The Immorality of Biomedical Animal Experimentation

Most arguments for the immorality of animal experimentation [AE] take one of two forms: Either they follow Peter Singer's lead and maintain that most animal experiments are morally unjustifiable on utilitarian grounds;[1] or they follow Tom Regan's deontological rights-based approach and insist that the animals being experimented on possess the very same rights-conferring properties which confer rights on humans and that, therefore, experimenting on these animals is wrong because it violates their rights.[2] When confronted with such arguments, proponents of AE tend to casually dismiss them by rejecting the ethical theories on which they are predicated. Consider two examples. In an effort to defend AE, Carl Cohen goes to great lengths to try to show, *contra* Regan, that nonhuman animals lack rights and that, therefore, it's permissible to experiment on them.[3] Richard Vance rejects Singer's and Regan's arguments for the immorality of AE because he rejects the analytical ethical tradition on which they are based. As Vance sees it, Singer's and Regan's arguments against AE 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) platitudes, interpretative communities, and the like. In comparison with such traditions, analytical ethics is abstract and thin…, no analytical model has been able to claim adequacy."[4]

A moment's reflection reveals the sophistry of such replies. Since no ethical theory to date is immune to objection, one could fashion a similar reply to "justify" virtually any behavior. One could "justify" slavery as follows: An opponent of slavery might appeal to utilitarian or deontological grounds to establish the immorality of slavery. Our fictitious slavery-proponent could point out that these ethical theories are flawed and *ipso facto* so too is any argument based on these theories. 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."[5]

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's 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. I aim to block this spurious reply by providing an argument for the

immorality of AE that does not rest on any particular highly-contentious ethical theory. Rather, it rests on commonsense moral beliefs that we all share. The significance of this argumentative strategy is this: All effective argumentation must start with premises one's interlocutor accepts.[6] The reason Singer's and Regan's arguments sometimes fall on deaf ears is because 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.

Although animals are used in all sorts of scientific research, including product testing and psychological experimentation, I will focus exclusively on the use of animals in biomedical research, for if it's wrong to use animals in experiments aimed at developing vitally important, life-saving drugs, it's wrong to use animals to test trivial products like a new floor wax or shampoo. Having clarified the scope, significance, and rationale for my argumentative strategy, I now turn to the argument itself.

1. 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 which 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*.[7]

These principles are not in dispute.[8] Even the staunchest defenders of AE embrace these commonsense principles. For example, Cohen explicitly endorses (P2) and (P3): "If animals feel pain, *we humans surely ought cause no pain to them that cannot be justified. Nor ought we kill them without reason*."[9] Elsewhere, Cohen writes: "Our obligations to animals arise not from their rights, 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."[10] Peter Carruthers also explicitly endorses (P2):

> . . . it will be useful to have a rough idea at the out-set of what our common-sense morality tells us about the status and appropriate treatment of animals. . . . Most 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*.[11]

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."[12] Thus, even these prominent AE-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.[13]

Principles (P1)-(P3) entail three additional principles directly related to the moral status of AE:

(P4) It is wrong to intentionally perform *harmful* experiments on conscious sentient animals *for no good reason*.[14]
(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.[15] Many of these experiments cause the animal subjects *severe pain*, and virtually all of them 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.

2. The 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 loathe to give them up. The belief that we need to experiment on animals in order to find cures for human diseases is such a belief.[16] 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 AE 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?

- Partly because the media constantly bombard us with animal research "successes" – 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.
- Partly because of scare tactics advanced by researchers and governmental propaganda: "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 on humans anyway in clinical trials before they are approved by the FDA.
- Partly because some claims made by AE-advocates are true, like the claim: "Some questions can be answered *only* by animal research." E.g., if you wish to know how much of a given substance *X* will prove lethal to 50% of rats, you must test *X* on rats. Why? Because LD50 [Lethal Dose 50%] results vary from species to species. The amount of *X* that will prove lethal to 50% of rats won't be the amount of *X* that will prove lethal to 50% of mouse subjects, nor will it be the amount of *X* that proves lethal to cats, dogs, or humans. So, if you wish to know how much *X* is lethal to rats, you must use rats. But why should anyone care how much *X* is lethal to rats? This idiosyncratic information has no relevance to human health and well being. Consider 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 mg/kg 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 to estimate the lethal dose of nicotine in humans would have had fatal results in humans. Nicotine is not unique in this regard.[17] In 1981, 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 is of very little use."[18]

Since LD50 tests provide no useful lethal-dose information that can be reliably extrapolated to human beings, all the pain the animal subjects are forced to endure in LD50 tests is done *for no good reason*. Consequently, (P1)-(P6) entail that conducting LD50 tests on 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? 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?

AE-advocates often try to manipulate us into providing affirmative answers to these questions via

emotional appeals 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] Consider two 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. Consider the 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 teratogenecity could not be readily reproduced in other species, thalidomide continued to be prescribed to pregnant women. Eventually, after testing thalidomide for teratogenecity in countless species, scientists were able to demonstrate a teratogenic effect in one breed of rabbit, but only at doses 25 to 300 times that given humans.[20] After still more testing, thalidomide was found to have a teratogenic effect in monkeys, but at ten times the normal human dose.[21] The crucial point is this: There was a significant lag-time—5 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 another species, a lag-time 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: Over 10,000 babies were born without limbs *because* researchers relied on unreliable animal tests and ignored the more reliable human clinical data.[22] 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 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 one 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's hardly unique in this regard. Here's a partial list of false negatives and their harmful consequences for humans:

- Diethylstilbesterol [DES] – animal models predicted that DES would prevent miscarriages; in humans DES caused spontaneous abortion, premature birth, and neonatal death.
- Zimeldine – the first SSRI, caused paralyzing Guillain-Barre syndrome in humans not predicted by animal tests.
- Isuprel – an asthma drug that is highly toxic to humans in the doses predicted safe by animal studies. Thirty-five hundred asthmatics died from the drug in Great Britain alone.
- Clioquinol – tested safe in rats, cats, dogs, and rabbits, but caused blindness and paralysis in humans.
- Opren – an arthritis drug that tested safe on monkeys, but killed sixty-one humans.[23]

Greek and Greek identify over 40 other examples of false negatives.[24] Several of these animal-safe drugs caused liver failure in humans, others caused seizures in humans, and still others caused heart attacks, kidney failure, and/or strokes in humans. 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.[25] 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.[26]

Animal models can also mislead along the efficacy vector. Drugs found *effective* in animals often prove *ineffective* in humans. For example, 22 drugs have been shown to be therapeutic for spinal cord injury in animals, but not one of these drugs is effective in humans.[27] Call such results "False Efficacy Predictors." False efficacy predictors don't usually 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 animals, 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.[28]

If false negatives and false efficacy predictors were relatively rare, say 1 out of 1000 or even 1 out of 100, AE might still be justified on the basis of its benefits to humans, but false negatives and false efficacy predictors aren't rare. On January 12, 2006, the Acting Commissioner of the FDA, 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."[29] 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%.[30] 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% prove to be false negatives and roughly 46% fall into the category of false efficacy predictors, barely better than flipping a coin along either vector. With its 92% 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 clinical trials at serious risk of grave harm and/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 *D* in humans can be completely *ineffective* in treating *D* in animals. 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—lifesaving 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 have been saved by a drug that is both safe and effective in treating cancer in humans that 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 examples of what are now known to be false positives include: acetaminophen (causes renal failure and death in cats in low doses), ibuprofen (causes liver failure in dogs at low doses), aspirin (teratogenic in mice and rats, and causes blood abnormalities in cats), depo-provera (causes cancer in dogs and baboons), digitalis (causes high blood pressure in animals), streptomycin (teratogenic in rats), prednisone (causes cancer in some rodents), cortisone (teratogenic in mice), and fluoride (causes cancer in rats).[31] 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 20th century. With it, the age of antibiotics was born. Collectively, antibiotics have saved more lives than all other medical advances combined. Alexander Fleming who observed penicillin kill 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.[32] 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 its on the order of hundreds of millions of lives. No doubt, we haven't been so lucky with other lifesaving pharmaceutical compounds that are now collecting dust in laboratories 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 drug in question either proved ineffective or toxic in other species,[33] but there are good reasons to think that the number is far from negligible. When the National Cancer Institute tested on mice 12 anti-cancer drugs currently being successfully used in humans, they

found that 30 out of 48 times the drugs were ineffective in mice—a false inefficacy predictor rate of 63%.[34] Animal models also prove very unreliable, when it comes to testing substances for carcinogenicity. Of 20 compounds known *not* to cause cancer in humans, 19 *did* cause cancer in animals—a false positive rate of 95%.[35] Couple this information with the fact that 98% 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,[36] and one can start to see the magnitude of the risk to human health posed by reliance on unreliable animal models. For every 600 drugs that enter preclinical testing on animals, only 12 advance to human clinical trials, and only 1 of these 12 receives FDA approval. That means for every 1 FDA-approved drug, 588 drugs are pulled from development *without ever being tested on humans.* Some of these drugs are pulled because of toxicity in animal models. Others are pulled because the compounds are ineffective in animals. Of the 98% 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 there are reasons to think that the number is quite large.

Consider one 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 that suggest just how prevalent false positives are. In the study that Levin presented, 28 potential new drugs were tested in rats for hepatotoxicity (i.e., liver toxicity). Seventeen of these drugs tested non-hepatotoxic in rats, and 11 tested hepatotoxic in rats. Twenty-two of these drugs advanced to human clinical trials anyway—14 of the 17 that tested safe and 8 of the 11 that tested positive for hepatotoxicity advanced. Of the 8 that had tested hepatotoxic in rats, 6 were found safe (i.e., non-hepatotoxic) in humans. A false positive rate of 75%![37] While it's unlikely that 75% of the 98% 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's 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 who 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.

The bottom line: Even if one only cared about humans, one would still have good reason to oppose using animals in biomedical research. The cost to human health of relying on unreliable animal models is staggering. 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 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 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. Plus, drugs that *would be* safe and effective in humans routinely test toxic or ineffective in animals. These drugs never make it to those human populations who 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.

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 think that animals should not be harmed or killed *for no good reason*, i.e., we accept (P1)-(P6). 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, and (2) people are forced to forego curative drugs because of misleading false positive and false inefficacy results in animal models. 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). Even those who value 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.[38]

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[1] See Singer, 2002; or his "All Animals are Equal" in Regan and Singer, 1989, 73-86.

[2] See Regan, 2004; Regan, 2003; or Regan's "The Case for Animals Rights" in Singer, 1985, 13-26.

[3] As if the latter followed from the former. See Cohen, 2001a and 2001b.

[4] Vance, 1992, 1715.

[5] Vance's response suffers from other devastating weaknesses, as well. 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, Ch. 7, 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 not to 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 AE are the best reasons the AE-advocate can offer, then the AE-advocate's position is hardly justified.

[6] 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.

[7] 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 both *X* and *Y* are an equally effective means to achieving some important end *E*. Then, strictly speaking, 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, e.g., 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 performing an action is necessary for bringing about a significant human benefit that will give us a good reason to perform it; and more importantly for present purposes, typically, 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 paper will turn on the subtle sorts of situations where necessity and the having of good reasons pull apart.

[8] 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" (Harman, 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 non-fictional 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 fight 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 conduct as morally deficient and/or depraved. These examples illustrate that principles (P1) – (P3) are partially constitutive of the very

concept of *moral wrongness*, and they confirm that no one seriously doubts (P1) – (P3).

[9] Cohen, 2001a, 46. To see Cohen's commitment to (P2) here, we need only recognize that justification 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.

[10] Ibid., 226. Strictly speaking, Cohen commits himself to (P2*) and (P3*). See footnote 46 for details.

[11] Carruthers, 1992, 8.

[12] Le Gallois, 1813, 19-21.

[13] For a further discussion of the moral ramifications of acknowledging that animals deserve non-negligible direct moral consideration, see Engel, 2001.

[14] 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 intentionally infected with pathogens. Other animals have diseases thought to model human diseases 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 just is what (P4) asserts. Principles (P5) and (P6) follow from (P2) and (P3), respectively, in equally straightforward ways.

[15] See the previous note for a cataloguing of just some of these harms.

[16] The belief that "Milk does a body good" is another such a belief, despite the fact that no other mammalian species drinks milk past the age of weaning and no other mammalian species drinks the milk of other species.

[17] For example, 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. Greek and Greek, 2004, 148.

[18] Zbinden and Flury-Roversi, 1981.

[19] Those who think that most people would volunteer their animal companions 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, etc. for their animal companions. These people could have their animal companions put to sleep and 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, 2000, 45.

[21] Ibid.

[22] Greek and Greek (2000, 45) 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, 1956, but in 1957 the drug was released anyway. A clinician from Australia, W.G. M^{c}Bride, 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.

[23] Greek and Greek, 2000.

[24] Greek and Greek, 2000: 61-68.

[25] "Animal Research on Trial," *Good Medicine* 14, Autumn 2005: 13.

[26] Anderegg, 2006, 11.

[27] Greek and Greek, 2004, 18.

[28] 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).

[29] Eschenbach, 2006, 1.

[30] In prepared remarks, Lester Crawford, Acting Commissioner of the Food and Drug Administration in 2004, reported that only 8% of the drugs that test safe and effective in animals, prove safe and effective in humans. (Crawford, 2004, 2). Also see Pippen, 2007.

[31] Greek and Greek, 2000, 70-76.

[32] 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 license, and possibly the whole field of antibiotics might never have been realized" (cited in Greek and Greek, 2000, 73).

[33] Partly because that information remains proprietary and partly because drugs that test highly toxic in animal models rarely make it to human clinical trials.

[34] Greek and Greek, 2004, 17.

[35] Ibid., 18.

[36] Pippen, 2007.

[37] Greek and Greek, 2004, 17-18. Of the 14 drugs that tested safe (i.e., non-hepatotoxic) in rats that were subsequently tested in humans, 6 proved hepatotoxic in humans—a false negative rate of 43%.

[38] Research on this project was supported by a generous grant from the Culture and Animals Foundation, for which I am extremely grateful. I would like to thank Tom Regan, Jeremy Garrett, Nathan Nobis, Ray Greek, Mark Kennedy, John Pippen, Steve Kaufman, Andrew Knight, Erika Engel, Jarrod Bailey, and Alka Chandna for their helpful suggestions. The present paper is much improved as a result.