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Introduction to *Establishing Medical Reality: Essays in the Metaphysics and Epistemology of Biomedical Science*

Harold Kincaid and Jennifer Mckitrick

Medicine has been a very fruitful source of significant issues for philosophy over the last 30 years. The vast majority of the issues discussed have been normative—they have been problems in morality and political philosophy that now make up the field called bioethics. However, biomedical science presents many other philosophical questions that have gotten relatively little attention, particularly topics in metaphysics, epistemology and philosophy of science. This volume focuses on problems in these areas as they surface in biomedical science.

Important changes in philosophy make biomedical science an especially interesting area of inquiry. Contemporary philosophy is largely naturalistic in approach—it takes philosophy to be constrained by the results of the natural sciences and able to contribute to the natural sciences as well. Exactly what those constraints and contributions should be is a matter of controversy. What is not controversial is that important questions in philosophy of science and metaphysics are raised by the practice of science. Physics, biology, and economics have all drawn extensive philosophical analysis, so much so that philosophical study of these areas have become specialized subdisciplines within philosophy of science. Philosophy of medicine approached from the perspective of philosophy of science—with important exceptions (Schaffner, 1993; Thagard, 2000)—has been relatively undeveloped.

Nonetheless, medicine should have a central place in epistemological and metaphysical debates over science. It is unarguably the most practically important of the sciences. It also draws by the far the greatest resources and research efforts of any area in biology. Yet philosophy of biology has focused almost exclusively on evolutionary biology, leaving the vast enterprises of immunology, cancer biology, virology, clinical medicine, and so on unexplored. Naturalized philosophy has emphasized the important interplay of historical and sociological aspects of science with its philosophical interpretation. Biomedical science as a large scale social enterprise is a natural target for such approaches. Relatedly, within philosophy there has been a growing interest and appreciation for the connections between issues of value and issues of fact in science (Kincaid et al., 2007). Biomedical science is a paradigm instance where the two intersect.

The upshot is that biomedical science is a potential rich area for philosophical investigation in areas outside biomedical ethics. This volume seeks to show that promise and to encourage its exploration.

Aside from this general naturalist philosophical perspective which all the papers in this volume share, a number of more specific themes emerge. Not surprisingly, given the central place that the concept of disease plays in medicine, the status of natural kinds is a recurring concern. Yet that concern surfaces in both a practical and skeptical way: practical in that concrete issues such as the nature of genetic disease or the reality of mental illness are at the fore, skeptical in that the notion of a natural kind itself is up for debate. A second recurring theme is the contestability of medical knowledge. None of the contributors argues that medical knowledge is a mere construct, but many are sensitive to the complex relations between data, conclusions, and practical necessities that exist in medicine. A related and third important theme cutting across the essays is that philosophy can learn from what other disciplines have to say about medicine. Medicine has important connections to other biological and social sciences; philosophy can gain much from interacting with other disciplines such as sociology in trying to understand how medicine works. A corollary here of course is that philosophical understanding of general issues in metaphysics and philosophy of science has much to learn from medicine and other disciplines as well. We expand on these themes below, as a preview to their extensive development in the essays to follow.

The interrelated questions “what is health?” and “what is disease?” are central to the philosophy of medicine. The answers to these questions bear on a number of questions related to medicine. People disagree about which conditions count as diseases, and this has implications for what happens in a health care system. The traditional role of medicine is to fight disease and restore health. The purpose of health insurance, both public and private, is to assist those who suffer from disease. So, it seems important to have a clear idea of just what it means for something to be a disease. A key issue in debates over how to define disease has been over the role of values in the analysis. While taking a broadly naturalistic approach, normativists claim that one cannot adequately define disease without employing value-laden concepts, such as harm, suffering, undesirability, and disability. Naturalists, on the other hand, claim to define disease without appeal to values, by employing purportedly descriptive concepts like abnormality and biological dysfunction. Four papers in this collection carve out four different positions in this debate.

In “Normality, Disease, and Enhancement,” Theodore Benditt joins the naturalist tradition in claiming that disease can be defined non-normatively, in terms of abnormality. He considers several challenges to this view, including the idea that someone can be “different” without thereby being in need of medical treatment. His response is two fold. He limits the types of abnormality that count as disease, but admits that his view will count more things as diseases than some would like. However, he distinguishes the question of whether something qualifies as a disease from the question of whether someone should do something about it. Even if something qualifies as a disease by Benditt’s standards, the practical implications for medicine are unclear. In this sense, he is skeptical about the concept of normality determining the physician’s role. Another role that the concept of nor-

mality might play in medicine is rejected by Benditt as well. Whether or not one thinks that a physician should always restore normal functioning, one might think that a physician should do *no more than* restore normal functioning—in other words, that doctors should at most cure diseases, not provide enhancements. Benditt compares this issue to the role of “normality” and “enhancement” in athletics. It’s common to think that athletic competition should be between normal, rather than enhanced, participants. While Benditt finds reasons to limit enhancements in athletic competitions following from the reasons competitions are held in the first place, he finds no comparable reasons to limit medical enhancements generally. Consequently, while the concept of normality plays important roles in some areas of life such as athletics, Benditt claims it is largely misplaced in medicine.

In “Holistic Theories of Health as Applicable to Non-human Living Beings,” Lennart Nordenfelt defends the normative or holistic approach against naturalist, bio-statistical approaches. According to the bio-statistical approach, a disease is an internal state which is an impairment or environmentally caused limitation of normal functional ability below typical efficacy, decreasing potential for survival and reproduction. Nordenfelt, on the other hand, defines disease as a bodily or mental process which tends to cause an illness—a state of suffering or disability experienced by the subject. While health may be thought of as absence of disease, it is better described on this view as a state in which a person can realize his or her vital goals (states necessary for minimal happiness in the long run) given standard circumstances. Of these two contrasting approaches, it seems as though the bio-statistical approach would have wider applicability than a holistic one. While medicine is primarily concerned with the health of people, the concepts of health and disease extend to non-humans—animals and even plants. The bio-statistical approach aptly accommodates the application of the concept of disease to nonhumans. Determining whether a plant or animal is unhealthy would be merely a matter of determining whether some organ or structure was performing its normal function with typical efficacy. It is less clear how to apply the holistic theory of health to non-humans. Nordenfelt’s approach defines health and disease in terms of the experiences of suffering or happiness, and many non-human organisms, most notably plants, are by all accounts, not capable of having these experiences. So, defining health and disease for non-humans presents a major challenge to holistic accounts as against bio-statistical accounts. Nordenfelt tries to meet this challenge by weakening the psychological components of the accounts of illness, health, and disease. However, his main claim against the naturalists seems to be that most organisms can have various interests (things that are in their interest, not necessarily things they’re consciously interested in) beyond survival and reproduction, and to say that only internal states which threaten survival and reproduction count as illnesses for any kind of organism is too narrow a view.

What if the normativist is right, and the concept of disease is essentially normative? What does that entail about the nature of disease? If diseases are normative, are they out in the world for doctors and biologists to discover, or do people invent or create diseases based on their values? If one is a moral realist, one can maintain that certain things are just bad for certain organisms, regardless of anyone’s perception of them as such. This,

in turn, would allow the normativist to be a realist about disease. However, if one thinks that values only arise when someone values something, whether an organism is diseased or healthy would also depend on what people value. If cultures differ with respect to devaluing homosexual desires, deafness, or goiters, for example, whether these conditions are diseases would vary from culture to culture. On this view, diseases are constructed rather than discovered, and do not exist independent of culture.

In "The Spread of Disease: How to Understand and Resolve a Dispute about the Reality of Disease," Robert D'Amico defends realism about disease against this social-constructivist approach. While he concedes to the normativist that medical concepts such as "health," "illness," "pathology," "suffering," and "harm" are essentially normative, he argues that the concept of "disease" is a special exception, and hence not subject to the relativization of other normative concepts. (Hence, D'Amico is a naturalist with respect to diseases, but not with respect to medical concepts generally.) Social constructivists argue that disease states have more in common with aesthetic or moral phenomena than with natural phenomena. Unless one is a thorough-going anti-realist or social constructivist, whether something is water, gold, or an acid is determined by something beyond the mind of any human perceiver. However, according to the social constructivist, whether something is ugly, bad, or sick, depends on the social group which makes this determination. D'Amico draws on concepts from the philosophy of mind and the philosophy of science, namely the distinction between strong and weak supervenience, to redescribe this debate. According to D'Amico, the social constructivist's claim ultimately comes down to the idea that while diseases may weakly supervene on physical states of organisms, they do not strongly supervene on such states. After casting his target in these terms, D'Amico marshals arguments to the contrary. For instance, it is reasonable to think that if a person has kidney disease, then necessarily, any perfect duplicate of that person, no matter the social environment, would have kidney disease. That is to say, there can be no difference in disease-state without some difference in physical state—strong supervenience holds.

While D'Amico and Benditt defend naturalist approaches to analyzing disease and Nordenfelt defends normitivism, Peter Schwartz challenges the distinction itself. In "Decision and Discovery in Defining 'Disease,'" Schwartz claims that a more meaningful classification of views is in terms of value-requiring and non-value-requiring on the one hand and dysfunction-requiring and non-dysfunction-requiring on the other. So, for example, a view that requires a disease to be a dysfunction need not be non-normative. But more importantly, Schwartz questions the utility of any of these distinctions. He charges that philosophers who offer analyses of disease are struck in the outdated analytic project of providing necessary and sufficient conditions for the application of a concept. Once you claim to have given necessary and sufficient conditions for having a disease, you open yourself up to the trench warfare of counterexamples, struggling to deal with variety of bodily states which defy your favored classificatory scheme. However, it has long been acknowledged that ordinary concepts are not adequately captured by necessary and sufficient conditions. Ordinary concepts admit of varying degrees of applicability, with better, worse, and borderline examples. Arguably, when people call a condition a disease,

they are not checking the condition against a list of criteria which it must satisfy. And quite plausibly, there is no one uniform concept of disease that we all share. So, rather than give an analysis of the ordinary concept of disease, perhaps what philosophers of medicine should do is to stipulate a particular concept of disease that will be useful to philosophy of medicine or to medicine itself. Schwartz suggests that the dysfunction-requiring accounts, while not an analysis of all or even most concepts of disease, can provide a useful explication of disease.

Assuming that disease exists, and that we have some idea what it is, we can further explicate the notion by breaking it down into genetic diseases, racially-linked diseases, psychological disorders, and many other sub-categories. However, some question the reality of these categories used to understand disease. Do these categories correspond to real distinctions in nature? Classifications of conditions as mental disorders are particularly susceptible to criticism that they merely reflect societal expectations, as the essays of McKittrick and Horwitz suggest (see below). Other essays in this volume explore related ontological/classificatory questions. Is it possible or meaningful to classify some diseases as genetic and others as nongenetic? Or does doing so reflect a misguided either/or attitude towards genetic determinism? While the category of race has been eliminated from most scientific contexts, can we still establish meaningful correlations between race and disease?

In "Race and Scientific Reduction," Mark Risjord notes that epidemiological research has identified a number of important correlations between race and health. Blacks suffer higher rates of HIV infection, diabetes, hyper-tension, and cardiovascular disease than whites. Since race has been eliminated from biology, this data is philosophically puzzling. If epidemiology and the other health sciences are based on biology, one would expect race to be eliminated from these disciplines too. Risjord sheds light on this puzzle by reflecting on scientific reduction and elimination more generally. Concepts like heat can be reduced to more fundamental scientific concepts, whereas concepts like phlogiston are better eliminated altogether. Should the concept of race be reduced, eliminated, or neither? According to "promiscuous realism," the study of race in medicine and the social sciences is ontologically autonomous—race exists as a social or medical entity, but not as a biological one. Against the promiscuous realists, Risjord argues for a causal-explanatory criterion of ontological commitment. Since, according to Risjord, race does not meet this criterion, race has rightfully undergone an ontological reduction, or elimination, at all levels. Race does not exist. However, questions about race and racial discrimination at the social and medical levels remain important. Risjord retains the role of the concept of race in these realms by distinguishing between epistemic and ontological reductions. While race has been ontologically reduced, it has not been epistemically reduced. This distinction allows Risjord to maintain the seemingly paradoxical conclusion that the study of race in medicine and the social sciences remains autonomous of biology, but neither medicine, sociology, nor biology are committed to the existence of race.

In "Towards an Adequate Account of Genetic Disease," Kelly Smith asks "What exactly is a genetic disease?" For a phrase one hears on a daily basis, there has been surpris-

ingly little analysis of the underlying concept. Medical doctors seem perfectly willing to admit that the causal origins of diseases are typically complex, with a great many factors interacting to bring about a given condition. On such a view, descriptions of diseases like cancer as genetic seem at best highly simplistic, and at worst philosophically indefensible. On the other hand, there is clearly some practical value to be had by classifying diseases according to their predominant cause when this can be accomplished in a theoretically satisfactory manner. The question therefore becomes exactly how one should go about selecting a single causal factor among many to explain the presence of disease. Smith argues that previous attempts to defend such causal selection have been clearly inadequate. In a spirit similar to Schwatz's advocacy of explicative stipulation, Smith proposes an epidemiological account of disease causation which walks the fine line between practical applicability and theoretical considerations of causal complexity and attempts to compromise between patient-centered and population-centered concepts of disease. Smith claims that the epidemiological account is the most basic framework consistent with our strongly held intuitions about the causal classification of disease, yet it avoids the difficulties encountered by its competitors.

A further question about genetic and other diseases is "why do they persist?" If evolution favors those who resist disease, one might expect subsequent populations to be less and less susceptible to disease. However, there is no evidence that this has happened or is happening. In "Why Disease Persists: An Evolutionary Nosology," Robert Perlman explains why. Natural selection is not the only process that changes gene frequencies in populations; mutation and other processes may introduce or increase the frequency of genetic diseases. Interactions between genes complicate the relationship between genotype and phenotype, and may result in the preservation of genetic diseases.

Variations in environmental resources and random developmental events further complicate the genotype-phenotype relationship and may also lead to disease. Natural selection increases fitness, but the declining force of natural selection with age is only one indication that fitness is not equivalent to the absence of disease. Natural selection acts on genes, cells, and groups, as well as on organisms; the outcome of evolution reflects selection at different levels of biological organization. Finally, the human environment is continually changing, largely because of the evolution of our parasites and because of changes in cultural beliefs and practices; genetic evolution is comparatively slow and lags behind environmental change. An evolutionary study of disease complements the traditional medical approach and enhances our understanding of the persistence of disease.

Regardless of which approach to defining diseases one takes, there will still be further tough questions about specific cases – arguments over whether some purported disease really is a disease or whether it is properly understood. The papers by Horowitz and McKittrick raise such issues about mental illness.

Horowitz in previous work had used a hybrid notion of disease as a harmful dysfunction to argue that much of what we call mental illness – e.g. much in DSM IV – are not diseases but problems in living. In "Creating Mental Illness in Nondisordered Communities," Horowitz looks at assessments of the prevalence of mental illness. Studies claim

to show that one in four Americans will have a major depressive episode in their lifetime and one of eight, social anxiety disorder. Yet the latter was described as “rare” in DSM III in 1980. These estimates of prevalence are done by strictly adhered to survey questions analyzed by computer. The surveys are unable to screen out cases where symptoms result not from underlying disease but from transient social stresses—depression due to the loss of a loved one gets categorized with depressive episodes of bipolar disorder; understandable nervousness for someone who rarely speaks in public gets categorized with someone who can never be around more than a couple people. There are vested interests—the NIMH and patient advocacy groups—who benefit if the estimates of mental illness are high and thus the survey process continues. In this sense mental illness is “socially constructed,” for the labeling process is indeed a sociological one. Horwitz holds that this is compatible with taking some mental illness to be fully objective diseases.

In “Gender Identity Disorder,” McKittrick examines a condition labeled a mental illness according to DSM IV. GID is generally thought to occur when a patient has a gender identity that is atypical for his or her biological sex. Gender identity is thus not determined by biological sex. Nor is it, McKittrick argues, determined by psychological properties—being effeminate does not mean a man is woman in a man’s body. Thus GID assumes there is an essence of gender identity that is independent of biological, psychological, and social characteristics. McKittrick takes this presupposition to be quite implausible, for if two individuals are identical biologically, psychologically, and socially, then they ought to have the same gender (gender should supervene on these traits). The solution to the problem is to see that gender identity has no essence. Gender concepts are cluster concepts that cannot be defined in terms of necessary and sufficient conditions. This does not mean that gender identity is a mere fiction. People do identify with different genders. Yet gender identity is a cluster concept that varies according to differing gender norms, not an essence. McKittrick concludes that the DSM IV definition really makes GID a matter of nonconformity to gender roles, something it denies. Recognizing that GID is a social conflict, not a disorder, calls for greater tolerance of social differences, not for treatment of an illness.

The remaining essays are concerned with the nature and role of evidence in biomedical science. There is now an exhaustive body of work in the philosophy of science showing that the relations between evidence, theory, explanation, and the social processes of science are varied and complex. These essays certainly second that opinion for the case of biomedical science.

In “Clinical Trials as Nomological Machines,” Bluhm raises interesting questions about the gold standard for evidence in contemporary medicine, namely, the randomized clinical trial. Every drug approved by the US FDA must show efficacy in two Phase III clinical trials where the treatment is compared to outcomes in a control group that gets either a placebo or sometimes a competing treatment. Bluhm’s main question concerns what success in these trials tells us about what should be done in clinical practice. Bluhm’s conclusion: they tell us much less than is commonly claimed.

Her argument relies on two concepts from the philosophy of science. The first is the idea of nomological machine. Cartwright argues that experiments in the natural sciences are about realizing a set of specific causal processes that are shielded from outside influences. Only in such cases, according to Cartwright, are we to expect science to find laws in the sense of universal regularities. Outside of the experimental setup we only have knowledge about capacities, not regularities. Clinical trials, Bluhm argues, are attempts to establish nomological machines—to provide a situation where interfering factors are sufficiently controlled that we can have reliable knowledge that the treatment caused the observed differences in outcomes.

The problem for the clinician is trying to tell what the experimental outcome tells us about how the treatment will work for the individual patient. There is a widespread tendency to think a successful RCT shows the real effect of the drug. However, it in fact only tells us about the drug's capacity—what it can potentially do—in situations *outside the experimental set up* and it does not give us very much information about that capacity. Clinicians thus must “reverse engineer” the experiment. “Reverse engineering” is Dennett description of how evolutionary biologists must go about trying to identify function in biological organization—by asking how the observed outcome might have been put together. For clinicians to apply RCT results to their own patients, they need to know all the decisions that went into designing the clinical trial in order to make a reasonable judgment about whether the results are relevant to their patient. Published results, however, seldom provide the needed information.

In “The Social Epistemology of NIH Consensus Conferences,” Miriam Solomon also applies ideas from recent philosophy of science to a fundamental epistemic practice in medicine. The practice is the NIH Consensus Conference. These are designed to influence the behavior of physicians by bringing together unbiased experts to reach objective recommendations based on what the evidence shows in a particular area of medicine where there currently is controversy and a gap between theory and practice. The ideas employed from recent philosophy of science concern the many different ways that bias can surface in science as revealed by many historical and case studies. Like Bluhm, Solomon reaches the conclusion that a gold standard in medicine—this time, the Consensus Conference—is not nearly as effective as advertised.

There are two fundamental questions about consensus conferences: do they produce objective decisions on controversial treatment decisions and do they change the medical practice of physicians on the ground? Solomon argues that they fail in the first goal for several reasons. Consensus conferences are preceded by government conducted meta-analysis of relevant studies and these results are announced before hand. Thus in practice consensus is often reached before the conference, not by the conference itself. Moreover, there is good evidence that disciplinary biases—biases that everybody in a given area share—are not weeded out. For example, a recent conference on dental treatments reached entirely different conclusions than the proceeding meta-analysis, but gave no explanation why; the most likely explanation is that the meta-analysis failed to show that standard dental practices are effective, threatening the interests of dentistry. Fi-

nally, obvious checks on the reliability of consensus conferences have not been done—no one has checked to see how frequently their recommendations hold up to further scrutiny.

Solomon concludes that consensus conferences primarily serve a rhetorical function—persuading physicians who are unlikely to be persuaded by meta-analyses. Solomon provides a variety of evidence showing that they are not very effective in achieving this goal as well and that when they do so, it is by changing reimbursement schemes rather than providing a seemingly objective analysis of the evidence.

Moira Howes in “Maternal Agency and the Immunological Paradox of Pregnancy” continues the theme of applying philosophical scrutiny to biomedical evidence. Her target, however, is not a generalized form of evidence as in Solomon and Bluhm. Rather, she focuses on accounts of the immune system in fertility and pregnancy. This has clear practical import, for immune explanations and treatments are gaining in popularity but have undetermined efficacy.

Howes challenges two assumptions in explanations of the immune system’s role in fertility: the ontological assumption that mother and fetus are separate entities and the description of their relation primarily in terms of conflict. Science studies has shown again and again that many assumptions go into the interpretation and explanation of data, assumptions that are sometimes underdetermined by the evidence and that reflect social values of scientists. Howes argues for a similar conclusion regarding the standard picture of maternal–fetal relations. That picture originated in large in Medawar’s “immunological paradox of pregnancy.” The paradox arises because according to Medawar the fetus is the immune equivalent of an organ transplant—it is a foreign body. Thus there must be in pregnancy various mechanisms to ward off an immune attack on the fetus; herein lies the relevance of the immune system to infertility. Other widespread analogies for pregnancy are the invasion of cancer cells or of a parasite.

This picture, Howes argues, is permeated with metaphors that reflect larger social values. There is much biological reason to think that the mother and fetus are not two separate entities in the way that a host and a parasite are. For example, they exchange cells that remain in each other for life. Pregnancy is not a disorder in the way the cancer analogy implies nor is the female body merely passive in the process of pregnancy as the invasion metaphor suggests. The parasite metaphor ignores the genetic ties between mother and fetus and the strong evolutionary reasons to think the mother’s body has a strong interest in protecting the fetus. Dropping the metaphors of foreign body, parasite, and invasive disease might help reproductive immunology to develop explanations that are truer to the real biological complexity of pregnancy and infertility.

The last two papers by Kaplan and Gifford are the only two in the volume directly concerned with policy and ethical questions. Yet in both cases it is the kind of complexities in biomedical evidence discussed by the other authors that drives the normative questions. Kaplan evaluates public health approaches to violence, Gifford, the ethical legitimacy of clinical trials.

Violent crime is a serious social problem internationally. In all cases a large proportion of the crimes are committed by a few young males. This fact has led many to suggest that there is a biological explanation for crime and that identifying that biological basis would provide useful information about how and when to intervene to reduce crime. In "Violence and Public Health" Kaplan rejects this conclusion. While he doubts that we currently have any very well confirmed understanding of the biology of crime, the main thrust of his argument is that even if we did, it would not provide useful information for preventing crime. Kaplan identifies two broad biological approaches to violent crime. One seeks to find biochemical differences between those who do and do not commit crimes or, in a more complex mode, biochemical differences interacting with social differences. Deficiencies in MAOA (monoamine oxidase A) in combination with abusive family environments is one such explanation that has gotten serious attention. The second approach uses evolutionary psychology to treat crime as an adaptive response that is triggered in response to environments with low life spans and low levels of social advancement.

While these explanations are intellectually interesting and might some day be well confirmed, Kaplan argues that they are not relevant for what we should do about crime. At present we have no biochemical correlate that predicts crime and no way to alter MAOA levels even if they were well correlated with criminal behavior. We also have only speculative reasons to think crime results from a genetically based strategy that maximizes reproductive fitness. But we do know that various social interventions can be effective, that the environments they target would be key explanatory variables in any biologically based account, and that the results they produce are generally good things for other reasons as well. Given this, focusing primarily on the biological basis of crime is an irrelevant and potential damaging distraction. Thus Kaplan illustrates well a theme mentioned earlier, namely, the complex interplay between value issues and evidence in biomedical science.

Finally, in "Taking Equipose Seriously" Gifford is concerned about the interplay between different degrees of evidence and the ethics of conducting clinical trials. The basic problem is this: the treating physician has an obligation to provide the patient with the best possible care. However, in clinical trials there may be some evidence that one treatment is better than another long before the trial is finished. So how can a physician ethically enroll a patient to be randomly assigned to either treatment or control if there is evidence that one arm is better? A standard and widely invoked solution Freedman's (1987) notion of "clinical equipoise." So long as the community of practitioners has not reached consensus on which treatment is best, enrolling patients in a trial is justifiable because it is not known which treatment is favored.

Most of Gifford's chapter consists in showing that this widely accepted answer is inherently ambiguous and that as a result the appearance of a solution to the ethical dilemma is misleading. Gifford identifies three different sets of variables that have to be specified to make Friedman's claim unambiguous: (1) community can either be taken in the narrow sense of experts or in the broad sense of physicians at large, (2) consensus can be given either a strict definition as unanimous agreement or a weaker definition as pre-

ponderance, and (3) community opinion might be on the decision whether the treatment should be approved for general use vs. the question of whether I would want to use it given what is known now. The latter distinction is motivated by the thought that we have higher standards of evidence for what will be generally used by the public than we do for decisions only affecting a single individual, echoing the role of consensus conferences discussed by Solomon.

With these distinctions in hand, Gifford argues that Friedman's equipoise solution is not supportable. One rationale given for that proposal is that individual physicians ought to take into consideration the views of the community, for that is evidence in itself. However, that justification supports using the preponderance of opinion reading of consensus. But if equipoise is disturbed as soon as there is a preponderance of community opinion, that can happen early in a clinical trial as the result comes in. So standard practice is still not justified. Moreover, even when there is community consensus in either sense about what to give an individual, there need not be consensus about whether the treatment in question should be publicly available. Yet it is the latter question clinical trials are trying to answer. Gifford concludes that there is no easy solution and that we need an explicit discussion of how to trade off various values in conducting clinical trials.

We end by noting one last common element to most of the essays, one visible in Gifford's conclusion. While various arguments and theses are found implausible by the authors, most do not claim to have the final word on how the various problems discussed should be decided. Simplistic solutions can be rejected, but more careful discussion and investigation is called for to handle the problems raised. That is a strong indication that biomedical science is a fertile ground for ongoing work in applied epistemology and metaphysics.

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References

- Freedman B (1987) Equipoise and the ethics of clinical research. *N Engl J Med* 317 (3):141-145
Kincaid H, Dupre J, Wylie A (2007) *Value free science: Ideal or illusion?* Oxford University Press, Oxford
Schaffner K (1993) *Discovery and explanation in medicine*. University of Chicago Press, Chicago
Thagard P (2000) *How scientists explain disease*. Princeton University Press, Princeton