The Origin of Speciesism

Hugh LaFollette and Niall Shanks

Anti-vivisectionists charge that animal experimenters are speciesists — people who unjustly discriminate against members of other species. Until recently most defenders of experimentation denied the charge. After the publication of `The Case for the Use of Animals in Biomedical Research' in the New England Journal of Medicine, experimenters had a more aggressive reply: `I am a speciesist. Speciesism is not merely plausible, it is essential for right conduct...'[1]. Most researchers now embrace Cohen's response as part of their defense of animal experimentation.

Cohen asserts that both rights and utilitarian arguments against the use of animals in research fail because they `refuse to recognize the moral differences among species'.[2] If we appreciate the profound differences between humans and non-human animals, he says, we would understand why animals do not and could not have rights and why animal pain does not have as much moral weight as human pain. Animal liberationists think speciesism is immoral because they mistakenly equate it with racism and sexism. Cohen claims this equation is `unsound', `atrocious', `utterly specious', and `morally offensive'. Doubtless Cohen is right that the charge of speciesism is founded on an analogy with racism and sexism. He is mistaken, however, in asserting that the comparison is categorically illicit.

Animal liberationists compare speciesism with racism to focus our attention on the human tendency to unreflectively accept contemporary moral standards. We are fallible. Even our deeply held views may be wrong. Our ancestors forgot (or never knew) this important lesson. Thus, although most were not evil people, they indisputably did evil things. We must be leery less we likewise err in our treatment of animals. Of course these historical observations do not entail that our treatment of animals is morally unacceptable. It does, however, suggest we should critically examine our treatment of animals, especially when liberationists have offered arguments which are plausible, even if, in the end, people do not find them conclusive.

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[1] C. Cohen, `The Case for Biomedical Experimentation', New England Journal of Medicine **315**, No. 14 (1986), 867.

[2] Op. cit. note 1, 868.

This is especially sage advice given the close historical connection between speciesism and racism. Historically the two are inextricably intertwined, the former being used to bolster, explain, and justify the latter.[3] Of course, it does not follow that contemporary speciesists are racists — or that all forms of speciesism are indefensible. It does show, however, that speciesism and racism are sufficiently similar so that analogies between them cannot be blithely dismissed as category mistakes.

Of course experimenters could argue that there are differences between speciesism and racism — differences which make speciesism morally justified and racism morally objectionable. But that must be shown. To show that the comparison between racism and speciesism is specious, apologists must argue that although we cannot justify treating blacks and whites differently simply because they are members of different races, we can justify treating humans and non-human animals differently simply because we are members of different species.

How, though, can that be shown? Humans and non-human animals are biologically distinct.[4] But the issue is not whether they are different, but whether they are different in morally relevant respects. Morality requires that we treat like cases alike. A teacher should give equal grades to students who perform equally; she should give unequal grades only if there is some general and relevant reason which justifies the difference in treatment. For instance, it is legitimate to give a better grade to a student who does superior work; it is illegitimate to give her a better grade because she is pretty, wears pink, or is named `Molly'.

Hence, to determine if speciesism is morally defensible, we must first determine if species differences are morally relevant. Speciesism, though, comes in either of two forms. The bare speciesist claims that the bare difference in species is morally relevant. The indirect speciesist claims that although bare species differences are not morally relevant, there are morally relevant differ-

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[3] According to nineteenth century Harvard biologist Louis Agassiz, the biblical Adam was only the Adam of the Caucasians. Different ancestors explained racial differences, differences which Agassiz thought indisputable. And prominent atheist David Hume held similar views, although justified in different ways. See S.J. Gould, The Mismeasure of Man, (New York: W.W. Norton, 1981).

[4] It should be noted, however, that numerous biologists are uneasy about how we draw distinctions between species — and what those distinctions signify. See B. Mishler, and M. Donoghue, `Species Concepts: A Case for Pluralism', Conceptual Issues in Evolutionary Biology, E. Sober (ed.) (Cambridge, MA: MIT Press 1994), 217-30.

ences typically associated with differences in species. We can illuminate that distinction by analogy: a bare sexist might claim that we should give men given certain jobs because they are men, while indirect sexists might contend men should be given certain jobs because they have certain traits which distinguish them from women.

BARE SPECIESISM

Are bare biological differences moral relevant? We don't see how. To say we are humans (rather than dogs or ducks) is just to say that we members of `group or population of animals potentially capable of interbreeding'. But a bare biological divide cannot be morally relevant. That is exactly why racism and sexism are morally indefensible: they assume a mere biological divide marks an important moral divide. Of course, there are differences between the races and the sexes, but so what? The differences are merely biological. Of course, there are differences between the species, but so what?

However, Cohen and other speciesists think species differences are more fundamental than racial and sexual differences. But exactly what this means — and why he thinks species differences are morally relevant — is not obvious. Why should our primary classification (whatever that means) be our species rather than biological class (mammals), biological order (primates), sub-species distinctions (race), or cross-species distinctions (gender)? For some purposes (identifying units of evolutionary selection) species may be considered biologically primary; for other purposes (identifying creatures susceptible to sickle cell anemia), sub-species distinctions may be primary; and for still other purpose (identifying creatures capable of giving birth) cross-species distinctions might be best. Finally, even if we could determine one and only one of these classifications was biologically primary, how would that make this particular biological divide morally relevant?

Stephen Post offers one answer. He claims speciesism is grounded in `species loyalty'.[5] Species loyalty is "the outgrowth of millennia of human evolution shaped by natural selection....[This] `kin selection' or `kin altruism' is deeply ingrained in the human `biogram'".[6] In short, speciesism is morally justified because it is biologically natural to favor one's kin.

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[5] S. Post, `The Emergence of Species Impartiality: A Medical Critique of Biocentrism', Perspectives in Biology and Medicine **36**, No. 2 (1993), 294.

[6] Op. cit. note 5, 295.

To say that such loyalty is natural, however, suggests it is unavoidable — something we do instinctively, something we cannot avoid. But, since some people are non-speciesists, speciesism cannot be natural in this strong nomological sense. Hence, when Post claims favoritism toward kin is `natural', he must mean something weaker — that biological creatures have a tendency to favor their own species. He must also believe this biological tendency should be encouraged by morality and law.

Why should we assume, however, that such a tendency (if it exists) is morally permitted, let alone required? There are other biological tendencies we think morality should constrain. Why is this particular tendency morally sacrosanct? For instance, we probably have a tendency to prefer those who look like us — those who have the same tint of skin and slant of eye. (Perhaps we think of them as kin?) We also have biological tendencies toward aggression. Our hormones sometimes move us to have sex at inappropriate times. But we do not encourage, praise, or morally sanctify these tendencies. Morality should tame them, not lionize them. Therefore, if some `natural' tendencies are morally permitted while others are prohibited, then the bare tendency cannot be what is moral (or immoral). In short, we are not convinced speciesism is natural; but even if it were, we see no reason to believe morality should promote or even permit it.

The deficiencies of speciesism can be vividly demonstrated by a bit of science fiction. Suppose aliens arrive on earth. They are phylogenetically discontinuous with humans — they are not even carbon-based life-forms. We find them aesthetically repulsive. They look like giant slugs — and we call them Slugantots. We have no natural sympathies for them. However, we find their behavior reveals that they are intelligent, purposive, sentient creatures — although the exact contours of their abilities elude us because of their peculiar embodiment.

Post, Cohen, and other speciesists claim that species loyalty gives us the right to favor humans over them — all other things being equal.[7] (It likewise gives them the right to favor themselves over us. Perhaps experiments on us would help them find a cure for their most dread diseases.) We do not see, however, how our aesthetic dislike for them justifies our favoring a human over a similarly situated Slugantot, just as an affinity for people of like tint does not give Caucasians the moral right to mistreat people of color.

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[7] If this is not their view, then they are not bare speciesists but indirect speciesists. That is, they assume that the mere biological difference does not a moral difference make. We assume, however, they are bare speciesists. If not, talk of species loyalty is out of place and unnecessary.

This should not be surprising. After all, moral properties and biological properties are categories from different domains. We cannot merely assume a particular biological property is morally relevant; we must show it is relevant. And even if it were shown, its relevance would be established by referring to moral principles, not brute biological facts. Bare speciesism — like racism and sexism — is simply indefensible. Of course, there are differences between humans and non-human animals, and these differences may be morally relevant. If they are, then treating humans and non-human animals differently would not be morally wrong. But it wouldn't it be speciesism either. The difference in treatment would be justified not by species' differences, but by citing the morally relevant characteristics of species' members.

Since bare speciesism is indefensible, defenders of animal experimentation should cease calling themselves speciesists. But since some defenders of research insist on referring to themselves as speciesists, we must be charitable and interpret them as advocating indirect speciesism — the view that there are morally relevant differences which accompany species differences. Before we can determine if differences between humans and non-human animals are morally relevant, we must first explore the relationship between moral properties and biological properties. That requires a detour into biological theory.

CAUSAL PROPERTIES, FUNCTIONAL PROPERTIES, AND MORAL PROPERTIES

> The biological world is
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> ... constructed not as a smooth and seamless continuum, permitting simple extrapolation from the lowest level to the highest, but as a series of ascending levels, each bound to the one below it in some ways and independent in others. Discontinuities and seams characterize the transitions; `emergent' features not implicit in the operation of the processes at lower levels, may control events at higher levels. The basic processes — mutation, selection, etc. — may enter into explanations at all scales...but they work in different ways on the characteristic material of divers levels.[8]

Put differently, biologically significant properties emerge from evolved hierarchical organization.

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[8] S.J. Gould, `Is a New and General Theory of Evolution Emerging?', Evolution Now, J. Maynard Smith (ed.), (New York: Freeman, 1982), 132.

The organization of biological entities at lower levels produces biological entities at higher levels (macromolecules to cells, cells to tissues, tissues to organs, organs to organisms). The actions at each level cannot be straightforwardly inferred by knowing the biological actions at the other levels. As Ernst Mayr explains it in rejecting the mechanistic atomism of the old physiology texts.

> Systems at each hierarchical level have two properties. They act as wholes (as though they were a homogeneous entity), and their characteristics cannot be deduced, (even in theory) from the most complete knowledge of the components, taken separately or in combinations. In other words, when such a system is assembled from its components, new characteristics of the whole emerge that could not have been predicted from a knowledge of the constituents...Indeed, in hierarchically organized biological systems one may even encounter downward causation.[9]

On the other hand, these levels are not entirely independent either. Emergent properties arise only when the lower levels are of sufficient complexity. Hence, to understand the nature of contemporary biology, we must understand the precise relationships between properties at each level. Specifically, we will need to know about those levels which are described in causal mechanistic terms, as well as those levels described in functional terms. Understanding the differences between them will be crucial for evaluating the practice of biomedicine.

Generally we describe and understand action at the lowest biological levels (the chaining of simple proteins or the shape of antibodies) in causal mechanistic terms — though even here it is possible to talk about evolved functional properties. On the other hand, we typically describe and understand the whole creature in functional terms — in terms of what the creature does and achieves. Intermediate level biological phenomena can be described in either causal or functional terms, depending on what we want to explain or understand. For instance, a physiologist might describe the operations of the liver in causal terms (e.g., the precise mechanism whereby it removes bilirubin from the albumin) or in functional terms (as purifying the blood).

Although these middle level phenomenon may be described in either functional or causal mechanistic descriptions, biomedical scientists will be interested primarily in an organism's causal mechanisms — even when the mechanism could be described, for other purposes, in functional terms. Thus, they are more interest-

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[9] E. Mayr, Toward a New Philosophy of Biology (Cambridge, MA: Harvard University Press 1988), 15.

ed in the liver's mechanisms for purifying blood than in the simple fact that it purifies blood. Or they want to know how an organism oxygenates the blood, how Parkinson's disease causes muscular tremors, how the body defends itself against invading bacteria and viruses. Only by knowing these causal mechanisms can physicians prevent or cure the disease or condition under study. Only then can they know how to intervene medically.

Of course scientists must identify an organism's functional properties before they can explore underlying mechanisms. For instance, the scientist cannot determine how a fish oxygenates the blood unless she first knows that it oxygenates the blood. And she cannot plumb the mechanisms of Parkinson's disease unless she first identifies a network of symptoms. Nor can she understand how the liver purifies the blood until she knows that it purifies the blood. In short, biomedical investigators typically note functional properties and then seek causal mechanisms underlying these biological functions.

Researchers realized that humans and non-human animals often had similar biological functions. Since they assumed similarity of biological function implied similarity of causal mechanism, they understandably thought they could safely generalize experimental results in animals to human beings. This assumption, though, overlooks the inferential gap between an organism's underlying causal mechanisms and its emergent functional properties.

Evolution leads us to expect this gap. Speciation, through genetic mutation, genetic drift, migration, insinuation into different ecological niches, and changing selection pressures will lead phylogenetically related creatures to find different mechanisms for achieving the same biological function. These differences can be biomedically significant. For instance all vertebrates (at least) must find a mechanism to oxygenate the blood. But each species' need to survive in different ecological niches may lead them to evolve different causal mechanism for achieving the same biological function. That is why evolution leads us to expect that similarity of precise causal mechanisms cannot be inferred from similarity of functional properties.

This expectation is borne out in the experimental literature. For instance, although the blood of all vertebrates must be oxygentated, the oxygen carrying capacity of the blood varies considerably, even between closely related species. Consider, for instance, the amount of oxygen carried in the blood. Mammals range from 14 to 32 ml of oxygen per 100 ml of blood.[10] These differences may be crucial in any number of biomedical contexts. It is not so much that we

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[10] P. Withers, Comparative Animal Physiology, (Fort Worth: Harcourt, Brace, Jovanovich, 1992), 734.

know where these ubiquitous differences are going to be significant, it is rather that we do not know where they will not be important. For example, in the course of simian evolution there have been evolved changes in amino acids that permit oxygen to bind more firmly to fetal hemoglobin and, `...the emergence of such a unique fetal hemoglobin may have been a precondition for the developmental changes that lengthened gestation and intrauterine fetal life in simian primates'.[11] There may be any number of toxicological contexts where these differences between rodent fetal hemoglobin and simian fetal hemoglobin are significant.

Or consider the metabolization of phenol. Phenol, is metabolized by a conjugation reaction which joins it with either glucuronic acid or sulfate. The function of this reaction is to enhance water solubility and thereby ease excretion. The relative extent of these two conjugation options varies greatly from species to species. The ratio of sulfation to glucuronidation in humans is 80% : 12%; in rats, 45% : 40%. By contrast, pigs metabolize phenol only via glucuronidation and cats only via sulfation.[12] That should not be surprising since these animals occupy divergent ecological niches. Evolution theory leads us to expect that the precise causal mechanisms of metabolism will differ.

Similarly, evolution leads us to expect that different animals oxygenate the blood differently. These differences are most apparent when we contrast fish with mammals. But even two creatures with lungs (mammals and birds) may have different underlying causal mechanisms, at the level of gross anatomy, for oxygenating the blood.[13] Even where species exhibit close functional similarities with respect to normal energy-yielding metabolism, the effects of stimulation by chemicals foreign to normal metabolism exhibit considerable differences even between closely related species:

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[11] M. Goodman, `An Hypothesis on Molecular Evolution That Combine Neutralist and Selectionist Views', The Unity of Evolutionary Biology, E. Dudley (ed.) (Portland, OR: Dioscorides Press, 1990), 886.

[12] J. Caldwell, `Comparative Aspects of Detoxification in Mammals', Enzymatic Basis of Detoxification, I, W. Jakoby (ed.) (New York: Academic Press, 1980), 94.

[13] `The peribronchial lungs of birds, ventilated in a unidirectional fashion using a series of air sacs, and the alveolar lungs of mammals, ventilated in a tidal fashion using a diaphragm, differ considerably in structure and mechanism. Yet, both ultimately produce the same effect — full oxygen saturation of the arterial blood'. WW. Burggren, and W.E. Bemis, `Studying Physiological Evolution: Paradigms and Pitfalls', *Evolutionary Innovations*, M.H. Nitecki (ed.) (Chicago: University of Chicago Press, 1990), 193.

> Indeed, among rodents and primates, zoologically closely related species exhibit markedly different patterns of metabolism...e.g., the metabolism of amphetamines in the rat is very different from that in the guinea pig, and the marmoset is very different from the rhesus monkey.[14] It is not unreasonable to hypothesize that these marked differences are the indirect consequences of metabolic evolution.

Species in distinct niches will need achieve broadly similar metabolic functions by causal routes that vary according to the precise evolutionary circumstances confronting a species as a consequence of its history. (For example, there are known to be at least 7 metabolic pathways that are unique to human and old world primates.)[15] When stimulated by substances foreign to normal energy-yielding metabolism, these causal differences become significant. To summarize, from similarity of biological function we cannot infer similarity of causal mechanism.

A related point is this: from differences in causal biological mechanisms we cannot infer differences in function. To use one of the previous examples, fish have gills and mammals have lungs. Evolutionary pressures determined that this common biological function (oxygenation of the blood) is achieved by different causal mechanisms. In short, creatures with similar biological functions may not have similar underlying causal mechanisms.

However — and this is crucial — similarity of causal mechanism <u>does</u> imply similarity of biological function. Why should this be so? Think, for a moment, about purely mechanistic systems. A collection of springs and gears, if suitably organized, will produce an analogue watch. An electrically powerfully quartz and suitable organized gears will also yield an analogue watch. Even in purely mechanical systems, similarity of function does not imply similarity of mechanism. However, exactly similar components organized in exactly the same way will produce the same function. This is true not only for mechanical systems, but for biological systems as well. Two creatures with identical properties <u>at all levels of complexity</u>, subject to <u>identical environmental stimulation</u> would exhibit identical functional properties. This is nothing more that the <u>Principle of Uniformity</u>, which is a presupposition of all science. That principle states that for qualitatively identical systems, "same cause will be followed by same effect".

Consequently, although we cannot infer similarity of causal

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[14] Op. cit. note 12, 106.

[15] J. Caldwell, `Species Differences in Metabolism and Their Toxicological Significance', <u>Toxicology Letters</u>, **64/65**, (1992), 653-655.

properties from similarity of functional properties, we can infer differences in causal properties from differences in functional properties.

This causal-functional asymmetry helps give shape to a dilemma for researchers, especially once we understand how moral properties are related to functional and causal properties. As we argued earlier, biological organisms are hierarchically complex. Fundamental structures and causal mechanisms, organized in certain ways, yield higher-order functional properties. Moral properties do not supervene on these lower level causal properties: We do not determine if a creature has moral worth by determining its mechanism for removing bilirubin or by ascertaining the geometric structure of its antibodies. Rather, moral properties supervene on a creature's organism-level functional properties.

For example, we all acknowledge that sentience is a morally relevant property. We think humans should not be made to suffer needless pain because they suffer. The moral properties are "connected" to the relevant functional properties. The exact causal mechanisms of sentience are morally irrelevant. Likewise sapience is a morally relevant property. We think humans deserve moral consideration because of their cognitive abilities; the exact causal mechanisms of sapience are morally irrelevant. In summary, what is morally relevant is what a creature does and experiences — not the stuff of which it is made, nor the organization of its components.

THE DILEMMA

Biomedical researchers offer scientific justifications of animal experimentation: since humans and non-human animals share significant biological mechanisms, experiments on animals can teach us a great deal about human biomedical phenomena. Researchers have also offered moral justifications for experimentation: humans and non-human animals are substantially different in morally relevant respects. At some level these justifications are at odds. Other writers have noted the tension. Rachels, for example, argues that these justifications conflict, at least for psychological research:

> The problem may be expressed in the form of a dilemma that can arise for any psychological research that uses animals for the human case. If the animal subjects are not sufficiently like us to produce a model, then the experiments may be pointless (that is what Harlow and Suomi went to such lengths in stressing the similarities between humans and rhesus monkeys.) But if the

> animals are enough like us to provide a model, it may be impossible to justify treating them in ways we would not treat humans. The researchers are caught in a logical trap: in order to defend the usefulness of research they must emphasize the similarities between the animals and the humans, but in order to defend it ethically, they must emphasize the differences. The problem is that one cannot have it both ways.[16]

As a dilemma for psychological research this has considerable plausibility even in this unqualified form. Moreover, although the dilemma poses some difficulty for all animal research, it is not entirely convincing as it stands. Since moral properties do not supervene directly on lower-level causal biological properties, it is logically possible that humans and non-human animals have (nearly) identical causal properties, yet substantially different moral properties.

Logically possible, but not biologically plausible. The causal-functional asymmetry shows why. Exactly similar causal mechanisms organized in the same hierarchical manner and identically stimulated will yield the same functional properties, but similar functional properties may not be supported by similar causal mechanisms. This asymmetry enables us to spell out the dilemma so that it has moral and scientific bite. To morally justify experimentation, the experimenter must identify substantial and significant functional differences between humans and experimental animals, differences which morally justify a difference in treatment. To scientifically justify experimentation she must identify substantial and pervasive causal similarities between humans and non-human animals, similarities which justify inductions from animals to humans. But how can she identify enough relevant functional differences to morally justify the experiments without finding sufficient causal dissimilarities to undercut their scientific respectability? Conversely, how can she identify enough relevant causal similarities to scientifically justify experimentation without finding sufficient functional similarities to undercut their moral acceptability? Given the causal-functional asymmetry, she cannot.

To see more clearly why this is so, let us ask: what are the respective functional properties which morally justify non-consensual experiments on non-human animals but not on humans. Many laypeople might answer that humans have a soul which non-human animals lack. But no sober-minded researcher would be willing to settle important questions of science and public policy

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[16] J. Rachels, Created From Animals, (Oxford: Oxford University Press, 1990), 220.

by appealing to religious beliefs which many people deny and no one can establish scientifically. Perhaps some scientists do think soul is the relevant difference, but they think that the presence (or absence) of a soul can be explained and identified in scientifically respectable terms. Such a view likely collapses into the view which identifies cognitive and emotional abilities as the functional properties morally distinguishing humans from non-human animals. People who hold this view claim that language, the ability to distinguish truth from falsity, and the ability to act morally or enter into contracts are traits unique to homo sapiens. As Carl Cohen put it:

> Animals... lack this capacity for free moral judgement. They are not beings of a kind capable of exercising or responding to moral claims. Animals therefore have no rights, and they can have none.[17]

How, though, do we scientifically explain the presence of these higher cognitive traits in humans and their absence in animals? The only plausible scientific answer is that humans have an advanced cerebral cortex which non-human animals lack. Fox, for instance, claims human mental superiority is reflected in the `encephilization quotient', `the ratio of the "brain weight of a species with the brain weight of an average animal of the same approximate body weight"... According to this formula', Fox claims, `the actual brain size of humans comes out to six times what should be expected of a typical comparable mammal'.[18]

Let's suppose, for purposes of argument, that humans and non-human animals have dramatically different mental lives.[19] Is it plausible to believe that cognitive differences (brain size, mental complexity, etc.) evolved without any other changes in the animal's biological systems? Are we to believe that whereas humans and only humans have these higher cognitive functions, that all

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[17] Op. cit. note 1, 866.

[18] M. Fox, The Case for Animal Experimentation, (Berkeley: University of California Press, 1986), 38.

[19] Although we recognize there are genuine differences in the cognitive abilities and humans and non-human animals, there is mounting evidence that the mental lives of non-humans animals are far richer than most people suppose. See D. Griffin, Animal Minds, (Chicago: University Press, 1992); and R.J. Hoage, and L. Goldman, (eds.), Animal Intelligence: Insights into the Animal Mind, (Washington, D.C.: Smithsonian Institution Press, 1986). Nonetheless, the current question does not require that we determine whether animals have sophisticated mental lives. The dilemma has bite no matter how we finally settle this question.

other biological systems — all livers, hearts, neurons, and enzymes — are the same in non-human animals as in humans? Of course not. There is no plausible evolutionary story which makes such an outcome plausible, likely, or even possible. These putative higher-order cognitive differences must both reflect and lead to causal differences in other systems and sub-systems of members of the respective species. To deny this evolutionary claim, researchers must embrace bio-cartesianism.

Bio-Cartesianism

Cartesians claim that the mind and the brain are ontologically separate. Having separated the mind and the body, they must now get the mind and body back together again. Animal experimenters have adopted, albeit unconsciously, what we call bio-cartesianism. They assume that the brain, although formed from the same ontological substance as the remainder of the body, developed in ways which neither reflected nor caused fundamental alterations in the body. But, of course, such a view makes no more sense for the biologist than it does for the Cartesian.

To reiterate an earlier point, biological organisms, mammals in particular, are complex hierarchical systems constituted by sub-systems that exhibit strong relations of mutual functional interdependence:

> Subsystems are highly interlocked....[P]roteins are needed to make catalysts, yet catalysts are needed to make proteins. Nucleic acids are needed to make proteins, yet proteins are needed to make nucleic acids. Proteins and lipids are needed to make membranes, yet membranes are needed to provide protection for all the chemical processes going on in a cell... The whole is presupposed by all the parts. The interlocking is tight and critical. At the centre everything depends on everything.[20]

Consequently, biological theory and empirical evidence leads us to expect that differences in higher mental functioning — differences required to morally justify animal experimentation — must, given a proper understanding of evolution, be differences associated with wider biological differences. This, the connection between an animal's cognitive abilities and its other biological functions, has frequently been noted by evolutionists:

> Thinking has conferred on us a priceless adaptive advantage. Evolutionarily speaking, we are successful because our ability

[20] A.G. Cairns-Smith, Seven clues to the Origin of Life, (Cambridge: University Press, 1985), 39.

> to think has enabled us to remain physically unspecialized. We are the supreme generalists. We prove it by our ability to live anywhere and make our living in a hundred different ways. We don't grow thick coats; we get them from other animals. We don't grow long necks; we invent ladders. We don't have teeth as big as apes do, nor are we as strong, pound for pound. We don't see as well as hawks. We don't run as fast as any large quadruped. But by our wits, and more recently by the devices we make, we can outperform all of them in every way.[21]

This point can be specifically illustrated even within closely related primates. Concerning primate EQs, zoologist Richard Dawkins notes:

> Monkeys are well above average, and apes (especially ourselves) even higher. Within monkeys it turns out that some types have higher EQs than others and that, interestingly, there is some connection with how they make their living: insect-eating and fruit-eating monkeys have bigger brains for their size, than leaf-eating monkeys. It makes some sense to argue that an animal needs less computing power to find leaves, which are abundant all around, than to find fruit, which may have to be searched for, or to catch insects, which take active steps to get away.[22]

That differences in lifestyle associated with differences in EQ have significant consequences in non-cognitive biological systems and sub-systems should not be surprising. One animal research handbook cautions:

> When selecting non-human primates because of their close relationship to humans, choice of species of non-human primate is important. For example, a completely vegetarian species may not be as useful because of differences in microflora of the intestine, which may affect drug metabolism.[23]

Biological isolationism

Bio-Cartesianism — the failure to recognize that higher mental functioning reflects and causes other physiological differences in the organisms — is one vivid instance of the researchers' more gen-

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[21] M.A. Edey, and D. Johanson, Blueprints: Solving the Mystery of Evolution, New York: Penguin, 1989), 383-384.

[22] R. Dawkins, The Blind Watchmaker, (New York: Norton, 1987), 189-190.

[23] B.M. Mitruka, H.M. Rawnsley, and D.V. Vadehra, Animals for Medical Research: Models for the Study of Human Disease, (New York: Wiley, 1976), 342.

eral belief in biological isolationism. Biological isolationism denies the strong interrelationship between biological systems and sub-systems. Although biologists sensitive to the insights of evolution are well aware of the interdependence of biological systems, defenders of animal researcher often act (and speak) as if systems were completely isolable. When conducting controlled experiments on non-human animals, researchers (a) isolate one of the animal's sub-systems and mechanisms, (b) apply stimuli, (c) observe results, and then (d) infer that similar stimuli will produce similar results in humans. This inference is plausible, however, only if there are no other sub-systems which are causally relevant to the biomedical phenomenon under investigation.[24]

Put differently, researchers must assume all other causally relevant factors are constant. However, they can reasonably assume all other causally relevant factors are constant only if test subjects and the subjects modeled are of the same species (ignoring intraspecific variation). As we argued earlier in the paper, the theory of evolution leads us to expect that at least some of these other mechanisms will be different in different species. As we there document, comparative physiology verifies this expectation. In summary, biological mechanisms are sufficiently interlocked so that they cannot be straightforwardly isolated in controlled experiments.

Of course none of this suggests that there will not be some causal similarities between the mechanisms whereby a rat achieves some biological function and the mechanism whereby the human achieves the same function. Doubtless there will be. But this fact is of no solace to biomedical researchers. For the discovery that such mechanisms are similar must be an empirical discovery — based on experiments in humans as well as the laboratory animal. But the purpose of many types of biomedical experimentation is to make predictions about how humans behave based on experiments on non-human animals: it is to save humans from being `guinea pigs'.

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[24] For further analyses of the use of animals in biomedical research, consult the following: H. LaFollette, and N. Shanks, `Two Models of Models in Biomedical Research', Philosophical Quarterly **45**, No. 179 (1995), 141-160; `Animal Experimentation: The Legacy of Claude Bernard', International Studies in the Philosophy of Science **8**, No. 3 (1994a), 195-210; `Chaos Theory: Analogical Reasoning in the Biomedical Research', Idealistic Studies, **24**, No. 3, (1994b), 241-254; `Animal Models in Biomedical Research: Some Epistemological Worries', Public Affairs Quarterly **7**, No. 2 (1993a), 113-130; and `The Intact Systems Argument: Problems with the Standard Defense of Animal Experimentation', Southern Journal of Philosophy **31**, No. 3 (1993b), 323-333.

And it is the legitimacy of this extrapolation which the previous analysis calls into question.

Oddly enough, the researchers recognize the strong inter-dependence of biological systems and sub-systems. As the AMA puts it in explaining why cell cultures are no substitute for experiments using whole animals:

> Cells in isolation, however, do not act or react the same as cells in an intact system...isolated systems give isolated results that may bear little relation to results obtained from the integrated systems of whole animals.[25]

And more generally,

> No other method of study can exactly reproduce the characteristics and qualities of a living intact biological system or organism. Therefore, in order to understand how such a system or organism functions in a particular set of circumstances or how it will react to a given stimulus, it becomes necessary at some point to conduct an experiment or test to find out. There simply is no alternative to this approach and therefore no alternative to using animals for most types of health related research.

In short, the researchers argue that there is a class of biomedically significant data that can be recovered only from the study of intact biological systems in non-human species. No other options are acceptable, they claim, because of the highly interlocked nature of an intact biological system. Experimenters cannot have it both ways. If the isolationist thesis were true, the rationale for using intact animal systems disappears. Conversely, if the intact systems argument captures an important element of biomedical phenomena, then because of evolved differences between distinct species of intact-system, we can no longer legitimately generalize from animals to humans.

We are now in a position to precisely state the moral dilemma: if the cognitive abilities of humans and animals are so drastically different as to morally justify experimentation, then those differences will both reflect and promote other biological differences which undercut inductions from animals to humans. On the other hand, if underlying biological mechanisms are sufficiently similar to justify scientific inferences from animals to humans, then the higher-order traits of the test subjects are sufficiently similar to human traits to make research morally problematic.

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[25] American Medical Association (AMA), The Use of Animals in Biomedical Research: The Challenge and Response, (Chicago: American Medical Association, 1988), 27.

We can see the force of this dilemma at work in the current public consciousness. Many people, including many researchers, think chimpanzees are the best test species since they are phylogenetic close to humans. Yet many of those same people are morally uncomfortable with experiments on primates, especially chimpanzees. Why? The same feature which apparently makes them good test subjects also makes them too close to humans for moral comfort. Even Claude Bernard, the father of modern physiology, refused to perform experiments on chimpanzees.[26]

This dilemma is equally potent against one form of argument frequently used by defenders of research: namely, that humans — but not animals — are members of a moral community. Michael Fox claims:

> [The moral community] ... is a group of beings that shares certain characteristics and whose members are or consider themselves to be bound to observe certain rules of conduct in relation to one another because of their mutual likeness. These rules create what we call obligations and derive in some intimate way from the characteristics which the beings comprising the moral community have in common.... [T]he beings in question possess certain salient characteristics, are capable of recognizing these in other, similar beings, and acknowledge possession by other beings of the characteristics in question as grounds for following certain rules of conduct toward them.[27]

An interesting argument. But, in fact, it is beside the point for the purposes of this dilemma. For creatures will be capable of participating in the moral community only if they have the requisite higher order cognitive traits. But, as we have already argued, experiments on animals with such traits would be morally problematic, while experiments on animals without such traits would be scientifically problematic, at least if the purpose of such experiments is, as it usually is, to make inferences about similar biomedical conditions in human beings.

The force of the dilemma can be specifically illustrated by looking at two closely related species: rats and mice. Available evidence suggests rats have somewhat higher cognitive abilities than mice, but the differences are not profound.[28] Consequently, if we deter-

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[26] J. Schiller, `Claude Bernard and Vivisection', Journal of History and Medicine **22**, (1967), 255.

[27] Op. cit. note 18, 49.

[28] R.K. Thomas, `Vertebrate Intelligence: A Review of the Laboratory Research', Animal Intelligence: Insights into the Animal Mind, R.J. Hogge, and L. Goldman, (eds.) (Washington, D.C.: Smithsonian Press, 1986), 50.

mine that rats have sufficiently developed cognitive and emotional abilities to give them some significant moral standing, mice will deserve the same standing. Conversely, if the abilities of mice are sufficiently limited so they do not deserve significant moral standing, then neither do rats.

In short, we have two creatures will similar moral standing — whatever it turns out to be. Yet available evidence suggests the non-cognitive biological systems of rats and mice are sufficiently different that they often react differently to chemical stimuli. Only 70% of the chemicals which induce cancers in rats will also induce cancer in mice.[29] If we test for site specificity, this figure drops markedly:

> Based on this experimental evidence from the CPDB involving prediction from rats to mice, from rats or mice to hamsters, and from humans to rats or mice, we conclude that one cannot assume that if a chemical induces tumors at a given site in one species it will also induce tumors at the same site in a second species; the likelihood is at most 52%.[30]

In short, rats and mice develop tumors at the same site in only 52% of the cases. It would be unrealistic to think humans would react more similarly to either rats or mice, than those species do to each other. Hence, on the most generous assumption, humans will react to carcinogens the same as rats or mice in only half the cases. Indeed, that assumption — though far from sustaining the researcher's case — is unduly generous. For although the site specific concordance rate between rats and mice is only 52%, the concordance rates between humans and rats and between humans and mice are only 44% and 31% respectively. Thus, the power of tests on rats or mice to predict carcinogenicity is humans is weak. Indeed, they are perhaps weaker than even these results suggest. In one test, rats developed cancers when exposed to 19 out of 20 probable human non-carcinogens.[31]

This now gives us a way to state the dilemma concretely: If rats are sufficiently different from humans functionally to justify a difference in moral treatment (non-consensual experiments on rats but not on humans), then we would expect the non-cognitive bio-

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[29] L. Gold, T. Slone, N. Manley, and L. Bernstein, `Target Organs in Chronic Bioassays of 533 Chemical Carcinogens', Environmental Health Perspectives **23** (1991), 233-46. See also L.B. Lave, F.K. Ennever, H.S. Rosencrantz, and G.S. Omenn, `Information Value of the Rodent Bioassay', Nature, **336** (1988) 631-633.

[30] Op. cit. note 29, 245.

[31] Lave *et. al.,* Op. cit note 29, 631.

logical differences between humans and rats to be even greater than the differences between rats and mice. Since these latter differences are significant, they will undermine biological inferences from rats to humans. On the other hand, if the non-cognitive biological differences between humans are rats are sufficiently slight to justify scientific inferences from rats to humans, then it may well be that their cognitive abilities are considerable closer than we standardly think. Thus, the differences between rats and humans — whatever they are — cannot both be scientifically acceptable and yet morally justify experiments on rats.

The reductionistic response

Our dilemma, some might claim, is based on a contentious, anti-reductionistic view of biology. And so it does. However, modern-day biological theory is decidedly anti-reductionist in spirit. Few biologists deny physicalism of some fashion — they are not vitalists. However, most think there are significant biological phenomena which emerge from the complexity of biological organisms, phenomena not straightforwardly reducible to simpler chemical or physical phenomena.

Previous quotes by Mayr, Gould, etc., and discussions in our previous papers (see references), show the pervasiveness of the `New Biology'. Most philosophers of science sympathize with the `New Biology'. After discussing the relatively high level attempt to reduce all of biology to molecular biology, Philip Kitcher notes, `the examples I have given seem to support both anti-reductionistic doctrines.... [D]espite the immense value of the molecular biology that Watson and Crick launched in 1953, molecular studies cannot cannibalize the rest of biology'[32]. And Elliott Sober, who suggests he rejects any strong form of reductivism, notes that, `the thesis of reducibility in principle does not seem to have any direct methodological consequences for current scientific practice'.[33] Put simply, the new biology is both scientifically and philosophically defensible.

However, even if we were mistaken, researchers committed to a reductionist biology do not escape the jaws of our dilemma. If anything, reductive biologists will be even more susceptible to the dilemma's bite. Here's why. They (and the philosophers who cheer them on) claim higher level biological explanation can ultimately be abandoned in favor of lower-level physical and chemical properties. As Hartry Field explains it:

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[32] P. Kitcher, `1953 and All That', Conceptual Issues in Evolutionary Biology, E. Sober (ed.) (Cambridge, MA: MIT Press, 1994), 398.

[33] E. Sober, Philosophy of Biology, (Boulder, Co: Westview Press, 1993), 26.

we should not rest content with a special biological predicate "has a haemophiliac gene" — rather we should look for nonbiological facts (chemical facts; and ultimately physical facts) that underlie the correct application of this predicate.[34] On this view of biology, all functional properties, and thus all moral properties, would be directly reducible to the lowest level causal properties. Consequently similarity of causal properties would straightforwardly imply similarity of moral properties and differences in moral properties would straightforwardly imply differences in casual properties. Thus, if the moral properties of humans and non-human animals were sufficiently different to morally justify animal experimentation, then the scientific worth of these experiments would be called into doubt, and if their causal properties were sufficiently similar to scientifically justify experimentation, then the moral appropriateness of these experiments would be called into doubt.

The only way for reductivists to avoid the force of the dilemma would be to embrace Bio-Cartesianism and biological isolationism. However, the previous adduced arguments against these views will be as telling against reductive biology as against the new biology we originally discussed.

CONCLUSION

Historically there have been two major routes for defending animal experimentation: deontological and utilitarian. Deontological arguments attempt to identify a fundamental difference between humans and non-human animals which morally justifies a difference in treatment. The previous argument does not directly challenge the claim that humans and non-human animals are relevantly different. However, the dilemma does indicate that animal experimentation is unjustifiable even if they are relevantly different. If humans and non-human animals are relevantly different, then animal experiments are scientifically questionable. And if the experiments are scientifically questionable, then experimentation will be morally objectionable since it wastes scarce public resources. On the other hand, if humans and non-human animals are relevantly similar, then experiments will be scientifically respectable, but morally objectionable.

It appears the straightforward deontological defense of exper-

[34] H. Field, `Tarski's Theory of Truth', Reference, Truth, and Reality, M. Platts (ed.), (London: Routledge, 1980), 92.

imentation is implausible. Some deontologists might acknowledge that non-human animals are sufficiently similar to humans to make experimentation both scientifically reputable and morally problematic. However, they might then argue that even as deontologists they recognize the (limited) relevance of consequentialist considerations. On this view, the benefits of experimentation are so overwhelming that they outweigh the cost of experimenting on animals — creatures of some moral worth. For those who wish to take such a tack, their fate will lie with consequentialist defenses of experimentation. But we have argued in a previous paper that a utilitarian defense of research is flawed.[35] It is no longer certain how the researchers can morally justify their practice.

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[35] H. LaFollette, and N. Shanks, `Util-izing Animals', Journal of Applied Philosophy **12**, 1, (1995), 13-24.