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Article in *The American Journal of Bioethics* · October 2007

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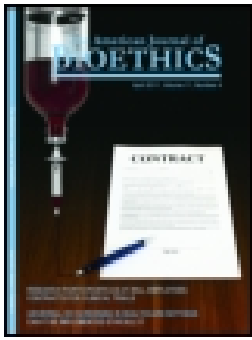


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To cite this article: Michael Henry , Jennifer R. Fishman & Stuart J. Youngner (2007) Propranolol and the Prevention of Post-Traumatic Stress Disorder: Is it Wrong to Erase the “Sting” of Bad Memories?, The American Journal of Bioethics, 7:9, 12-20, DOI: [10.1080/15265160701518474](https://doi.org/10.1080/15265160701518474)

To link to this article: <http://dx.doi.org/10.1080/15265160701518474>

 Published online: 12 Sep 2007.

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Target Article

Propranolol and the Prevention of Post-Traumatic Stress Disorder: Is it Wrong to Erase the “Sting” of Bad Memories?

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The National Institute of Mental Health (Bethesda, MD) reports that approximately 5.2 million Americans experience post-traumatic stress disorder (PTSD) each year. PTSD can be severely debilitating and diminish quality of life for patients and those who care for them. Studies have indicated that propranolol, a beta-blocker, reduces consolidation of emotional memory. When administered immediately after a psychic trauma, it is efficacious as a prophylactic for PTSD. Use of such memory-altering drugs raises important ethical concerns, including some futuristic dystopias put forth by the President’s Council on Bioethics. We think that adequate informed consent should facilitate ethical research using propranolol and, if it proves efficacious, routine treatment. Clinical evidence from studies should certainly continue to evaluate realistic concerns about possible ill effects of diminishing memory. If memory-attenuating drugs prove effective, we believe that the most immediate social concern is the over-medicalization of bad memories, and its subsequent exploitation by the pharmaceutical industry.

Keywords: propranolol, post-traumatic stress disorder, memory, president’s council

INTRODUCTION

Neuroethics has emerged as a new frontier in bioethics. Technological advances in neuroscience, such as imaging, neurological implants, and psychopharmacology, have enabled us not only to better understand the brain, but also to manipulate its functional capabilities. As Farah and Wolpe (2004, 36) have written, because “the brain is the organ of mind and consciousness,” interventions in the brain have “different ethical implications than interventions in other organs.” In addition to the usual issues of safety, efficacy, informed consent, and access, new developments in neuroscience raise issues of privacy, confidentiality, enhancement, assuagement and social control. While advances in the new genetics have raised many of these same issues, as Farah and Wolpe (2004, 35) point out, “ethical questions of neuroscience are more urgent, as neural interventions are currently more easily accomplished than genetic interventions.”

In this essay, we will examine the use of drugs to blunt and even avoid the debilitating effects of post-traumatic stress disorder (PTSD). According to the National Institute of Mental Health (Bethesda, MD), approximately 5.2 mil-

lion adults in the United States suffer from PTSD throughout the course of any given year. According to an unpublished NIMH report by Narrow et al. in 1998, PTSD affects a wide range of individuals, from students and homemakers to soldiers and individuals involved in attacks and accidents (Narrow et al. 1998). One recent study indicates that 17% of soldiers returning from Iraq display symptoms of PTSD or other psychological disorders (Hoge et al. 2006).

To date, most research on the disorder has been concerned with treatment to reduce its symptoms. More recently, researchers have studied ways to prevent PTSD in individuals who have been exposed to traumatic events but have not yet developed symptoms. Experimental prophylactics include drugs such as propranolol (a beta-blocker), which attenuate the memory and emotions associated with a traumatic event.

In this article, we will consider the potential ethical problems of using propranolol to prevent PTSD. First, we will examine issues of safety, efficacy, informed consent, and access. Next, we will examine the potential long-term negative social consequences, concerns that are inevitable with a drug that could alter personality and medicalize what

Received September 26, 2006; accepted April 17, 2007.

Acknowledgement: The authors would like to thank Heath Demaree and Eric Juengst for their helpful comments and suggestions on earlier drafts.

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have previously been considered “normal” human adaptation and coping. To date, there has been little exploration of these potential problems. We will consider two such efforts. The first, by Adam Kolber (2006), gives serious consideration to how the use of drugs to attenuate traumatic memory could potentially interfere with legal proceedings that rely on just such memories. Kolber gives a strong argument for protecting what he calls “freedom of memory”—in this case, the right to choose to maximize mental health by attenuating memory at the expense of being a better witness. The second is the President’s Council on Bioethics 2003 publication, *Beyond Therapy: Biotechnology in the Pursuit of Happiness* (President’s Council on Bioethics 2003). Whereas pharmacologic memory suppression could be problematic in a theoretical future, we argue that the prophylactic use of propranolol for potential PTSD victims appears to have minimal risks and potentially high benefits, and deserves further study through clinical trials. We are critical of the President’s Council’s approach, which has the potential to encourage irrational opposition to such medical innovations. When it comes to weighing the risks and benefits of cutting edge medical research, reasoned debate and careful data collection must shape the discussion. If memory-attenuating drugs prove effective, we argue that the most immediate social concern is the over-medicalization of bad memories and its subsequent exploitation by the pharmaceutical industry. There is evidence to support this concern.

WHAT IS POST-TRAUMATIC STRESS DISORDER?

The *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition Text Revision (DSM-IV-TR)* characterizes PTSD as “the re-experiencing of an extremely traumatic event accompanied by symptoms of increased arousal and by avoidance of stimuli associated with the trauma” (American Psychiatric Association 2000, 424). Events that cause PTSD include witnessing death or injury to another individual, injury to the patient in question, the threat of such death or injury, or even learning of a traumatic event that befell another individual, such as death or injury. Examples include terrorist attacks, rape, robbery, incarceration, abduction or any other type of assault or threat to the person. These events can be experienced or witnessed. Symptoms include, but are not limited to, avoidance, anxiety, nightmares, irritability and detachment. Furthermore, the presence of PTSD in a patient puts him or her at higher risk of developing other related problems (American Psychiatric Association 2000), including suicide and attempted suicide (Ferrada-Noli et al., 1998; Kotler et al. 2001; Tarrier & Gregg 2004). Clearly, PTSD poses a serious threat to quality of life.

Current available treatments include various methods of psychotherapy, such as cognitive behavioral therapy (Bryant et al. 1999; Chemtob et al. 1997), group therapy (Lubin et al. 1998), and psychodynamic psychotherapy (Leichsenring 2005; Leichsenring et al. 2004). In some cases, drugs such as benzodiazepines, anti-adrenergic agents, and selective serotonin reuptake inhibitors are used (Kent et al. 1998; Marks et al. 1998). None of these methods is considered

routinely effective, and all of them may take months or even years to work (Bryant et al. 1999; Kent et al. 1998; Leichsenring et al. 2004). It is claimed that PTSD costs the government approximately \$4 billion annually (Marchionne 2005).

USE FOR PREVENTION OF POST-TRAUMATIC STRESS DISORDER

Studies have shown that shortly after a trauma there is a period in which the memory of the event is encoded and consolidated in the brain. The strength of the memory, as well as its emotional content, is directly correlated to the release of endogenous stress hormones such as adrenaline. When the psychic stimulus is particularly strong, over-release of adrenaline causes elevated levels of noradrenaline (norepinephrine). Increased levels of noradrenaline result in overconsolidation of the memory’s trace. The presence of this overconsolidated memory trace is what specifically generates symptoms of PTSD (Glannon 2006; Pitman & Delahanty 2005; President’s Council on Bioethics 2003; Reist et al. 2001). If beta-adrenergic antagonists (beta-blockers) such as propranolol are administered for a very short period of time either before or after the trauma, the consolidation of the memory and the emotions that cause PTSD can be significantly reduced by blocking the effect of noradrenaline (Cahill et al. 2002; Grillon et al. 2004; Maheu et al. 2005; Pitman & Delahanty 2005; Reist et al. 2001; van Stegeren et al. 2005).

In several studies (Cahill et al. 2002; Maheu et al. 2005; Reist et al. 2001; van Stegeren et al. 2005), subjects were randomly given propranolol or placebo before exposure to a tragic and emotional story and to a mundane and neutral story. When the subjects’ recollection of the stories was tested, the placebo subjects recalled significantly more of the emotional story than the propranolol subjects. Furthermore, there was no difference between the propranolol and placebo groups in recall of the emotionally neutral story. Reist et al. (2001) studied 37 subjects who received oral doses of either 40 mg of propranolol or placebo 60–90 minutes prior to stimulus exposure. The stimulus consisted of 11 slides that told a short story. In the non-arousal version, a young boy witnessed a car accident on his way to the hospital to visit his father. On arrival, the hospital staff was practicing an emergency drill. In the arousal version, the boy himself was injured in the car accident and sent to the hospital, where physicians attempted to reattach his severed legs. Seven days after stimulus exposure, subjects were asked whether they had any recollections of the slides during the seven-day period. They were asked to recall the slides they had seen, as well as the specific details. Lastly, the subjects each took a 76–question, multiple-choice test that examined memory retention.

The Reist et al. (2001) study concluded that propranolol had a significant effect on attenuating memory in subjects who viewed the arousal story. Additionally, the heart rates of subjects who took propranolol were significantly lower than those of subjects who received the placebo. If heart rate is considered a proxy for adrenergic activation, these results

raise the likelihood that this excess activation contributes to PTSD development via augmented memory consolidation. It is important to note that no difference was observed between the PTSD and control subjects in terms of the effect of the drug on the story. If this were accurate, it would indicate that there is no altered relationship between emotional memory and arousal in PTSD patients. Reist et al. (2001) note, however, that 13 of 17 patients were on other psychotropic medications that may have modified the observed response. These researchers believe that further investigation is warranted (Reist 2001).

Studies such as these indicate that individuals in the fire, law enforcement, military and rescue fields might benefit from receiving propranolol prior to a traumatic stimulus (Marmar et al. 1994; Marmar et al. 1996). Such individuals might be treated with short-term memory repressors prior to entering a situation that could put them at risk for later developing PTSD—for example, a plane crash in which rescue workers must enter the site to look for survivors, treat the wounded, or remove the deceased (Fullerton et al. 2004).

It is more likely, however, that propranolol will be used in emergency room settings to treat patients who seek medical attention shortly after having been attacked, abused, raped, molested or involved in any sort of accident that may cause psychological trauma. There have been preliminary empirical studies in actual emergency situations that demonstrate the efficacy of propranolol in reducing PTSD symptoms. Pitman et al. (2001) used 41 emergency department patients who had experienced a trauma likely to precipitate PTSD (based on *DSM-IV-TR* requirements). Within six hours of the occurrence of the traumatic event, subjects were treated orally with 40 mg of propranolol; the dose was repeated four times daily for 10 days, with a nine-day taper period. After one month, symptoms of the disorder were detected in 30% of subjects given the placebo, and 18% of those given propranolol.

Another clinical study of 19 subjects (Vaiva et al. 2003) demonstrated that 37.5% of those who refused propranolol had PTSD symptoms, in contrast to 9% of those who accepted it. Subjects received 40 mg of propranolol orally, three times daily for seven days, with an eight- to 12-day taper period. Although these two studies suggest that post-trauma use of propranolol may be useful in preventing or diminishing PTSD, they also raise important ethical questions about the risks and benefits of such a pharmacologic intervention. Further National Institute of Mental Health (NIMH)-sponsored clinical trials are currently underway (available online at <http://clinicaltrials.gov>, accessed March 11, 2006).

BASIC ETHICAL ISSUES

We will discuss the ethics of using propranolol as both an investigational drug and, if it is found safe and effective, as an accepted therapy. For the most part, these issues are straightforward. During the informed consent process for any research or treatment, the investigator/treating clinician must reveal the risks and benefits of the proposed intervention (Appelbaum et al. 2001). Preliminary studies have demon-

strated enough of a benefit to justify large-scale clinical trials of propranolol's ability to prevent symptoms of PTSD.

The potential side effects of propranolol are well known as tens of millions of people have taken the drug chronically for hypertension, arrhythmia, migraines, and angina pectoris. The usual initial dosage is 40 mg Inderal twice daily, whether used alone or added to a diuretic. Dosage may be increased gradually until adequate blood pressure control is achieved. The usual maintenance dosage is 120 mg to 240 mg per day. So we need to change the text to read "from 120 to 240" instead of 80–240 (Anonymous 2006). Common side effects include fatigue, dizziness, constipation, nausea, and impotence (Walgreens Pharmacy 2004). Short-term memory loss is also noted in the *Physicians' Desk Reference* (2007, 3430). More serious side effects have been reported for patients with serious heart problems. Because research subjects or patients offered propranolol to prevent PTSD would only have to take the drug for a finite period (e.g., six weeks), the side effects would likely be more tolerable. Nonetheless, patients or research subjects could make their own risk/benefit assessments. They could refuse study participation for prophylaxis, or could stop taking the drug if side effects developed. Because the potential benefits far outweigh the risks, we believe that it is ethical to conduct research in which informed consent is obtained.

The President's Council on Bioethics (2003) has argued that one risk of taking propranolol may be the loss of episodic memory—the memory of actual events and potentially the loss of emotionally positive memory. We agree with the Council that these concerns must be taken seriously. Memory and its relationship to emotion clearly are vital to human functioning and flourishing and are very complex (Evers 2007). Our understanding of memory is only in its infancy. Although the ideal goal of propranolol therapy for PTSD would be to attenuate the *emotional* impact of the memory, it is reasonable to be concerned about its (or future memory-altering drugs') potential effect on episodic memory—the memory of actual events. Many of the studies previously cited indicate that there is attenuation of memory beyond emotion; the degree of loss and its implications are unclear. It is worth noting that no severe memory problems have surfaced among the tens of millions of individuals who have taken propranolol for heart conditions and high blood pressure. There are other United States Food and Drug Administration (FDA)-approved drugs that have been shown to interfere with memory; in some cases, this side effect did not emerge until after the drug was approved for marketing. Ambien (zolpidem tartrate) (Sanofi Aventis, Bridgewater, NJ) is the best-selling prescription sleeping pill in the United States. As the number of prescriptions increased after its release on the market, reports began surfacing about its amnesiac effects—for example, people driving their cars without any recollection of having done so (Saul 2006) and binge-eating (Morgenthaler & Silber 2002) while under the influence of the drug. Although this side effect of the drug was considered rare before its approval (referred to as *confusional arousals*), it now seems to be more common than previously thought.

Propranolol has the advantage of having been prescribed for many years for other uses. Any significant problems with attenuated memory would most likely have been reported by now.

Although memory loss is listed in the *Physicians' Desk Reference* (2007) as a potential side effect, its incidence in patients taking propranolol for high blood pressure have not been systematically studied; we rely on anecdotal reports to the cardiologists, internists and family physicians who have prescribed it. A large prospective or retrospective epidemiological study of persons who take or have taken propranolol for high blood pressure would help answer some of the questions about types and degrees of memory loss (e.g., emotional, episodic, positive) that might occur. Such a study might even shed light on the effectiveness of unrecognized and unwitting treatment of PTSD symptoms (e.g., a person taking propranolol who is in a bad car accident and simply continues taking the propranolol for high blood pressure). Unfortunately, such systematic data are not available. Careful attention to memory-related side effects is possible in prospective studies about propranolol's effects on *memory* conducted by psychiatrists and psychologists. Future studies of propranolol and other drugs developed to treat emotional memory could and should provide just this opportunity.

Another potential concern about propranolol research is the competence of research subjects or patients to give informed consent in the immediate aftermath of a severe psychic trauma. However, victims of rape and witnesses to murder, for example, are generally considered competent to accept diagnostic and forensic tests, as well as psychotherapeutic and psychopharmacologic intervention (e.g., tranquilizers). Considering the potential benefits of propranolol far outweigh its risks, it is likely that health professionals will accept a lower threshold for competence (Roth et al. 1982).

Researchers or clinicians using this intervention to prevent PTSD must take decision-making capacity seriously. If a person is judged to be incompetent, we do not believe that he or she should participate in PTSD research, even with surrogate consent. No risk, however small, should be imposed, even by a surrogate until benefits have been demonstrated by careful research. If and when the efficacy and small risk of the intervention are borne out by research trials, we believe that proper surrogates could agree to intervention if patients agree to take the oral medications. We do not believe that prevention of PTSD with propranolol constitutes a medical emergency, defined by Meisel (1979, 436) when "the consequence of withholding treatment is that death will ensue or the patient's health will be substantially compromised." Therefore, patients who competently refuse propranolol, or whose surrogates consent for them when the patients are not competent, should never be physically forced or psychologically coerced into taking the drug. In addition to being an unacceptable form of paternalism, such heavy-handed behavior would likely place an additional psychic burden on an already vulnerable person.

Distributive Justice

The introduction of new therapies sometimes raises questions of social justice. Will they be affordable to everyone? Will they be in such short supply and/or so expensive that only the very wealthy will have access to them? Because propranolol is an extremely cheap and widely available drug, issues of distributive justice are negligible. Walgreens' online pharmacy (Walgreens Pharmacy 2004) sells propranolol in various forms and doses. Based on the reported doses used in the Pitman et al. (2001) and Vaiva et al. (2003) trials, prophylaxis would cost approximately \$13.99 (no author 2005c). When compared with the potential costs of hours of psychotherapy and chronic treatment with pharmacological agents such as antidepressants, the financial benefit of prophylaxis with this drug is clear.

THE LEGAL IMPLICATIONS OF FORGETTING

Adam Kolber (2006) suggests that the use of drugs that affect traumatic memory could pose a thorny ethical conflict between the right of society to protect itself from criminals (by not allowing the altering of valuable evidence) and the rights of individuals to control their own minds (in this case, their memories) (Kolber 2006, 1560). In addition to reducing the emotional impact of memory for the victims of crime, he notes that drugs such as propranolol might "reduce the socially-valuable information that may be vitally important to prosecuting the perpetrator and protecting others from harm" (Kolber 2006, 1579). For example, should a physician who effectively prescribes propranolol to a rape victim be prosecuted for tampering with evidence or obstructing justice? Would victims of "tortiously-caused physical and emotional trauma" hesitate to reduce their own suffering through the use of propranolol in order to create a stronger case in court (Kolber 2006, 1584)? Kolber argues that, although memory dampening might require regulation at some point, any such regulation should be thoughtful and based on research and reasoned public debate about the proper boundary between "an individual's right to modify his memories and society's right to stop him from altering valuable evidence" (Kolber 2006, 1560).

THE PRESIDENT'S COUNCIL ON BIOETHICS

In its monograph, *Beyond Therapy: Biotechnology and the Pursuit of Happiness* (President's Council on Bioethics 2003), the President's Council on Bioethics (the Council) raises questions about how memory loss could affect personal identity and responsibility. After briefly acknowledging that "in certain cases, traumatic memories grossly distort and disfigure the individual's psyche... [and] can cast a shadow over one's whole life, making the pursuit of happiness impossible" (2003, 220), the Council launches into a series of slippery-slope arguments about the dangers of using drugs to blunt the "sting" of bad memories. Although many of these arguments are rhetorically interesting, the Council acknowledges that they are "speculative, at least for now" (2003, 209). However, some of the Council's

dystopic scenarios are a bit exaggerated and distract from more realistic concerns about the use of beta-blockers that could and should be addressed by clinical research and policy analysis. The Council's report too often demonstrates an approach characterized by Macklin (2006, 38) as the use of "metaphors and slogans as substitutes for empirical evidence and reasoned arguments," and an epistemology that presents its intuitions as "immutable truths." While never actually calling for restrictive policy, the document hints at a deeply conservative moral agenda—one that is demonstrated more candidly by Leon Kass, the head of the Council, and other Council members in their own publications (Kass 1991; Kass 1997; Krauthammer 2004; Meilander 2003). Kolber characterizes the tone and content of *Beyond Therapy* as a form of "invasive" and "hard paternalism" that "imposes" values. He calls the Council's concerns "suspect" (Kolber 2006, 1611–1612).

Unsubstantiated Premises

The Council bases most of its arguments on hypothetical premises. It very briefly addresses the issue of "memory-numbing drugs" given preemptively, *before* traumatic events and in non-clinical situations. There are many individuals who are in danger of exposure to a traumatic event—e.g., firefighters, rescue workers, and civilians under bombing attack. The Council discusses the possibility of preparing soldiers for battle "to kill (or kill again); to dull the sting of ones' own shameful acts; to allow a criminal to numb his or her victims" (2003, 224). Although this morally wrought area may be worthy of more careful consideration, the Council raises an alarm but fails to elaborate the moral complexities. Is the Council implying that it is morally wrong to help soldiers to kill? Is this part of a general pacifist stance by the Council? Alternatively, is the Council suggesting that memory-dulling drugs should simply be treated like other chemical weapons and banned by international convention? Or, are there only some situations in which military killing should not be facilitated? If so, which ones? Kolber (2006, 1621–1622) wonders whether the Council has the same concerns about advanced medical technologies for treating soldiers' physical wounds so they may return to battle sooner.

Furthermore, the Council fails to raise more realistic concerns. Would taking propranolol before battle put soldiers in greater danger by lessening or removing their evolutionarily evolved "fight or flight" mechanism? And, might the military be concerned that warning soldiers about PTSD before battle might make them less anxious to enter into it? Could this problem also put soldiers and rescue workers in even greater danger? These are empirical questions that can and should be investigated.

The Council spends most of its effort discussing the use of memory-dulling drugs *after* traumatic events. Here, its first concern is that preventing PTSD would necessarily entail a prospective and quick judgment about "whether a particular event is sufficiently terrible to warrant preemptive memory blunting," and which patients are "destined to have pathological memory effects" (2003, 226). Because

there is no evidence that a six-week course of beta-blockers is or would be harmful, these concerns seem overblown. They certainly do not present a compelling argument for stopping research or denying someone a potentially effective treatment for a potentially crippling illness. Results should be carefully monitored in all study participants to determine who benefits most, who benefits least, and why.

The Council also worries that blunting traumatic memories with drugs could thwart "normal psychic work and adaptive value of emotionally charged memory" (2003, 226). In other words, drugs might interfere with what therapists call "working things through." Normal grief, for example, is a condition that is clearly helped by talking and thinking about painful memories over time, often with social and professional assistance. Pathological grief, however, can resemble major depression and often responds to pharmacotherapy. In PTSD sufferers, the memories and associated emotions are often too powerful to work through. Moreover, there is no existing evidence that memory and emotions will be blunted to such an extent that psychotherapy will not be possible. In the early 1970s, one of the authors (Youngner) remembers that rigid psychoanalysts moralized against the use of antidepressants in severely depressed patients. While acknowledging that the medication might remove the unbearable symptoms, they argued that it would also prevent patients from working through their "underlying" problems. With experience, however, clinicians learned that psychotherapy is almost impossible in a severely depressed patient; when antidepressant therapy lifts the crippling effects of depression, it often frees people up to "work" on their problems. Is it different with PTSD? Studies and experience should answer this question—just as they should with any new therapeutic intervention.

The Council (2003) fears that beta-blockers will be abused by people who do not really need or deserve them—for example, persons who are not suffering from PTSD but who are simply seeking to escape bad feelings attached to bad memories. Yet, there is no evidence that any of the tens of millions of people who have taken beta-blockers for years at a higher dosage for hypertension and cardiovascular disease have discovered or abused such an "off-label" use. Policy and practice should address the possibility of abuse on the basis of careful study.

As a result of the use and abuse of memory suppressors, Council warns that "the notion of moral responsibility would largely unravel" and that "there could be no justice or even the possibility of justice. . . and no forgiveness or the possibility of forgiveness" (2003, 232). It conjectures that memory dulling would allow victims of traumas to forget the horrors they have experienced, creating a scenario in which victims would not demand apologies or retribution. The Council considers a hypothetical intervention in which Holocaust survivors are treated with memory-blunting drugs, and finds the intervention "deeply troubling" because the "human race" would be ill-served by such a "mass numbing of this terrible but indispensable memory" (2003, 231). In our opinion, the idea that such a horrific event could be easily erased by a drug is insulting to those

who experienced it. This is an all too common example of the trivialization of the Holocaust.

The Council (2003) also examines the idea of giving perpetrators of crimes memory-dulling drugs. It says that memory and its associated emotions make us feel the “sting” of conscience, and that “evildoers” can and *should* feel the psychic pain that accompanies their “cruel, brutal, or shameful deeds.” The Council implies that the use of propranolol would eliminate individual conscience of immoral acts, specifically those of “evildoers.” In other words, people would be able to commit heinous crimes without feeling enough guilt afterwards. It is by no means certain that most “evildoers” feel the sting of conscience at all. Did Hitler and Stalin (who did not have access to propranolol) lie in bed awake at night worrying about what they had done? Does the average psychopath who does not have propranolol suffer the pangs of guilty memories? The evidence suggests that the sting of conscience is not likely in these individuals, whom most persons would be willing to label as “evil” (Arendt 1963; Lifton 1986; Stout 2005).

Moralizing and a Hidden Agenda

Presidential bioethics commissions or councils are, inevitably, political; in this respect, the President’s Council on Bioethics is no different (except for its politics) than the National Bioethics Advisory Commission of the Clinton administration. The conservative social and political views of President Bush, Leon Kass, and a majority of the Council are not a secret. These views are not specified in *Beyond Therapy* (President’s Council on Bioethics 2003). While one could argue, as the Council does, that the report simply raises interesting questions, the premises about evil and evildoers (without a definition of *evil*) are hardly examples of the Socratic method. The Council never disavows its conservative agenda, but also never delivers the punch line—i.e., identifying what is evil and shameful, how that is decided, and by whom. For example, the Council warns about drugs such as propranolol making it easier for people who perform shameful deeds. But who decides which deeds are shameful or cruel and which are noble and heroic? Politicians? Judges? Priests? Bioethicists? President’s Councils? Would it be wrong for a fundamentalist Christian psychiatrist to give propranolol to a teenager who is ashamed about masturbation or homosexual fantasies, but permissible for a liberal secular psychiatrist to do so? Is one of those two moral viewpoints the right one? The Council completely fails to address or even acknowledge the profoundly important questions of who should make such decisions. Such an approach is troubling in a liberal democracy—particularly at a time when reason, science and open public debate are threatened by a religious fundamentalism that claims divine knowledge of right and wrong, and would like to use the state as its enforcer. If this were not the Council’s intent, it surely would have reassured the reader to the contrary.

Biomedicalization and the Codification of New Diseases

In the final chapter of *Beyond Therapy*, the Council briefly indicates that one of the lingering effects of new biomedical technologies is that they medicalize what were heretofore considered “normal” states of being (2003, 305). We think this phenomenon is worth exploring in greater depth. We also wish to take the Council’s position one step further by linking medicalization to the politico-economic landscape of contemporary American biomedicine and the role of the pharmaceutical industry.

Medicalization, as defined by sociologists in the 1970s and 1980s, describes at least two processes: 1) placing what had previously been conceived of as a “normal” aspect of the human condition under the medical gaze; and 2) taking something that was deemed by society to be deviant and placing it under the jurisdiction of medicine (Conrad and Schneider 1980; Parsons 1979). Examples of the first process include phenomena such as childbirth, menopause and death (Conrad 1982; Zola 1972). The second process is exemplified by alcoholism, gambling, hyperactivity in children, and even political dissent. In each of these latter cases, the condition was originally under the jurisdiction of another social institution (e.g., religion, law, education), and was then placed within the realm of biomedicine.

In recent years, new processes of biomedicalization have appeared that are particularly germane to this argument. We have witnessed the expansion of the diagnostic conditions of an illness to include more symptoms and include greater numbers of people (Clarke et al. 2003; Moynihan and Henry 2006; Zita 1998). This expansion has been documented in cases of clinical depression (Healy 1997) and bipolar disorder (Healy 2006); it is particularly evident in the expansion of attention deficit hyperactivity disorder to include far greater numbers of children (Lakoff 2000) and a burgeoning adult population (Conrad & Potter 2000). Biomedicalization occurs as the result of a conglomeration of societal forces, including: medical professionals who diagnose the diseases; advocacy groups who fight to have their suffering recognized by biomedicine in the form of a diagnosis; and changing societal norms that expand or contract with changing mores. The pharmaceutical industry is a primary actor in this process.

The expansion of the diagnosis, and sometimes the codification of the disease category itself, is encouraged and promoted by pharmaceutical companies that manufacture drugs prescribed to treat the disorders—e.g., depression (Healy 1997); bipolar disorder (Healy 2006); attention deficit hyperactivity disorder (Phillips 2006); erectile dysfunction (Lexchin 2006); female sexual dysfunction (Fishman 2004); and premenstrual dysphoric disorder (Greenslit 2002). Pharmaceutical companies sponsor disease awareness campaigns, advertise prescription drugs directly to consumers, and target physicians at continuing medical education conferences and in their offices to encourage them to prescribe their drugs. Sometimes referred to as “disease mongering” (Moynihan & Henry 2006), this newer process of medicalization gives pharmaceutical companies the ability to capitalize

on human suffering and exploit insecurities and unhappiness in order to increase drug sales. Indeed, pharmaceutical companies seem poised to reconfigure the landscape of disease and illness categories within biomedicine.

Propranolol may be ripe for pharmaceutical “re-branding.” An enterprising pharmaceutical company that wishes to manufacture and market a newer version of propranolol for the treatment of PTSD need only slightly alter its chemical composition to obtain a new patent and market the drug under a new prescription name. It might, for example, promise fewer side effects or longer-lasting effects than the generic propranolol. Or, like NitroMed’s (Lexington, MA) patent on BiDil (isosorbide dinitrate/hydralazine) (BiDil-Nitromed, Lexington, MD), the “new” drug to treat heart failure in African-Americans, which combined two already available generic prescription drugs into a single drug, we can imagine a new combination pill of propranolol and, perhaps, benzodiazepine marketed and repackaged to prevent PTSD. The pharmaceutical company responsible would then be able to brand the “new” and now more expensive drug and market it under a new name, let us call it “ProBenz” with a new patent for the “new” ability to prevent PTSD. A marketing campaign to consumers and clinicians alike is sure to follow.

Various scenarios become possible. Patients would be made aware of and offered of ProBenz in the aftermath of a traumatic event. To sell more drugs, the pharmaceutical company would want to delineate the range of traumatic events for which its drug should be prescribed—e.g., rape, violent crimes, death of a loved one. This is where medicalization processes come into play. Trauma—our conception of it, its parameters, what “counts” as trauma—is necessarily culturally and socially defined, *not* medically. Yet, the definitions of trauma would become codified by the FDA through its indications for use of ProBenz and the pharmaceutical company that makes it would continually attempt to push the boundaries of trauma outward in order to sell more drugs. For example, a drug advertisement in which someone is encouraged to take propranolol after an embarrassing or humiliating experience at the office. Here we have reason to be concerned that a private company seeking to sell more pills will promote an expansive set of PTSD causes and symptoms (to physicians and patients alike), altering both our sense of the illness and our interpretations of the experiences that might cause it.

This seems particularly acute in terms of the use of ProBenz given as a prophylactic to trauma. Although the President’s Council on Bioethics and others have primarily focused the preventive uses of propranolol for groups such as the military or emergency rescue teams, with ProBenz now on the market, the company that makes it would attempt to market its prophylactic use directly to consumers. Assuming the FDA approves it for this use, it once again becomes a question of the breadth of traumas for which we think ProBenz is appropriate. What is seemingly a social question becomes defined in large part by the pharmaceutical company looking to sell more drugs. If ProBenz becomes a prophylactic drug, then it also could be marketed

to consumers who *may* be exposed to a trauma in the near future. Perhaps everyone should have ProBenz on hand to take either before or after exposure to a trauma. Falling in line with the previous cases of methylphenidate for attention deficit hyperactivity disorder and selective serotonin reuptake inhibitors for depression diagnoses, propranolol may be positioned as another catalyst of “diagnostic bracket creep” (Kramer 1993, 15), in which the availability of a new drug encourages the expansion of a diagnostic category. This is made all the more complicated in this case with the added nebulous category of “prevention” rather than treatment where the potential for expansion is even greater.

We believe the President’s Council on Bioethics (2003) is right to raise forward-looking concerns about the introduction of a new medical technology. If anything, modern history has taught us that scientific breakthroughs are almost always double-edged swords (Evers 2007). However, while the language of “evildoers” and “pain” that is “deserved” has resonance these days in high political circles, it has little utility in the scientific and sober evaluation of a new medical technology and its potential dangers.

Research about the use of beta-blockers to dull memory is in its infancy. Propranolol might well turn out to be ineffective for the treatment of PTSD. Some people may even be harmed by it. Good clinical data and reasoned public debate should determine the balance between harms and goods. We do not believe the President’s Council on Bioethics (2003) raised concerns that would justify ending research into the use of beta-blockers for prevention of PTSD, nor discouraging the clinical use of these drugs if research proves them effective. One of the benefits of propranolol research may indeed be the rehabilitation of an older, widely available, and affordable drug for a new and important use. We do, however, see the need to keep a careful eye on the potential exploitation of the research on propranolol and PTSD by pharmaceutical companies. ■

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