of deformity increased, scientists frantically attempted to reproduce teratogenesis from thalidomide in animals of all varieties. They gave thalidomide to scores of animals looking for proof in animals of what they already knew occurred in humans—that thalidomide could cross the placenta and drastically damage unborn offspring—and they could find none. Since animal testing had not indicated a problem with thalidomide, its use persisted. Hence, animal testing delayed the recall of this highly teratogenetic drug. (2000, 45)

21. Just how much money are we talking about? Lester Crawford, acting commissioner of the Food and Drug Administration in 2004, estimates that it takes a staggering $1.7 billion to produce one FDA-approved drug (Crawford 2004, 2).

22. In prepared remarks, Lester Crawford, acting commissioner of the Food and Drug Administration in 2004, reported that only 8 percent of the drugs that test safe and effective in animals, prove safe and effective in humans (Crawford 2004, 2). Also see Pippin 2007.

23. Fleming himself made a similar observation: “How fortunate we didn’t have these animal tests in the 1940s, for penicillin would probably never have been granted a licence, and possibly the whole field of antibiotics might never have been realised” (Parke 1994, 208).

24. Of the fourteen drugs that tested safe (i.e., nonhepatotoxic) in rats that were subsequently tested in humans, six proved hepatotoxic in humans—a false negative rate of 43 percent (Greek and Greek 2004, 17–18).

25. Cohen offers just such an objection:

The results of experiments using tissue samples, however favorable, will not be enough to warrant use of a new drug in humans until we have done our best to learn its full organic impact. . . . The first use of a new compound on a living organism is inescapably experimental. The subject of that experiment will be a human or another animal. The use of humans in such experiments we will not permit, understandably. If, therefore, the use of nonhuman animals is also not permitted, there will be no such experiments. (2001, 13)

References


14

Nathan Nobis

Morality is the effort to guide one’s conduct by reason—that is, to do what there are the best reasons for doing.

—James Rachels

A Methodological Focus

Synopsis: A focus of this chapter is on how to evaluate reasons for and against moral positions.

This chapter is designed to help people rationally engage moral issues regarding the treatment of animals, specifically in experimentation, research, product testing, and education.

Little “new” philosophy is offered here, strictly speaking. New arguments are unnecessary to help make progress in how people think about these issues. What is needed are improved abilities to engage the arguments already on the table, for example, stronger skills at identifying and evaluating the existing reasons given for and against conclusions on the morality of various uses of animals.

To help improve these abilities, this chapter sets forth a set of basic but powerful “logical skills” for rationally evaluating arguments. These skills emerge from reflection on some historical moral issues: an argument in defense of slavery, an argument against women being educated, and, as a nonhistorical case, an argument in favor of eating meat. These skills help us see these arguments’ exact faults. And they are generally useful, for being applicable to any moral issue.

Dozens of common moral defenses of animal use are then evaluated using these skills. This application reveals that all of these arguments are unsound: they all have either false premises (and most animal experimenters agree, since they reject these premises) or premises that are in need of rational defense: questions about them need answers; and objections


