Chimpanzees and Medical Research

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Introduction

The foundation of the animal rights movement and the motivation behind the efforts of groups that promote animals’ rights (AR) such as NEAVS is the premise that animals such as chimpanzees are sentient.

Sentient is variously defined as: having sense perception; conscious; experiencing sensation or feeling; responsive to or conscious of sense impressions; aware; finely sensitive in perception or feeling; able to experience physical and possibly emotional feelings; having the capacity to receive sensations; able to perceive.

Among the reasons the AR movement believes chimpanzees and other animals are sentient are the observed similarities in the central nervous systems of humans and other animals. (These similarities are what scientists refer to as gross similarities or similarities on the gross level. Gross meaning: of general aspects or broad distinctions; large; broad; or general.)

Humans have the anatomy (for example the presence of neural tracts in the brain) and physiology (for example the presence of various chemicals) that scientists have discovered are sufficient for the sensation of pain and other sensations. Humans also exhibit behavior consistent with the experience of pain; they try to escape from pain-causing stimuli. These things, taken together, provide evidence that humans can feel pain, hence are sentient. They also provide evidence that animals classified as vertebrates, and perhaps other groups of animals, can feel pain as well. The subjective experiences of members of different species may not be exactly the same but then neither do any two humans respond exactly the same to painful stimuli. For example, some people will have a high pain threshold while others will have a low pain threshold. Men typically describe the pain associated with a heart attack differently than women.
All organisms have similarities. Organisms are classified in various ways depending upon the degree of their genetic similarity. Biology textbooks order forms of life by classifying them into groups with increasingly finer distinctions. One common method classifies organisms into seven increasingly narrow groupings. These are named: kingdom, phyla, class, order, family, genus and species.

Plants are alive but differ from animals in important ways and therefore are classified in a different kingdom, the largest, most general distinction. Dogs and humans have similarities but because of the differences are classified in different orders. Monkeys and humans are in the same order but are placed in different families. Gorillas, orangutans, chimpanzees, and humans are in the same family, but we are all in our own genus. The similarities between organisms place the organisms into the same order, family, and so forth while the differences place them into different subdivisions of that category such as genus and species.

An example of complete classifications for a few species may help make this system more understandable. (Note: by convention, the genus name is always capitalized.)

<table>
<thead>
<tr>
<th>Kingdom</th>
<th>Phyla</th>
<th>Class</th>
<th>Order</th>
<th>Family</th>
<th>Genus</th>
<th>Species</th>
<th>Common name</th>
</tr>
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<tbody>
<tr>
<td>animalia</td>
<td>chordata</td>
<td>mammalia</td>
<td>carnivora</td>
<td>canidae</td>
<td>Canis</td>
<td>familiaris</td>
<td>domestic dog</td>
</tr>
<tr>
<td>animalia</td>
<td>chordata</td>
<td>mammalia</td>
<td>primata</td>
<td>hominidae</td>
<td>Homo</td>
<td>sapien</td>
<td>modern human</td>
</tr>
<tr>
<td>animalia</td>
<td>chordata</td>
<td>mammalia</td>
<td>primata</td>
<td>hominidae</td>
<td>Pan</td>
<td>troglodytes</td>
<td>chimpanzee</td>
</tr>
<tr>
<td>animalia</td>
<td>chordata</td>
<td>aves</td>
<td>ciconiiforme</td>
<td>ardeidae</td>
<td>Ardea</td>
<td>herodias</td>
<td>great blue heron</td>
</tr>
</tbody>
</table>

Life can be classified by other means as well, but for our purposes the above system will be most helpful.

Evolutionary biology explains why all life has characteristics in common and why all life has characteristics that distinguish one form from another. Humans have hearts as do chimpanzees, a similarity. But while humans suffer from AIDS, chimpanzees do not, a difference. The question we will examine is: “How many properties or characteristics must one individual have in common with another in order to use the first as a model for the second?” In other words, since chimpanzees and humans have hearts can we use chimpanzee to study heart disease in humans? Or, as we said above, since chimpanzees and humans share various brain characteristics does it follow that chimpanzees are sentient and should have rights in society? And, if chimpanzees do share the characteristics that result in rights, does it follow that chimpanzees should make good models for the study of human diseases of the brain?

It appears, at first glance that all this results in quite the quagmire but closer examination will reveal that it really doesn’t.

**Similar and Different**

The argument the vested interest groups use to scientifically justify chimpanzee use in biomedical research is based on evolutionary biology. Chimpanzees are humans’ closest
living relatives. Many scientists today think humans and chimpanzees are in fact in the same genus. Regardless of how one classifies chimpanzees and humans, scientists agree that they are our closest relative and as such should respond to drugs and disease more like us than any other animal. If any species can be used to model humans it should be the chimpanzee.

When considering medical research however, many similarities can be outweighed by one difference. This concept manifests in differences between individual humans and differences between species. For example:

**Drugs**

What makes one drug good and another bad? All chemistry, medical, and pharmacology students are taught that the dose determines the poison. In light of current scientific knowledge that should be amended to the genetic and environmental factors influence at what dose a chemical becomes a poison. A drug that kills one may cure another and the above in part explains the reasons for this. When one attempts to determine what a drug’s efficacy or toxicity will be – using other humans as test subjects – the outcome is far from reliable; using a different species is exponentially more problematic. Consider the following:

RotaShield was a vaccine against rotavirus, a virus that causes roughly 500,000 deaths per year. That sounds like a much-needed vaccine, right? But one child out of 2500 in the United States suffered the side effect of intussusception, a potentially lethal bowel condition. Further more, not many children in the U.S. die from rotavirus. However, worldwide, one child out of 200 dies from rotavirus. Outside of the U.S., the risk benefit ratio was very favorable. But since the vaccine could not be sold in the U.S., it was judged too cost ineffective and discontinued. Millions of children worldwide died as a result of not having the vaccine available. Was this a good drug or a bad drug?

Troglitazone, also known as Rezulin was very effective for many patients in controlling diabetes. But in others it caused liver failure. Thalidomide caused birth defects in thousands but today is used as a cancer treatment for some. Aspirin causes life threatening allergic reactions in some patients, but for most is an excellent anti-inflammatory drug. Penicillin, likewise causes life threatening allergic reaction in some but has saved millions of lives. Cisapride, also known as Propulsid, was an effective drug for gastrointestinal disorders, but some patients suffered heart rhythm abnormalities and died.

Are these good drugs or bad?

Allen Roses, worldwide vice-president of genetics at GlaxoSmithKline, a giant pharmaceutical company, said fewer than half of the patients prescribed some of the most expensive drugs derived any benefit from them. Roses said. "The vast majority of drugs— more than 90%—only work in 30 or 50% of the people." Most drugs have an efficacy rate of 50% or lower.
Among ten medications withdrawn from the U.S. market between 1998 and 2001, eight were withdrawn because they demonstrated more severe side effects in women than in men.\(^2\) Similarly, a study in \textit{Science} revealed that one strain of mice (a \textit{strain} of mice is like a breed of dog) could have a gene removed without obvious adverse effects while a similar strain of mice would die without the gene.\(^3\) Considering how closely related strains of mice are that is quite a big difference! If men cannot predict the effects of a drug for women and one mouse strain cannot predict what will happen to another if a gene is removed, it is likely that scientific and medical research has reached the level of study where the tiny genetic differences that distinguish one species from another – and even individuals of the same species from each other – can account for a profound difference in outcome.

Another example is Actinomycin-D, one of the first chemotherapy drugs used in humans. Actinomycin-D kills monkeys.\(^4\) Similarly, drugs known to damage the human fetus are found to be safe in 70% of cases when tried on nonhuman primates.\(^5\) J. Caldwell stated:

\begin{quote}
It has been obvious for some time that there is generally no evolutionary basis behind the particular-metabolizing ability of a particular species. Indeed, among rodents and primates, zoologically closely related species exhibit markedly different patterns of metabolism.\(^6\)
\end{quote}

In light of the knowledge we have obtained about interspecies differences, vis-à-vis the Human Genome Project, evolutionary biology, and studies like the above, it should come as no surprise that trans-species extrapolation is unreliable. Even \textit{intra}-species extrapolation (applying results from one human to another human or one dog breed to another) is troublesome as we saw above. Examples of \textit{intra}-species extrapolation being unreliable abound:

By examining the records of 786 patients and then another 1,093 women and 1,355 men, scientists found that women treated with 5-FU-based chemotherapy for colorectal cancer, had more severe stomatitis and leukopenia compared with men.\(^7\)

Caucasians and African-Americans have a similar prevalence of early age-related macular degeneration. However, the progression to the late form of this disease, which is characterized by proliferation of new vessels in the pigmented layer of the eye (known as the choroid), is very rare for African-Americans.\(^8\)

Similarly, infantile hemangiomas of the skin are commonly seen in Caucasians but are rare in African-Americans.

Currently, an estimated 4,000 to 6,000 Americans die each year while awaiting a bone marrow match. Only about 60 percent of white Americans now find a suitable donor; the rates for minorities range from just 20 percent to 50 percent. Physicians have more difficulty finding a kidney or bone marrow match for Blacks than Whites because Blacks have more antigen combinations on their cell surface and some of the antigens are very rare in non-Black populations. There are other important differences between races, individuals, and sexes. Black women have a 50% higher incidence of breast cancer prior
to age 35 than Whites. They also have a greater probability of developing aggressive
tumors and have the highest incidence of pre-menopausal cancer.

An article published in the *New England Journal of Medicine* in May 2001 revealed that Blacks did not respond as well to the medications known as ACE-inhibitors, medications routinely used to treat heart failure. One theory as to why this is the case is that Blacks have less nitric oxide, a chemical important in how ACE-inhibitors work. This theory led to the development of a medication named BiDil, a heart drug that increases the amount of nitric oxide. It appears to work very well in Blacks, when given to Whites it worked no better than a placebo, as would be expected if Whites already had adequate amounts of nitric oxide.¹

The aforementioned list of drugs and disease and how they effect different people differently gives us a hint as to why many scientists think using animals, even chimpanzees, to model human disease and test drugs is no longer a viable way to do things. If humans cannot predict what a drug will do to other humans how can a different species?

**Infectious Diseases**

The study of infectious diseases is no different.

The vested interest groups frequently say the vaccine against hepatitis B could not have been developed without chimpanzees. Let’s examine that claim.

Scientists could not find the hepatitis A virus except in humans until 1972, when it was discovered in marmosets, one of our more distant primate relatives. Research on marmosets has not yielded results of clinical significance. Hepatitis B was differentiated from hepatitis A based on human clinical studies. The animal model for hepatitis B (HBV) was and is the chimpanzee, which remains essentially asymptomatic when infected. Chimpanzees continue to produce the virus as long as it is in their body; humans don’t. The liver, which is the organ primarily affected, is not affected in chimpanzees in the same way as it is in humans. Liver enzymes, which are measured to assess the progression of the disease, respond differently in humans and chimpanzees. Meaningful comparisons of the human disease with that of the chimpanzee have proven to be impossible. The first hepatitis B vaccine was made from blood of infected humans and is now made from bacterial culture.

It is also suspect to claim that without chimpanzees there would be no HBV vaccine. Just because chimpanzees were used in manufacturing the vaccine does not mean that there would be no vaccine today had they not been used. Clearly, history unfolded as it did and the use of chimpanzees was part of that history. But when we consider where we are today we must consider everything:

1. There is a HBV vaccine that came about in part due to the use of chimpanzees as bioreactors but not because chimpanzees were used to model the course of HBV.
2. There is no cure for HBV. Guha et al: “Despite the existence of a preventative vaccine, HBV represents a substantial threat to public health, suggesting the need for research to develop new treatments to combat the disease.”11 Akbar et al: “Despite the presence of an effective prophylactic vaccine since 1982, more than 350 million people of the world are now chronically infected with hepatitis B virus (HBV). In one scenario, a considerable number of chronic HBV carriers would eventually develop serious complications like liver cirrhosis and hepatocellular carcinoma. In another, chronic HBV carriers would be permanent sources of HBV infection and transmit HBV to uninfected healthy individuals. Taken together, chronic HBV infection represents a major global public health problem, especially in the developing nations of the Asia and Africa, where most of the chronic HBV-carriers reside. Unfortunately, there is no good curative therapy approach for these patients. The prospect of treatment of chronic HBV infection by antiviral agents like type-1 interferons and lamivudine is not satisfactory due to their low efficacy, considerable side effects and high costs.”12

3. There is still no adequate cell culture system for HBV. Guha et al: “A major obstacle to the research on the development of drug and gene-based therapies for HBV infections has been the lack of an efficient cell culture system or a readily available small-animal model, permissive for viral infection and replication. Lack of a robust in vitro cell culture system has seriously hampered the progress of HBV research…For reasons that are not clear, infection of primary hepatocytes and established cell lines with hepatitis viruses has produced poor viral replication and low viral yields and has suffered from poor reproducibility although the addition of polyethylene glycol to primary hepatocyte cultures maintained in the presence of 2% dimethylsulfoxide markedly increases the infection of HBV. In vitro cell culture models can at best demonstrate infectivity by the virus but are not suitable to study viral life cycle because of very low levels of viral replication. They could still prove useful for drug studies.”

If chimpanzees had not been used, the HBV vaccine would not have been developed as it was. It does not follow that it would not have been developed at all. It might have been developed later, or even in the same time frame using tissue cultures, or perhaps even earlier. If money spent on chimpanzee-based research had been allocated to the development of tissue culture, the knowledge gained might have led to a cure. After all, necessity is the mother of invention. When one plays the what if game, as the vested interest groups often do to frighten the public and lawmakers into accepting animal-based research on human disease, one must be careful in drawing conclusions.

Insulin is an example of this concept. After insulin was harvested from pigs and cows it was purified for use in treating diabetes. Researchers essentially thought the problem of diabetes was solved. However, non-human insulin was problematic for diabetics. Many of these problems are no longer an issue now that we have learned how to manufacture human insulin. For nearly fifty years, very little effort had been made to develop human insulin. Why bother, if it could be collected at slaughter? When the inadequacies of cow and pig insulin became apparent, research was directed toward synthesizing human insulin.
HIV/AIDS is another example of the failure of the chimpanzee to model human disease. No animal species has been more studied for the cause and effects of a disease than chimpanzees (and recently monkeys) have been for HIV/AIDS. But what about the results?

Well, one result was the transfusion of HIV-infected blood to French citizens. Claude Reiss, an eminent French biologist with 40 years of research experience, including many years spent at the prestigious French National Centre for Scientific Research (CNRS), stated:

We recall that at the beginning of the 1980s, the observation that HIV was innocuous to great apes convinced experts that the virus was of negligible harm to man. The green light had thus been given in France for the distribution of contaminated blood samples, whose consequences we know. The true cause of the contaminated blood scandal is the animal model. The emergence of other scandals, maybe even more dramatic, is to be feared if the animal model continues to be used as a basis for gauging health risks.13

Reiss’s statement is supported by Pierre Tambourin, then head of the life science department of CNRS* when he testified before the board of Ministers of Parliament on July 9, 1996: "What are the chances of developing a prion disease following ingestion of contaminated meat? Nobody knows, but we must not repeat the error we did in 1983-1985 with AIDS, when we referred to animal models to dramatically underestimate the risk to which humans are exposed." As MPs asked later for more precise wordings, he admitted that he alluded to negative chimpanzee experiments which convinced experts that transfusion of contaminated blood is devoid of risk.14

Once again small differences between, in this case closely related species, have resulted in profound differences in response to disease. Chimpanzees appear to be less susceptible to AIDS, malaria, hepatitis B virus, Alzheimer’s disease, and cancer.15

HIV is a very simple virus; it has only nine genes. Unfortunately for researchers seeking to develop a vaccine, it lacks the usual repair mechanisms and as a result, mutations are common. In ten years HIV can undergo the human equivalent of one million human-years worth of mutations. That is a mutation rate of about 1% per year. (Consider that the 1% difference between humans and chimpanzees took about 5 million years.)

Many breakthroughs seen in nonhuman primates in vaccine development have not transferred to humans.16 The vaccine made by VaxGen, AIDSVAX showed promise when given to chimpanzees17 but failed when tested on 3,330 humans, mostly men. An equal percentage of those receiving the vaccine contracted HIV compared to the controls – those who did not receive the vaccine.18

* National Center for Scientific Research, the largest research organization in Europe. Tambourin indirectly supervised over 2500 researchers and 4000 engineers and technicians, all civil servants.
The virus used to infect the chimpanzee named Jerom (the only chimpanzee to come down with an AIDS-like illness) at the NIH Yerkes Primate Center in Atlanta was different from the type that usually infects humans. After Jerom was infected, his blood was transfused into other chimpanzees that then dropped their CD4 counts, but in contrast to Jerom they did not exhibit signs of illness.

When HIV infects humans for the first time it binds to a part of the cell membrane called the CCR-5 receptor and then it develops a preference for the CXCR-4 receptor. The virus used to infect Jerom relied on the CXCR-4 receptor from the outset. These small differences have proven to be very significant.

The reasons HIV do not infect chimpanzees as it does humans are myriad and again, the result of differences between species.

- In chimpanzees, HIV does not reproduce well.
- Chimpanzees have higher baseline levels of T8 cells, a greater proliferative response, and a lower ratio of T4/T8 cells. Since T4 cells are selectively attacked by HIV, this difference is not insignificant.
- Unlike humans, chimpanzees do not drop their T4 counts to zero with infection. They do go down, but not as dramatically.
- Chimpanzees lack the “killer cells” which humans have.
- B-lymphocytes produce more antibodies in chimpanzees and they produce them earlier, thus stopping disease spread.
- Humans drop their antibody count prior to systemic illness; chimpanzees do not.
- Chimpanzees have HIV only in their blood cells, while humans also have the virus in plasma.
- Chimpanzees exhibit only a flu-like illness in response to being infected with the virus, while humans go on to full-blown AIDS.
- Humans develop opportunistic infections and cancers associated with HIV, which chimpanzees do not.
- Chimpanzees do not reveal classic changes in the central nervous system that humans do.
- Chimpanzees do not have virus particles in saliva or cerebral spinal fluid.

Margaret I. Johnston states about HIV in chimpanzees:
With few exceptions, HIV-1 infection of chimpanzees is universally mild with no notable decline in CD4+ T-cell levels, immunosuppression or other signs of an AIDS-like illness. Rather, HIV-1 infection of chimpanzees results in detectable plasma HIV that decreases within 2–3 months of infection and becomes low to undetectable within a few years. The ability to detect or culture HIV after this initial time period is variable.\textsuperscript{21}

More simply, the same virus has a different effect on different species. This comes as no surprise.

Because of differences such as these, the \textit{Handbook of Laboratory Animal Science}, in 1994, called primate models of AIDS “unsuccessful.”\textsuperscript{22}

(For more on the role of chimpanzees in HIV/AIDS research see \textit{Sacred Cows and Golden Geese: The Human Cost of Experiments on Animals} (Continuum 2000) and http://www.curedisease.com/aidsoverview.html. For more on the role of animal models in infectious disease research, specifically bioterrorism, see http://www.curedisease.com/Pathways/autumn_04.pdf)

\textbf{Times Change}

If animal models – chimpanzees in particular – were at least somewhat useful in the past for studying disease and testing drugs, what has changed in the last few decades that make it obsolete today? Mainly two things:

First: The level of examination has changed since the 18th and 19th centuries. As our examination of living systems has become increasingly fine-grained, we have found that subtle differences between organisms tend to outweigh gross similarities, when it comes to explanations of biologic activity. Today, science is seeking answers to very different questions than when chimpanzees were dissected in the 2\textsuperscript{nd} century by Galen. Science successfully used chimpanzees and other animals to shed light on gross shared functions, however, today we are studying drug response and disease at the level that defines not only a species, but in many cases the individual.

Secondly: our knowledge has increased. Today, science studies human disease and drug response in light of complexity theory, evolutionary biology, gene expression and gene regulation. If we had to single out one thing that has led to the demise of the notion of trans-species extrapolation it would be the concept of gene regulation and expression.

The mouse and human genomes do not appear to be qualitatively very different. They both contain about 30,000 genes, with mice having 300 humans don’t have and vice-versa. Humans and mice both have the genes that in mice result in a tail. In humans, the gene is turned off while in mice it is turned on. Same gene just regulated differently. The differences between species reside in the regulations of the same genes. Gene regulation
also determines drug reaction and disease response. Because of differences in gene regulation, even identical twins may respond differently to disease and medications.

We are living at the beginning of the age of *personalized medicine*. Soon, your genetic profile will be known to you and to your physician. This will allow tailor-made treatments. You will be able to take measures to avoid the diseases for which you are at risk and the most appropriate medications will be selected for you. You will be prescribed drugs that complement your genetic makeup, rather than fight it. If we are to expand and refine our current gene-based treatments, our medical research must be more narrowly focused, not broadly focused for example on entirely different species such as *Pan troglodytes*.

If you are thinking to yourself: “All this sounds very complicated” we suggest you remember what Alfred North Whitehead said: “Seek simplicity but distrust it.” Everything should be made as simple as possible, but no simpler. In simpler times we could and did learn thing about humans by studying animals but today we are studying humans as complex systems and at that level the differences between species outweigh the similarities.

It may also seem to contradict common sense to think that using chimpanzees in research will not benefit humans and that testing drugs on chimpanzees before we give them humans is a good idea. Albert Einstein said: “Common sense is the collection of prejudices acquired by age eighteen.” Einstein also said: “Insanity: doing the same thing over and over again and expecting different results.” Using chimpanzees has resulted in much human suffering and will continue to until we abandon it.

Or you might be thinking that we don’t have an open mind on all this. Arthur Hays Sulzberger once said: “I believe in an open mind, but not so open that your brains fall out.” There is ample evidence that using chimpanzees is unreliable and dangerous. The US Patent Office no longer considers patent applications for perpetual motion machines since it violates the laws of physics. Based on current knowledge of genetics, evolutionary biology, complexity theory, and so forth we should no longer be using chimpanzees to study human disease.

**Conclusion**

Using chimpanzees to model humans is an archaic paradigm that began in the 2nd century A.D. many years before Darwin’s theory of evolution and before the discovery of DNA. When scientists lacked knowledge about these fundamentals, it appeared that humans and chimpanzees had more in common than not. And we did in fact learn things about humans from studying chimpanzees and other animals. Chimpanzees have hearts and other organs, suffer from infectious diseases, and think with their brains, and so forth, as do humans. But modern-day biomedical research is not looking for answers that can be found in chimpanzees. Very small differences between species, on the genetic level, the level we cannot see, lead to lethal errors in the practice of medicine. And not just
differences between species but even between men and women of the same species or between siblings.

One argument that is frequently used to justify the current bias against using chimpanzees is the high cost of maintaining them in labs. We find this without basis. Chimpanzees have been kept in cages hanging off the floor so that their waste will fall out and can be hosed away easily and quickly. The cost of their care and housing in a typical laboratory environment where inexpensive “monkey chow” is their main food, minimal cage requirements are as small as 5’ x 5’ x 7’ and minimum enrichment is offered must be low compared with the cost of say a CT machine, a research assistant's salary, other lab equipment, and so forth.

The cost of maintaining chimpanzees in laboratories is in fact incredibly low compared to making a genetically modified mouse. If chimpanzees fulfilled the requirements for good models we would have no need of genetically modified mice. The fact that we have so many varieties of genetically modified mice speaks volumes about the utility of chimpanzees as a model of human disease.

Further, the reason chimpanzees make such poor models also rules out the use of genetically modified animals generally: complex systems react to change in a nonlinear fashion. Simply changing one or two genes will not result in a complex system that can be used as a good model. If it did, changing a few genes in a mouse would transform it into a human.

If chimpanzees actually were productive models, who can imagine that we wouldn't have chimpanzee labs at every institution? Would we really forego a productive method of investigation?

As we started this essay by discussing sentience we should end it by pointing out that gross similarities, for example sentience, do exist between life forms such as members of the same taxonomic order or genus or species and of course between individuals of the same species such as men and women or Blacks and Whites.

Gross similarities can have ethical implications. For example, just because men and women do not respond the same way to certain drugs implies nothing about the rights of men or women. It has enormous implications for drug administration and other treatments for disease. Similarly, chimpanzees are sentient because of similarities seen on the gross level, but because of subtle differences they are not good biomedical research models for humans. There is no contradiction here as the vested interest groups are wont to claim. Closer examination of most issues is enough to clarify why people who make money from an enterprise want to confuse their audience.

Society funds research on chimpanzees because it believes that doing so will lead to cures for diseases like AIDS, Alzheimer’s, cancer, diabetes, heart disease, multiple sclerosis, Parkinson’s, stroke, and so forth. It is time to put our funds toward the future and leave antiquated chimpanzee-based research behind.
Ethically conducted human-based research such as that occurring today using human tissues, stem cells, autopsies, clinical research and epidemiology, and advances in technology such as that which led to artificial neural networks, balloon angioplasty, mammography, hip and knee replacements, and functional MRI and PET scanners, and advances in the basic sciences such as physics, chemistry, molecular biology and math will lead to the cures and treatments we so desperately need.

(For more on why research using animals persists see Sacred Cows and Golden Geese: The Human Cost of Experiments on Animals (Continuum 2000) and Specious Science (Continuum 2002) and http://www.curedisease.com/introductoryFAQs.pdf and http://www.curedisease.com/why_animal_experimentation_persists.pdf)

(For more on what researchers can do to find cures, instead of using animals in biomedical research, see our book: What Will We Do If We Don’t Experiment On Animals? Trafford 2004. For a more in-depth examination of chimpanzees and other nonhuman primate species used in medical research see “A Scientific Case for the Elimination of Chimpanzees in Research.” Also see http://www.curedisease.com/Pathways/PathwaysFall03.pdf)

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14. Report 3291of the National Assembly: From mad cow to scape cow; vol 2 (1996). Tambourin was since 1985, and is still, president of the Society for Animal Experimentation.


