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Distorted Packaging: Marketing Depression as Illness, Drugs as Cure

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Prominent consumer depression manuals issued in recent years circulate a standard depression script as scientific knowledge. The script, asserting that a broad spectrum of depressions are brain illnesses that require antidepressant treatment, is in fact highly contested among researchers. This paper reviews the logical problematics of these manuals, and how such discourse promotes the diagnosis and pharmaceutical treatment of behaviors ranging from mild symptoms to severe depression. In keeping with the trends of pharmaceutical advertising and State health policy, these manuals encourage consumers to self-scrutinize risky behavior, and to treat common behavioral and mood distresses with antidepressants. Ultimately, these activities of self-management function to produce a more productive citizen population.

KEY WORDS: depression; antidepressants; pharmaceuticals; neuroscience; consumer culture; marketing; productivity.

> *[Scientific discourse] is a form of shorthand in which facts, once admitted, need no longer retain the history of their fabrication.*
>
> Paula Treichler, 1992a, p.86

Ubiquitously, State health policy and researchers tell us: "Mental illness is an illness, and Depression is an illness." Prozac maker Eli Lilly greets depressed consumers with "Welcome Back," while Wellbutrin makers declare "There's Hope for Depression." Depression researchers, State policymakers and mental health advocacy groups would have us believe that scientists *know* what depression is—a disease of the body—and therefore know how to fix it. An array of consumer

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literature also proclaims depression to be an illness and promotes psychopharmaceutical use, but simultaneously admits that research has not found *conclusive* evidence that depression is *caused* by the ill body. The general expert claim from within neuropsychiatry, then, is that depression *is* the ill brain, while consumer literature suggests that the ill brain *causes* depression. Is the difference important to consumers? Vociferous protestations of neuropsychiatric framing of depression are voiced by psychosocial researchers, "Prozac Survivors,"[3] social workers and sexual abuse victims, among others, who argue that environmental and situational factors (i.e., abuse, neglect, overwork, and gender role imperatives) should be considered "causes" of depression. Such critics are concerned that social and psychological measures be taken to prevent depression and to heal it. Still, the neuropsychiatric assumption that depression is always an ill body, largely belies and frames contemporary depression literature, playing a pivotal role in constructing popular cultural knowledges of depression and making depression an American epidemic.

This paper addresses the discourses of scientific and consumer literature on depression to unfold a story of how depression became marketed as a widespread biological malady, and how biotechnical treatments were successfully sold to consumers as an appropriate response. This study reveals instances where *contradictory* scientific discourses and findings, simultaneously circulated in both scientific and consumer materials, are presented as tenable depression knowledges, and where consumer literature fails to reveal theoretical controversies regarding widely used antidepressants. Psychiatric science, in other words, is addressed as a product of State, cultural and industrial practices, not an objective and isolated entity, and one largely situated in neuroscience. This study does not seek to *invalidate* neuropsychiatric science, but to trace how it epistemologically constructs the idea that broad populations of the American public are biologically ill with depression. The term biopsychiatry in this paper is employed to refer to this phenomenon.[4] The paper seeks to reveal how neuropsychiatric reasoning and discourses on depression edges its way into the national psyche. That is, how do dominant depression discourses of scientific and consumer literatures circulate through culture in tandem, constructing a popularized "common sense" script of depression that is difficult for consumers to think outside of. Finally, the paper inquires into the relationship between the widely-circulated dominant depression script and the consumer market,

[3]This self-named group contends that Prozac is a drug that causes some users to become violent or suicidal. The group's Website (http://www.pssg.org/; accessed 9/23/01) offers legal packages and attorney referrals for consumers who wish to pursue a case against Prozac manufacturer Eli Lilly. As of late, the group has been validated by Eli Lilly's production of a new, improved Prozac that eliminates the violent "side effects" that the company previously ruled out as inconclusive.

[4]The term biopsychiatry in this paper refers to the post-modern era of biopsychiatry, where depression research predominantly focuses on neuroscience, addressing brain chemistry and genes, with less research on cognitive and behavioral psychotherapy and little psychoanalytic research. As such, problems are often framed as neuroscientific dilemmas, assuming that mental disorders are due to sick brain. The terms are juxtaposed in this paper to reveal this tendency.

asking whether consumer media on depression adequately informs consumers of drug action and researcher's debates regarding treatment options, thereby allowing consumers to make informed decisions. I suggest that constrained media discourses feed a psychopharmaceutical market and tend to reify dominant research paradigms.

STATISTICS AND EPIDEMIOLOGY

The National Institute of Mental Health (NIMH) and the National Alliance for the Mentally Ill (NAMI) claim that major depression allegedly afflicts 18.6 million people annually in the United States, (NAMI, 1999a) or nearly 10 percent of adult Americans ages 18 and older each year (NIMH, 2000c). These organizations suggest that 50% of the diagnosed experience a recurrence in 1–2 years, and that only half of the afflicted seek treatment (NAMI, 1999a). Since the early 90's depression has grown from a marginal affective or mood "disorder" to a broadly diagnosed one, a label assigned to symptoms ranging from suicidal plans to malaise and situational grief. The population of those considered at risk is believed to be broadening: new studies allege that 2.5 percent of children and up to 8.3 percent of adolescents suffer depression annually, while American children as young as four years old have been treated with antidepressants untested on children (NIMH, 2000a).[5] Though the NIMH admits that only 1–2% of seniors (over 65 years of age) suffer depression, the organization claims that up to 13–27% suffer "subclinical" depressions that put seniors, too, at risk of depression (NIMH, 2000a). Even while the spectrum of depression and the population of those "at-risk" broadens, science still largely considers the "disorder" to be a women's disease. Nearly twice as many women (12 percent) as men (7 percent) are afflicted with depressive illness annually according to NIMH data, and recent federal health policy efforts such as "Healthy People 2000 and 2010" rigorously survey adolescent girls and senior women as populations most at risk.[6]

Psychiatric literature produced by the State and by neuroscientists also contends that depression is strongly linked to environmental problems. The primary western diagnostic taxonomy, *The Diagnostic and Statistical Manual of Mental Disorders* (the *DSM*), for example, claims that depression is an illness, and that depression is indeed often precipitated by social stressors. While most neuroscientific researchers admit that both environment and the body can be causes of depression,

[5]These numbers are quoted so often in newspaper articles that they are popular knowledge among experts and researchers.

[6]While it is estimated that one in fifteen men will suffer depression in his lifetime, the rate jumps to a one in five chance for women (NAMI, 1999a). These are conservative estimates and roughly the same for seniors, though it is notable that being unmarried or widowed increases one's chance of acquiring depression (NIMH, 2000b). Women are also considered hormonally at risk of depression—the NIMH suggests that an astounding 20–40% of women suffer Premenstrual Syndrome, which can cause "mood swings and physical symptoms that can interfere with work and social life" (NIMH, 2000b).

they contend that the body ultimately becomes ill, thus prioritizing research on the ill, depressed brain.[7] Single cause research models are also predominant due to the practical difficulty in constructing studies that look at environment and genes or biochemistry as *dual* causes of depression. Consumers are offered a collapsed version of this psychiatric narrative, one that polarizes environment and body as depression causes and leaves the consumer believing that an ill body is best treated by psychopharmaceuticals. Why are consumers asked to choose between dualized epistemologies of cause that even scientists don't embrace? Why are consumers not allowed entrance to the debate regarding whether cause is irrelevant to treatment or the appropriateness of severely limiting research on environmental or dual causes? Answers to these questions lay not only in the politics of conflicting research programs and paradigms, but in the social history of psychiatry, its relationship to the State, and the State's stake in neuroscientific research.

Biopsychiatry, which focuses on the sick brain, rose to respectability throughout the 20th century with support from philanthropic organizations and State research monies, and with the 1954 publication of the *DSM*.[8] Biopsychiatry's status blossomed in the late 20th century following successes in sheep cloning and other genetic research, accompanied by mass media accolades for new biotechnologies. Two effectively publicized events—cited successes of new psychopharmaceutical medicines to treat schizophrenia and manic depression and new SSRI antidepressants (Selective Serotonin Reuptake Inhibitors),[9] particularly Prozac—presented biopsychiatry as *the* successful psychiatry. Additionally, widespread data collection undertaken in the 1984 Epidemiological Catchment Area (ECA) study produced startling, controversial statistics claiming that mental illness rates were 50 times higher than earlier believed.[10] Importantly, ECA research and NIMH reports on depression consistently link mental illness with personal and State economic productivity, suggesting that the State embraces biopsychiatry to ensure a

[7]These insights come from reviews of the field, as well as conversations with neuroscientist Dr. Ginger Hoffman, who performed research on brain proteins believed by some to play a role in depression, and psychiatrist Jonathan Metzl.

[8]Specifically the Rockefeller and Carnegie corporations supported biopsychiatric research in the early mental hygiene years prior to the 1950's and into the 1960's. The *DSM* manual initially provided a taxonomy of mental disorders for use in record keeping and communication and has grown today to include symptom clusters and recommendations for diagnostic practice. With expansive influence in the field, these two phenomena helped to legitimate biopsychiatry in psychiatric science.

[9]These drugs are no more effective than previous antidepressants, according to numerous federally funded studies. The most comprehensive to date study conducted for the NIMH by Elkin, et al., 1989, for example, found that nonmajor depression is best treated by psychotherapy while major depression is best treated by a combination of psychotherapy and antidepressants.

[10]Not since the Carter administration, which quantified numbers of mentally ill persons to plan for future service needs, has US health policy put such effort into collecting data on mental illness. The 1984 studies were intended to update data and meet conservatives' insistence on cost-control and accountability of service provision efficiency. The study, for example, declared that each year 24.1% of Americans suffered a mental disorder (Kutchins & Kirk, 1997). These highly exaggerated rates have worked in service to the State agenda to increase depression diagnoses and treatment rates.

productive economy. For example, the 1999 Surgeon General's Report on Mental Illness contends that mood disorders have profoundly deleterious consequences not only on quality of life, but also on *economic productivity*, matching the impact of heart disease and greater than the effect of peptic ulcers, arthritis, hypertension, or diabetes. The equation of mental and economic health is summed up by Pablo Pasquino: "Population is wealth, health is value" (1991, p.115). In epidemiology, population "problems" of age, sex, occupation, birth and death are often blamed on poor health, making the public pursuit of health a reasonable, even necessary "solution."[11]

Mental health statistics succeed in making depression epidemiology seem legitimate, sick "populations" factual, and pharmaceutical solutions appear the appropriate remedy. Paula Treichler (1992a) terms statistics "bad science infected by rumor and fantasy" that present the illusion of control over the disease or disease as *unmediated* epidemiological phenomenon (p. 391).[12] Statistics control public *reception* so that we *desire* science to reveal a simple disease truth we can accept and employ to seek a solution (Treichler, 1992). Statistical studies constrain the terms and breadth of the so-called problem: by terming the problem of depression as biological, blame is averted to the body, making State worries about the impact upon national productivity appear reasonable rather than callous, and alleviating consumer guilt. The process of discovering cause is streamlined by a broadly entrenched public trust in epidemiology; this affords individuals the luxury of accepting diagnosis and quick fix drugs without desiring to research, critique, or worry. In other words, the action plans of biopsychiatric research and epidemiology converge, each "discovering" alleged criteria for depression, large "populations" at risk, suggesting biochemical or genetic cause, and highlighting the accompanying deleterious effects on the national economy. Biopsychiatric research and epidemiological discourse thus function in tandem, creating a public that is broadly-diagnosed with a variety of depressions, who have a primary concern in reinstating or improving their productivity levels.

Depression, which is considered a menace to mood and national productivity, is overdetermined by scientists as a primarily biological problem that requires a biotechnical solution. In turn, an overly broad population agrees to diagnosis individuals and thus pharmaceutical treatments are over-prescribed and over-consumed. In this sense, today's depression is similar to 19th century hysteria; each was assumed to be caused by biological dysfunction and termed a social menace requiring rigorous medical confrontation (Oppenheim 1991; Chesler, 1972;

[11] Where the Carter administration sought to reverse the under-servicing of people in need, the foremost concern of today's policy planners is to reduce the *cost* of mental illness due to lost workdays and State health service provisions.

[12] Epidemiologists seek to discover a "web of causes." Through the surveillance of populations and systematic data collection of disorder frequency, changes are observed, and hypotheses constructed between disease and cause in order to find causal variables (Oppenheimer, 1992). Since the cause of depression is unclear, this linkage is tenuous.

Ussher, 1991; Russell, 1995). This framing allowed the overdiagnosis of each "disorder" to occur at alarming rates and new, largely untested treatments to receive broad public support. Like the cultural panic that caused the epoch of hysteria, depression can be viewed as a panic that grew to epidemic proportions (Showalter, 1997) fortified by the marketing of epidemiological research.[13] The use of soundbite phrases to market antidepressant drugs illustrates a neuroscience culture that condenses certain expert "findings" for consumers considered either incapable of understanding or uninterested in depression science, or who rely on experts to filter health information. The following section addresses how the rationalities and assumptions belying depression epidemiology and genetic and biochemical research on depression are translated into consumer scripts and soundbites that appear reasonable but in fact distort the breadth and findings of research.

DEPRESSION RESEARCH TRENDS

Most neuropsychiatric depression research assumes, at the very least, that the *potential* to develop depression is passed through genes, and much assumes that genes are a definitive cause of depression. This is the norm despite that a genetic or biological cause of depression has not been confirmed and is instead *suspected* due to biochemical or brain wave differences among some affected individuals. Major genetic research on twins and on genes themselves has resulted, however, only in suggested findings that genes *cause* depressive symptoms. These suggestions are largely presented as conclusive findings in consumer literature, even as such texts admit that *research* has *not* in fact shown that genes or biochemical changes *cause* depressed symptoms.

In their well-known consumer text, *Overcoming Depression* (1997), writers Demitri and Janice Papolos direct readers through the maze of depression diagnostic and treatment information in a manner representative of the field.[14] The pair, who are a doctor and writer, respectively, admit that genetic research on families and genes has yielded *hints* but no proof of genetic determinism. While claiming that studies determined a 67% incidence of depression among separated identical

[13]Diagnoses of hysteria in 19th century England are now considered responses to a panic among doctors and husbands that created an epidemic (Porter, 1987; Smith-Rosenberg, 1985; Showalter, 1985, 1993). Showalter and Smith-Rosenberg characterize hysteria as a diagnosis of social role anxiety, where women, generally of upper class status, who were unable to fulfill expected social roles were diagnosed with hysteria and then treated with torturous therapies that encouraged gender role compliance. During the second half and particularly the last quarter of the 19th century, such disorders of "nerves" were considered actual disorders of the nervous system ("degeneracy"). It was believed that a healthy human nervous system was required to exercise reason, which was coordinated by will (Oppenheim, 1991, p.269). The values of science, modernist culture, and social Darwinism (suggesting that hysteria proved some humans to be inferior) colluded in this British period so that disorder was believed to compromise social purity, national morality, and mental hygiene.

[14]The text was originally published in 1987. Demitri Papolos is a psychiatrist who also authored a core book about depression, which was published by the National Alliance of the Mentally Ill. His wife and co-author, Janice Papolos, is a well-established medical researcher. I refer often to this text, as it is most liberal and thus least likely to overstate research findings to consumers.

twins, Papolos and Papolos admit that the 33% rate of non-depressed twins might indicate that the disorder is not wholly genetic (1997, p. 57). The pair also criticizes the widely hailed "Amish" study of genetic depression markers noting that later studies failed to replicate findings.[15] They conclude it is likely that depression among such groups could be *either* genetic or situationally-induced, writing: "While there appears to be a significant genetic component to the *risk* of depression, it is by no means certain that a gene or genes are necessary or sufficient to produce unipolar or bipolar depression in all circumstances" (my italics, 1997, p. 64).

Similarly, in their consumer text, *A Comprehensive Guide to Mental Health* (1995), Hales and Hales, also writers and not research scientists, admit that the cause of depression is unknown: "Various combinations of different factors—biological, genetic, chemical, psychological, social, developmental, environmental—*may* lead to major depression"[16] (my italics; p.17). Noting the *lack* of causal findings, the pair still contends that neurobiological research has *shown* depression to be "a complex biological illness that affects the delicate balance of brain chemicals, the signaling system used for communication between neurons, the flow of blood through the brain, the hormones that regulate dozens of body processes and the mechanisms involved in sleep and wakefulness" (1995, p. 57). Even while arguing for a holistic approach to determine cause, Hales and Hales reduce depression to a neurobiological malfunction. Though research suggests that depressed individuals have different brain images and biochemical levels, to deduce that depression is *therefore* a biological illness is to ignore other correlated variables and assume the biopsychiatric paradigm—a tendency that occurs across many consumer texts that report depression research.

Most depression research focuses on the genetic potential to develop the disorder, employing single cause models that, in fact, do not concur with widely accepted neuropsychiatric theories of the "complex" brain. Neuroscientists admit that the brain is a not a simple organ, but a "plastic" one that operates in a complex system (meaning systems within systems), where functioning and dysfunction are produced by *multiple* causes. Though researchers generally agree that environment plays a part in causing depression, they have not overcome difficulties in measuring the multi-faceted phenomena of "environment" in addition to genes or neurochemicals as depression *causes*. Contending that biological differences ultimately signify a biological depression, researchers settle for single-cause models that *assume* but do not test environmental influence.[17] In the process, single cause

[15] Because Amish communities marry and reproduce internally, many scientists consider the "population" to be an ideal research site.

[16] Still, they still attribute a likelihood of biological cause, writing: "As with so many medical conditions, some individuals appear to have a biological vulnerability that makes them especially susceptible" (p. 17).

[17] Complex systems are characterized by the ability to be resilient in the face of change—to make adjustments and maintain harmony. Some researchers assume that their simple system research findings can be reevaluated thereafter in a complex system model. In other words, findings that

models are represented as appropriate in research summaries, health policy promotions, and consumer and advocacy literature. As a result, consumers are likely to consider the single-cause model as unproblematic and thus view the body *alone* as cause. Today's mental health consumers are misinformed by distorted depression literature, but believe themselves to be highly *informed*. As such, consumers accept diagnoses of major depression and antidepressant subscriptions to treat a broad range of depressive symptoms ranging from suicidal ideation to everyday blues. In the new millennium, antidepressants have become some of the most widely used drugs in America, and neuropsychiatry is culturally confirmed as the expert psychiatry.

NEUROBIOLOGICAL RESEARCH

The newest neurobiological research investigates hormonal, biochemical and brain image differences, employing single-cause models. Though these studies greatly outnumber psychosocial research studies, the lack of comparable numbers of depression studies in psychosocial research is rarely addressed in consumer texts or popular media.[18] A small piece of the convoluted history of serotonin research illustrates the tenacity of neurotransmitter researchers, despite findings that continually question these theories. Serotonin (5HT) is a neurotransmitter that allows nerve cells to communicate with one another by transmitting messages from one nerve cell to another. Essentially cell 1 fires, whereby serotonin is shot into the synapse between 1 and 2, activating cell 2 to fire and so on. It is believed that in the "normal" reuptake process, serotonin is removed from the synapse after cell 1 fires—that its channels open and serotonin is sucked back into cell 1. Depression is said to occur when there is too little serotonin in the synapse, so that the two cells cannot communicate. A class of drugs called Selective Reuptake Inhibitors

serotonin underproduction *causes* depression can then be pressed into a complex model that later considers environment. In such a case, however, serotonin is already established by science as "the" cause, making any other finding an instigator or conspirator in the serotonin cause. In that faulty model, stress could in fact be a cause of depression, and serotonin changes a *response to* depression. This model seeks single causes to explain functioning, in vacuum-like designs that generally fail to account for the possibility of other factors (i.e., somatic of the body or environmental stresses). Most depression research thus produces findings that are imperfect, often contradictory, and insignificant without contextualization in a complex system paradigm.

[18]Quite to the contrary, Papolos and Papolos boast that "the progress made in the study of mood disorders in the past decade is unmatched by that in any other area of psychiatric research, especially visible in the areas of clinical diagnosis, epidemiology, sociology and psychology of depression and mania, as well as the areas of genetics and molecular biology" (1997, p. 72). Still, the authors are forced to admit that "with all these recent advances, there is still no answer to the question 'what causes these disorders.'" Genetic, biological and psychological studies provide "clues, but clues only . . . few of these findings have achieved status as fact and none of the theories provides a broad enough framework to encompass the findings" (1997, p. 73). Though Papolos and Papolos are compelled by biopsychiatric findings, they do infer that the research paradigm is overly narrow. Additionally, as shall be shown, biopsychiatric research methods tend to be inconsistent with scientific method rules, and systematic only in seeking a discrete cause.

(SSRI's), which includes Prozac, are believed to inhibit reuptake by blocking the channels that would suck away too much serotonin.[19]

The mass media's embrace of neuroscience would have consumers believe that serotonin theories and SSRI drugs are widely embraced by depression researchers. Serotonin theories, however, are hotly and continually debated. Neurotransmitter researchers have deviated between concentrating their efforts on serotonin and norepinephrine throughout the past decades, due to ongoing findings that challenge existing neurotransmitter theories. It was found, for example, that tricyclic antidepressants prevented reuptake of both serotonin and norepinephrine. As a result, a subsequent theory—notably convenient for the pharmaceutical industry—was constructed, contending there now existed *two different* types of depression: one caused by norepinephrine and the other by serotonin metabolic disorders. New controversy arose when other new antidepressants successfully treated depression without significantly inhibiting *either* serotonin or norepinephrine, and when amphetamines—that *do* prevent reuptake—*failed* to relieve depression. Perhaps the greatest challenge to serotonin theories is the newer tricyclic antidepressant Tianeptine. This drug, which does the exact *opposite* of SSRI drugs, enhancing rather than preventing serotonin reuptake, has been reported to be effective in treating depression.[20] But such findings rarely get much media attention. As such, the cultural authority granted to serotonin theories is likely due to the mass media's touting of SSRI's and reports of extensive consumer antidepressant use, in addition to consumer response to pharmaceutical advertising claims of drug effectiveness and fewer side effects.

It is rarely reported that Prozac and other SSRI's are *not* effective on many individuals, and that neuroscientists do not know *why* SSRI's work to relieve only *some* people's depression. The paradigm of serotonin theory is indeed contested by many researchers, including psychiatrist Peter Breggin.[21] Despite a lack of

[19] Serotonin and norepinephrine have been most implicated in depression and mania. It is alleged that depression results in cases where too few serotonin or norepinephrine neurotransmitters are synthesized and released (whereby messages are not moved to receptors), and mania results when too many neurotransmitters are released. These findings form the theory for the popular antidepressants, Selective Serotonin Reuptake Inhibitors, such as Prozac, that inhibit firing cells, *block* neurotransmitter reuptake and thereby maintain transmitter levels so that messages can be *moved through* synapses (Papolos, 1991, pp. 81–82).

[20] Western studies predominantly show effects in rats (Nowakowska, Kus, Chodera, & Rybakowski, 2000). In Eastern Europe, the drug was found to be effective in humans with late-life depression (Andrusenko, Sheshenin, & Iankovleva, 1999) and depression coexisting with other psychiatric disorders (Vein & Vorob'eva, 2000). Recently, Wagstaff, Ormrod, and Spencer (2001) report that Tianeptine appears to be as effective as fluoxetine (Prozac).

[21] Some consider Breggin to be extreme in is his conception of depression as an emotional problem and his anti-drug stance. Many of his criticisms, however, are supported by a myriad of psychiatrists and citizen groups alike. For example, numerous University research studies, international medical journal *the Lancet*, the (Ralph) Nader Public Citizen Health Research Group and the Prozac Survivors Support Group warn of the dangers of Prozac, that include the risk of violence and suicide. There are various arguments against the organization of SSRI research. Breggin (1991) problematizes research that bases the faulty transmitter theory on SSRI *impact*. The research, he contends, is primarily done

understanding of SSRI's, and despite continual challenges to neurotransmitter theories, however, researchers continue on a path specifically seeking to uncover a neurotransmitter reuptake problem.[22] In turn State health policy primarily supports the use of antidepressants to treat broad spectrum depression, despite *not knowing* how the drugs work, or why they are not broadly effective while other drugs that don't fix serotonin levels are sometimes effective.

What should concern researchers, scholars, doctors and consumers is that the mass media and consumer literature each fail to report that neuroscience paradigms *are* contentious, that harmony does not reign within this community. Consumers have an obvious stake in learning that drug makers and researchers don't know how SSRI's work. Still, depression research summaries, mass media reports, Prozac labeling information, Eli Lilly's Prozac Website and Prozac advertisements in print and on TV all fail to note that scientists don't know how the drug works.[23] Additionally, consumers are rarely made aware that Prozac is approved for use on individuals with major depression, but is primarily and most effectively used to treat individuals with minor or shadow depression.[24] These sources also fail to report that Prozac was approved by the FDA based on research that is questionable given that testing lasted only five to six weeks, and involved only 230 people.

on "sluggish" serotonin, rather than normal or hyper serotonin levels, and thus, there is no evidence that making serotonergic nerves more active can help people overcome emotional problems. The theory is that reduced serotonergic neurotransmission causes loss of impulse control, leading to an extraordinary range of behaviors including murder, suicide, and out of control behaviors such as hyperactivity in children, delinquency in teenagers, and depression. The inclusion of depression in the category of impulsivity requires "a stretch of the imagination" according to Breggin. Still, it has gained support in national efforts to explain social problems such as violence in America (p.155). It is more likely, Breggin suggests, that the *drugs* themselves, which block the normal regulatory serotonin activities of the brain or intend to produce brain imbalances, cause dysfunction (1994, pp. 155–156). Breggin also contests Eli Lilly's claims that Prozac is a "clean" drug that only affects serotonin levels; he argues that because serotonin itself affects the whole body—all its points and functions—no one can grasp its overall impact on the brain and body.

[22] Scientists readily admit that they are unsure exactly how antidepressants work. Papolos and Papolos write that it often takes three weeks for a person to experience relief from depression "for reasons the researchers are still puzzling" (p.153). And while researchers know that SSRI drugs work on serotonin levels, they are unsure how the drugs decrease depression, why they work only for some depressed individuals, and why the SSRI drugs *sometimes* effectively treat *other* ailments including drug abuse, eating disorders, and obsessive-compulsive disorder, among other phenomena.

[23] Prescribing information on www.prozac.com (accessed July 9, 2001) states that "the antidepressant, anticompulsive, and antibulimic actions of fluoxetine are presumed to be linked to its inhibition of CNS neuronal uptake of serotonin." In employing the word presume, the statement only vaguely notes that Prozac seems to inhibit serotonin reuptake. The subsequent information provides study results that are shaped to engender confidence in findings that could create skepticism. For example, study periods of Prozac are often only 13 weeks long, though the drug is said to take 6–12 weeks to achieve an effect, hence the findings could be placebo effects. More, placebo effects are often strikingly similar to Prozac in the treatment of depression, OCD, and bulimia. Finally, myriad types of depression, from mild to major, are collapsed in the information as simply "depression."

[24] The 1994 tests that won the drug FDA approval were conducted on individuals with *major* depression (Kramer, 1993, p.66). Since then, a few studies have tested Prozac (fluoxetine) on minor depression, with mixed results. Robinson et al. (2000) found nortriptyline significantly more effective in treating minor depression (in stroke victims). Tollefson et al. (1993) found that fluoxetine was associated with statistically significant lower depression scores in individuals with major and minor depression.

Additionally, over the past decade, researchers have contended that Prozac is no more effective than other antidepressants (Breggin 1991, pp. 168, 162).[25] Consumers need to know that the prevailing depression script is widely contested. The stakes—the biochemical condition of our brains—are high. Instead, the lack of attention in mass media and advertising to the disharmony and multitudinous theories of depression cause and treatment suggests to consumers that the soundbites are correct: depression is an illness, antidepressants work.

DESPITE QUESTIONABLE EFFECT, SSRI's AS CURE

Biased antidepressant information constructed for and promoted to consumers has the credibility of scientific knowledge due to specific marketing strategies. In most consumer depression texts antidepressants are recommended as *cures*, independent of causal findings—genetic, biochemical, environmental or other. This is consistent with the trend where many psychiatrists recommend drug use to treat depression despite a lack of etiological knowledge of depression, and despite that antidepressants often don't work or are merely as effective as placebos.[26] Indeed, consumer texts present problematic research findings as proof that antidepressants are the route to cure across a broad spectrum of depressions.

To wit: Hales and Hales (1995) contend that "most cases" of major depression can be treated successfully, usually with psychotherapy, medication or a combination of the two.[27] Papolos and Papolos concur, proclaiming that the effectiveness of antidepressant drugs is "now firmly established in research studies" (1997, p.121).

[25] While Breggin and others have questioned the validity of the FDA testing, the mass media have generally not done so. Breggin notes that in the late 1980s, the *Physician's Drug Reference* (known as the PDR) confirmed the lack of long-term controlled trials, noting that Prozac had not been confirmed to have long-term effectiveness. Additionally, he notes that the journal *Treatments of Psychiatric Disorders* had a conservative response to Prozac, noting that it not yet been proven to have greater efficacy or reduced toxicity in consumers. Finally, prior to the height of Prozac hype in the early nineties, the *American Psychiatric Press Textbook* noted that Prozac was as effective as Imipramine, one of the oldest antidepressants (Breggin, 1991).

[26] Peter Breggin cites a number of double-blind research trials conducted in 1992 showing that a large percentage of patients felt improved as a result of taking a sugar pill, demonstrating that new and old antidepressants were no more effective than placebos. The drugs tested were Amoxapine, Maprotiline, and Trazodone, by Roger Greenberg and Seymour Fischer of the Department of Psychiatry and Behaviors Sciences at the State University of New York Health Sciences Center in Syracuse. Drug companies have been so confounded that now, prior to testing an antidepressant, they often first conduct a "placebo wash-out" to screen out all patients responsive to placebos (1994, p. 204). In the past year, too, studies have shown that placebos are as effective as antidepressants in treating major depression (Walsh et. al., 2002).

[27] Individuals who do not respond to one therapy are likely to respond to another, and short term psychotherapy (especially cognitive-behavioral therapy and interpersonal therapy) alone works in over half of mild to moderate episodes of depression. Hales and Hales write "antidepressants are effective in more than half of those with moderate and severe depression and may be useful in treating mild depression in individuals who do not improve with psychotherapy alone" (1995, pp. 67–69). The pair also claims that severe or chronic depression *usually* requires biological treatment, "most often with medication," though they fail to cite studies backing up these recommendations (1995, pp. 67–69).

Yet, critics of rampant antidepressant promotion in the US contend that, though it is difficult to determine the effectiveness of depression drugs, effectiveness has indeed *not* been proven. A host of research over the past decade has, in fact, determined there is often little discernible benefit to using drugs rather than psychotherapy, particularly for minor depression, and has found placebos to be as effective as antidepressants.[28] Having reviewed the most recent studies comparing psychotherapies (interpersonal, cognitive and behavioral) used to treat depression and tricyclic antidepressants, Dr. Michel Thase (1999) contends that antidepressants are effective in combination with psychotherapy *only in the treatment of individuals with severe depression.* Subsequent studies verify these conclusions.[29]

[28]Breggin notes that 25% of depressed individuals spontaneously recover during the first month and 50% recover *spontaneously* in a few months (1991, p.158). Fischer and Greenberg (1989) conclude that the most positive reviews by drug advocates showed *no difference* between antidepressants and placebos, with substantial improvement found in only 25% of cases. Controlled studies of the newer, strongly promoted antidepressants (including SSRI's) showed that 62% of consumers showed no response to the drug, while other studies favored *psychotherapy* over drugs (Fischer & Greenberg, 1989, pp. 16–19). The authors also charge that powerful investigator bias was at work since some teams found no efficacy while others repeatedly found considerable efficacy, with placebo efficacy ranging, for example, from 0 to 91% (p. 21). Additionally, according to Fischer and Greenberg, evaluative criteria of improvement were not standard but behavioral, often referring to the patients' weight gain, sleeping habits, or self-reported psychological state. The authors thus conclude that years of research have not provided a justification for antidepressant use. The most comprehensive study to date of antidepressant effectiveness conducted for the NIMH by Elkin et al. (1989), compared cognitive-behavioral therapy, interpersonal psychotherapy, antidepressants, and placebo over a sixteen-week period. Senior author Irene Elkin commented in the *Archives of General Psychiatry* that antidepressants were shown to be successful at treating depression *at the same rates as all other treatments*—including short-term and long-term therapy, placebo pills and doing absolutely nothing. More specifically, less depressed patients, representing 60% of the study, showed equal responses, while severely depressed individuals representing 40% of the study, received a relatively greater benefit from a combination of antidepressants and interpersonal psychotherapy rather than the placebo. Dr. Philip Long contends that the drugs are not effective in treating minor depression, and that most nonsevere depression spontaneously recovers, citing a recent study finding that 40% of depressed people recovered in three months, 60% in six months, and 80% in a year (Long, 1998, p. 2). The study, asserts Long, demonstrates that in mild cases of depression lasting four months or less, brief therapeutic visits are sufficient until the depression spontaneously recovers. Breggin (1991) goes a step further, contending that the study devalues the effectiveness of various forms of *nonpharmaceutical* therapies to treat depression, which are proven by other studies.

[29]In cases of *minor* depression, it was found that psychotherapy was equally or more effective than drugs and that the effects were longer lasting than with drug therapy. As of 1999, studies of SSRI antidepressants were still in progress, and none had compared drugs such as Prozac (fluoxetine) to psychotherapies for depression (Thase, 1999). Similarly, in regard to children and adolescents with MDD and dysthymia, a 10-year review of literature concluded that fluoxetine and cognitive-behavioral therapy were equally effective, recommending a combined course and further research (Birmaher, Ryan, Williamson, Brent, & Kaufman, 1996). A 1998 study of children and adolescents with mild to moderate major depression, also found that fluoxetine was no more effective than cognitive-behavioral psychotherapy, thus questioning the use of drugs to treat children (Birmaher, 1996). More recent studies of individuals with major depression again show that psychotherapy in combination with fluoxetine (Prozac) was more successful than the drug treatment alone, the former successfully treating 59.2% of individuals, versus the latter treating 40.7% (DeJong , Kool, van Aalst, Dekker, & Peen, 2001). Additionally, a systematic Medline review of depression treatments in acute care elderly individuals with major depression found that short-term treatments of fluoxetine and other SSRI's, heterocyclics, or psychotherapy alone were each found to be only modestly effective,

In addition to the lack of knowledge regarding how antidepressants work, studies have found that many antidepressants have deleterious side effects. While side effects of MAOI's can be life threatening and tricyclics can be severe, SSRI's (including Zoloft, Prozac, Paxil, and Luvox) are contrasted in consumer literature as having the least severe side effects.[30] Still, it is problematic that manuals often fail to inform consumers that the drugs have not been tested for long-term use. In some populations, such as children, drugs have been used off-label for over a decade and have only recently begun to be tested for such use.[31] Additionally, many consumers and psychiatrists contend that Prozac causes depressed people to become suicidal or violent. While Eli Lilly has denied the charge, the company has recently received approval to market a new version of Prozac, devoid of the chemicals that make consumers violent.[32] Even Prozac celebrant Peter Kramer

disputing claims that fluoxetine alone had a superior effect (Cole, Elie, McCuster, Bellavance, & Mansour, 2000).

[30]The use of MAOI's (a class of tricyclic antidepressants called monoamine oxidase inhibitors) can cause seriously high blood pressure in response to numerous medicines and a large array of food items including meat, soy, alcohol, yeast, and fruits. Side effects for tricyclics include sedation, increased heart rate, dizziness, rashes, tremors, altered orgasmic function, and weight gain (Papolos, 1997, p. 154). Lithium, used to treat manic depression, can result in toxic drug levels, and interact negatively with many medications. Antidepressants such as Haldol and Thorazine (to treat symptoms of depression and psychosis) cause involuntary facial muscle spasms, akinesia (stiffness and spontaneous gestures and speech) and akathisia (internal restlessness) symptoms treated with other drugs. Additionally, neuroleptics (to treat bipolar disorders) cause the severely disfiguring, untreatable effect of tardive dyskinesia, characterized by facial grimacing, lip smacking, chewing, sucking and other movements of the tongue, and physical writhing. As such, other medications are generally substituted on a short-term basis. Wellbutrin (bupropion, a widely used antidepressant of the nineties, distinct from the tricyclics, MAOI's and SSRI's) initially caused seizures and was withdrawn from the market, though a lower dosage is under consideration by the FDA. Wellbutrin also shares common SSRI side effects, including insomnia, nervousness, nausea, diarrhea, and headaches, which subside within two to three weeks according to Eli Lilly. Less common side effects of SSRI's include drowsiness, yawning, sweating, rashes, and waning libido (Papolos, 1997, p. 165).

[31]Many critics, including Breggin, contend that the FDA approves psychiatric drugs whose trials are faulty or insufficient, and where pharmaceutical influence peddling is a common practice, such as in the case of Prozac (1995, pp. 166–167).

[32]There are different reports of the 1990 study conducted by Harvard researcher Martin Teicher and colleagues, published in the *American Journal of Psychiatry*, whereby 6 patients became obsessed with suicidal thoughts two to seven weeks after starting to use Prozac, four of whom made violent or suicidal attempts. Papolos and Papolos argue that the media failed to report that four of these patients used additional other medications, while five of the patients had suicidal thoughts or actions in the past though they were not assessed as actively suicidal at the time of Prozac use. Dr. Teicher contends that the responses among patients in the study were not significant. Teicher, however, speculates that because the drug effects the serotinergic system that *may* play a role in mediating aggression, Prozac may cause some individuals to become aggressive. Breggin concurs, arguing that some studies show that brain systems can become sluggish as a result of Prozac use, and refers to studies cited in the previous footnote, warning of the dangers of Prozac, including risks of violence and suicide (1995, p. 158–159). David Healy contends that over 70 studies prove, Prozac causes violence—cases that have been accepted into legal cases against Eli Lilly (Prozac Survivor's Website, http://www.pssg.org/forsyth.htm; accessed 8/00.) Teicher's study was followed up during the 90s by numerous studies (including King et al., 1991; Rothschild & Locke, 1991; Wirshing et al., 1991) showing that suicidality appeared to emerge in individuals taking fluoxetine. A decade later more research has been compiled linking Prozac to suicide, as noted in Joseph Glenmullen's text *Prozac Backlash* (2000).

contends that concern over unforeseen or tardive effects of Prozac are warranted because the drug has not been around long enough for anyone to know its long-term effects. (1993, p.312)[33] However, addressing present day consumption alone and dismissing claims of violent side effects, Papolos and Papolos confidently claim that over 21 million people have used Prozac safely and effectively. Thus, while some consumer manuals fleetingly admit to shortcomings in the neuropsychiatric paradigm, they generally fail to present comprehensive information of contentious research paradigms and findings, and widely recommend antidepressant use as the common, safe and logical treatment choice.

IDEALIZING THE MAGIC BULLET

Why does the spin of muddied discourse as knowledge appear plausible to consumers and to consumer manual authors? This constrained information appears reasonable due to the historic rules and assumptions of psychiatric discourse that have become habitual in culture and thus normalized.[34] The narrow, modernist approach of depression research is constrained by tenets of biomedicine it adopted in the mid-20th century in the U.S, which have become common sense in the US with publicized medical advances. Like the discreet disease medical model, the single cause model seeks a cause that is theoretically treated by a single agent. The mind is thus reduced to an organ understood and repaired by biomedicine—via the so-called magic bullet theory.[35] In turn, biopsychiatric logic is reductionist—it dilutes things to their smallest parts and studies them in isolation. Thus, biopsychiatry *assumes* that the allegedly sick body part or function, i.e., neurotransmitter function, because it is of the body, can be deemed as cause. Belying this assumption is a psychiatric system based on the dualization of mind/body, and an unfailing faith that biotechnology can repair the biological (regardless of its etiologies, including abuse and trauma) and is more efficient than environmental treatments such as psychotherapy. "Cause," then, is conscripted into the service of neuropsychiatry—it is believed to be knowable via reductionist methods. Circulated through campaigns for antibiotics, vaccinations, and the AIDS epidemic, consumers "know" that cause produces disease and disorder.

[33] Also, Breggin worries that Prozac could cause short-term behavioral problems and long term brain damage and dysfunction; he argues that clinical studies on rats have shown that the body responds to the increased availability of serotonin in the brain as an intrusion. One way that the brain tries to overcome this effect is through "downregulation" where it reduces the number of receptors for serotonin. Studies show that over time in animals, receptor numbers diminish, even in the highest centers of the brain such as the frontal lobes and cortex, which regulate thinking and feeling (1994, p. 88). Researchers are unsure whether downregulation is a permanent effect in humans.

[34] I am referring to Michel Foucault's theory of discourse analysis, that calls for an analysis of discursive assumptions and rules, particularly contradictions that systematically embraced, become incorporated into discourse as reasonable (1966, 1976).

[35] American psychiatry adopted the biomedical model in the 1950's—a controversial move, as it suggests that psychiatric symptoms can be decontexualized as primarily biological or genetic phenomena.

The modernist model also reifies the taxonomization of feelings into scientific categories, which again become part of a cultural commonsense consciousness. Due to its modernist trust in observation, psychological or social symptoms are considered real when made *visible*—interpreted as biological. Distress, defined as "symptom," is filtered through a framework of dichotomies and reduced to biological illness. Thus, biomedicine defines nature in a manner that *excludes* consideration of cause or etiology. The nature of a thing is considered physical and knowable *independent* of representation, its structure can be "laid bare in morbid pathology as a pathogonomic thing" (Kleinman, 1995, pp.29–30). As such, the simple observation of distress, framed as symptom clusters, is sufficient to introduce psychiatric diagnosis, which is then linked through reductionist practice to a biological cause, such as neurotransmitter problems. This scenario is all the more curious given that biopsychiatry assumes a *complex* model where *multiple* "causes"—environmental and biological—result in an ultimately ill (depressed) brain. Belied by faith in observation, neuropsychiatry employs reductionist practices to study *single* sick components of the brain.

Consumer literature idealizes the single cause model, failing to note this problematic, and failing to consider broader, more complex models. Consumer awareness and alternative treatment choice is limited by the act of obscuring reductionist logic of the depression script. The logic assumes that a single factor existing in a complex system, serotonin for example, can be analyzed in isolation. It then assumes that the newly theorized item (i.e., the theory that serotonin reuptake impacts mood) can be reinserted into the untheorized complex system, impacting all other items of the system. Such theories create a "worldview," constructing assumptions and affecting understanding about all other elements in the system—without actually studying them. Reductionist science creates global "knowledges" about the role of the complex brain in depression; for example, it prioritizes the role played by serotonin in the complex phenomenon called depression, when serotonin has only been studied *in isolation*. Isolated serotonin research contributes to the larger story of neurotransmitters as essential to stable mood, reifying the idea that depressed individuals need brain drugs. This lack of critique in consumer literature's presentation of the discreet disease model translates as abiding support of a presumably comprehensive, little contested model, and thus fosters consumer faith in biopsychiatry.

CONSTRAINING THE PSYCHOSOCIAL, CONSTRUCTING THE BIOPSYCHIATRIC

The findings of psychosocial research and choices for psychosocial treatment options are rarely visible to consumers for many reasons, the most obvious being the dominance of neuropsychiatric research and SSRI's in media coverage of psychiatry, consumer desires for quick fixes and HMOs' tendencies to pay for cheaper

drugs.[36] Despite its lack of attention in consumer literature, psychosocial research has shown stress to be a causal factor in major depression and minor depression, and in other disorders where depression is a symptom[37] (American Psychiatric Association, 1994, p.342). The *DSM-IV* admits that episodes of major depressive disorder "often follow a severe psychosocial stress, such as the death of a loved one or divorce." The predilection for biopsychiatry is evident even in studies that consider how stress affects neurochemicals and neurotransmitters, thereby "causing" a biological depression. Consumer and psychiatric literature fail to mention psychosocial theories of cause, instead, reducing a host of conflicting biopsychiatric research to the statement that depression is a biological phenomenon. The practice sets in motion the repetition of discourses that represent biopsychiatric logic as the expert science and psychosocial stressors as ancillary factors irrelevant to consumer's treatment choices. Fostering and enabling informed consumer choice is evidently not among the literature's goals. Uninformed that psychotherapy is often equally affective in treating depression, consumers learn that depression is due to sick genes or biochemistry, and is best treated by psychopharmaceuticals. This logic collapses a broad spectrum of depression, links depression with pharmaceutical treatment, suggests that antidepressants are *generally* effective and safe for a

[36]Psychiatrist Peter Breggin (1994) is among those who critique the biopsychiatric framing, instead defining depression as an emotional state of desperation, despair, or other responses. Depression does not necessarily have a biological or genetic cause, contends Breggin, but rather *feels* physical and can produce physical changes in the body such as hormonal, cardiovascular, stress, and other problems (1994, p.186).

[37]Depression has been strongly correlated with stress, though researchers call for more studies to understand the multiple factors, including stress, that can contribute to bringing on major depression (Meyer, Chrousos, and Gold, 2001). It is notable that many researchers currently call for investigating the neurobiological impact of stress. (Kaufman & Charney, 2001; vanWest & Maes, 1999). Depressed symptoms are also prominent among individuals diagnosed with "adjustment disorders" and posttraumatic stress disorder, for example, where stress is believed a causal factor in the depression. Psychosocial research has also correlated depressed symptoms with abuse. Research shows that diagnoses of depression, as well as borderline personality disorder, dissociative disorder, and posttraumatic stress disorder tend to be assigned to women survivors of sexual abuse (Meiselman, 1990). Depression is the most consistently reported *effect* of child sexual abuse, among a cluster of other symptoms including low self-esteem, fear, guilt, interpersonal problems, and self-destructive behaviors (Molnar, Buka, & Kessler, 2001; Browne & Finkelhorn, 1986). A 1993 study found that as many as 67% of children surviving incest were diagnosed with depressed symptoms (Koverola, Pound, Heger & Lytle, 1993, p. 393). Depression is also regularly correlated with suicide in abuse survivors. These findings challenge assertions that depression has solely genetic or biochemical origins, and trouble research approaches that single out biological causes without consideration of psychosocial stressors. Standard diagnosticians seek to distinguish between a stressor-induced depression and so-called "organic" depression, but lack substantive "data" demonstrating *how* stressors induce depression, since little psychosocial research receives State or pharmaceutical funding. Instead, surveillance tools and questionnaires are employed to locate depressed symptoms, the client is assumed to be biologically depressed, and evidence of intense traumatic experience precipitating the symptoms are considered irrelevant in treating the "ill" brain. The current biopsychiatric trend links personality and other disorders with depression, thus increasing numbers of doctors and psychiatrists who pursue any depressed symptoms as possible signs of major depression and increasingly prescribe antidepressants. Research that targets violence against women and children as causal factors and remedies that call for changes in social structure and male behavior lack the glamour of quick-fix pharmaceuticals, and are not widely funded or publicized by the press.

broad range of depressed individuals, and ultimately ushers a broad public (whose distresses range from the blues to suicidal ideations) into standard depression diagnosis and treatment.

Finally, research reports and consumer literature fail to address whether it is important to determine cause in depression research—for example, whether stress-induced depression might be most efficiently and safely treated by psychotherapy. Some critics contend, for example, that biological changes in depressed people are simply evidence of biological differences, not of a biologically caused depression. Others contend that changes in biochemistry can be a *result* of depressed symptoms (sadness, hopelessness) rather than a cause. Depression researchers declare these questions insignificant, contending that biological differences signify defect or illness—that depression is ultimately biologically based. The question of cause(s) is made mute, since the biological phenomenon is "the problem." This intriguing claim is rarely laid bare for scientists or consumers to critique. Indeed the depression research paradigm makes two very different claims. At first, the paradigm admits to—but does not investigate—environmental influence as a causal factor in depression. On the other hand, the paradigm that is deployed in consumer literature seeks a single biological cause and remedy for depression. The first approach assumes that environment can affect and even repair the biological, meaning that psychotherapy can cause biological repair; the latter fails to realize that assumption. The first admits to environmental interference and the possibility that single cause research findings might be problematic; the latter does not. The first does not necessarily link cause to manifestation and treatment; the latter infers that linkage and thus represents biotechnical therapies as logical treatment.

Medical theorists Parker and Hadzi-Pavlovic (1996) comment that the history of classification and use of antidepressants makes a single biopsychiatric cause and treatment *seem* truthful. Current categories are simplistic, static, force homogeneity across heterogeneous elements and "almost certainly provide invalid definitions across melancholia" (p. 277).[38] The discrete disease model of depression is preserved by literature that serves as a fortress, glossing the research process, overestimating findings, and simply declaring depression to be an illness. Bolstered by this claim, pharmaceutical companies and consumer manuals promote antidepressants as logical treatments for ill bodies. Because the paradigm of depression research is not clarified in popular literature and because biopsychiatric research remains dominant while psychosocial studies are marginalized,

[38] Additionally, research relates only to the very core (neuro) processor in psychopathology, thus restricting broader etiological investigation. In regard to new antidepressant treatments, for example, the pair contend that the successful response to antidepressant treatments (Lithium, MAOI's, and the newer tricyclics and ECT) became a *criteria* for depression diagnosis in the *DSM-III*—antidepressants became a signifier of the true nature of depression. Additionally, the taxonomical limitations restrict the identification and quantification of etiological factors and treatment to depressive disorder that prejudices treatment. In neglecting to consider both etiology (cause) and context (environment) in diagnosis, this restrictive, inadequate biopsychiatry ultimately fails the patient.

depression research appears to be a unified, troublefree arena. Polarized from framings that address environmental influence and celebrated as the sophisticated science, biopsychiatry becomes a discursive strategy, a camp of "expert" depression knowledge. Uninformed consumers view single cause research finding as "knowledge" that all types of depression are illnesses, and that depression means the brain is sick and requires psychopharmaceutical treatment. In turn, pharmaceutical companies overestimate their findings, claiming that antidepressants simply work. These glossings are well-packaged for sound-bite promotional consumer information: depression is an illness, antidepressants work.

FROM CAUSE TO CURE

Packaged depression information for consumers includes the representation of biopsychiatry as a route to a cure, even while at present antidepressants only "manage" depression. Like the biomedical assumption that a single infecting agent is best treated with biomedicine, ill brain components are considered curable with biotechnology. John Erni (1994) suggests that treatment science produces a knowledge structure based on a circular logic, where the sophisticated micro-inspection of single agents justifies a need for "designer" drugs that reciprocally legitimize that knowledge as key to disease intervention (p.10). The single enigma script "resolves the whole question about 'cause' and thus forges a focus of research," while drugs, i.e., antidepressants, reify the psychiatric paradigm serving as "an interlocutor who holds the entire scientific vision together" (Erni, 1994, pp.10–11). Though cause is not investigated, the most important cause is *assumed* to be the neurotransmitter, leaving other possible causes out of the scenario. This also explains depression research where the *effect* of antidepressants on subjects is used to prove or disprove a theory of neurotransmitter activity.

The paradigm of consumer depression literature is self-replicating—together the *lack* of verifiable findings of cause, of a broadly usable, antidepressant with minimal side effects, and the *lack* of a cure, create a perceived *need* for further research on both cause and treatment. That is, the literature fails to reveal that cause is irrelevant in depression research, and falsely implies a knowledge of cause, and it also promotes antidepressant effect independent of cause. Not only does cause indicate cure, but cure is used to affirm the existence of a depression, and to suggest that a discoverable cause is possible and will lead to cure. Antidepressants hold together this logic.[39] Notably, this framing is *not* destabilized by the logical

[39]Erni is referring to AIDS discourse and AZT research. In regard to HIV research Erni argues that a "microbiological understanding of the virus justifies a need for 'designer' drugs that reciprocally legitimize microbiological knowledge as the key 'truth' necessary for effective intervention into the disease" (p.10). Thus, "AZT validates and perpetuates the whole structure of scientific knowledge, serving as the interlocutor holding the whole scientific vision together."

reversal practiced in research and diagnosis: namely: If A appears depressed, then B (antidepressant) should be attempted; B works (seems to control the depression for this subject, at least for a short time), therefore A was depressed.[40] If a single cause of depression can not be known, then (apparent) drug effect is used to *suggest* a single cause. This dually reversible cause-effect logic is troublesome in that it assumes that cure dictates cause, and, again, avoids addressing possible environmental causes. The assumption that the apparent effect points to a single cause minimizes the *possibilities* of causes and effects, as well as possible relationships between the two, or a broader, more complicated role for "cause."

A byproduct of this scenario is further funding for drug development both because the drugs seem to work, *and* because they don't work well enough. Because drugs are imperfect—they have side effects and work effectively only on some—scientists get funding to improve them. At the same time, antidepressants are promoted as efficient—useful in preventing recurrence and safe for long term use. As Erni (1994) suggests, through the repetition of competing discourses that define the "disease" as both curable and incurable, the question of curing is circumscribed and contained.[41] Because drugs are both reliable and imperfect, a deeper consideration of cure is prevented from arising. In turn, pharmaceutical companies and consumer literature are able to construct the *continual use* of antidepressants as recovery that constitutes a cure, more or less.[42]

Expert and consumer confidence in pharmaceutical drugs is due to discourses that constrain assumptions and framings of the "problem" of depression as well as its treatment. Depression science contains and controls depressed *symptoms* via a paradigmatic framework that *allows for change* (i.e., more broadly defined, or somatically expressed symptoms), but also *confines* scientific practices or changes to biopsychiatry. New depression symptoms are observed and constructed as a *biological* depression, while new drugs offer maintenance depicted as cure. The unbalanced promotion of antidepressants as the first line of defense in depression research summaries and consumer materials of advocacy groups including

[40] Many clinicians seem to find it reasonable to insert problem-treatment logic into the cause-effect logical mode to problem-solve, or rather to determine "the problem": If A problem, then *apply* B solution; if B solution *seems to work*, then A was the problem. This odd logic does *not* use the findings of an apparent antidepressant effect to simply inform the clinician or researcher. Instead, the apparent effect of antidepressants (due to the *assumption* that the medication worked) is used to *infer* that the patient was depressed. In regard to research on sluggish serotonin, the apparent effect of an antidepressant is used to prove the serotonin theory to be either *correct* (the single transmitter problem alone caused depression) or only *partly faulty*. In the latter case, the theory would be expanded to include other neurotransmitters, but not other causal phenomena.

[41] Despite the multitude of competing discourses, a specific biotechnical research paradigm emerges "through the portal" of AZT, buoyed by certain truth claims (Erni, 1994, p.37).

[42] John Erni's insights into AIDS research are instructive. He contends that the fantastic theory of containing the malady "couples curing with controlling in a regime of maximized hyper-rationality. It too is enacted within a discursive boundary that activates—or habitually resuscitates—a set of narratives . . . while, additionally there is an ongoing attempt to disqualify any alternative forms of treatment" (1994, p.36).

NAMI,[43] and the Surgeon General[44] is made possible by a discourse that equates maintenance with cure. Overzealous drug use and promotion, then, is due not only to pharmaceutical company influence, but to the repetitive biopsychiatric script that floats contradictory research findings, reifies the single cause model, and recognizes antidepressant drugs as the route to a (near) cure. Contradictory claims praising and critiquing antidepressants are largely nonexistent in consumer literature, encouraging public confidence and ongoing research monies. The constrained discourse translates easily into marketing schemes.

THE CITIZEN-CONSUMER OF MARKETING PLOYS

Antidepressant marketing schemes sell the idea that depression is an illness, antidepressants work, while authorizing the larger depression script and paradigm. As I have argued, depression literature and the mass media increasingly report depression epidemiological and lab findings in easy-to-digest soundbites that package a range of emotions (from sadness, to lack of motivation and hopelessness) as severe disease symptoms of a biological depression which, *therefore*, require a pharmaceutical cure.[45] By framing the problem as biological, biotechnical solutions are presented as a logical next step by the marketing ploys of State health policy, mass

[43]The NAMI (National Alliance of the Mentally Ill) booklet "Understanding Major Depression; What You Need to Know About This Medical Illness" (1999a; made possible by an "educational grant" from antidepressant producer Wyeth Ayerst Laboratories) recommends drugs to treat major depression, and psychotherapy to treat associated interpersonal problems. The group writes: "The objective of treatment is to lessen the duration and intensity of the episodes of illness and to prevent their recurrence" (p.9). Though treatment can presently only offer the control or prevention of episodes, the booklet's overestimated research findings hold out hope that genetic and serotonin research will, in time, bring a cure. NAMI discourse suggests that, in the meantime, antidepressants that prevent episodes (in some individuals) are *almost* as good as a cure.

[44]The Surgeon General's 1999 report cited an epidemic of untreated mental illness in the U.S. based on NIMH studies, and touted psychopharmaceuticals and antidepressants as remedies.

[45]In the 1980's, the National Institute of Mental Health sponsored what Kutchins and Kirk term the most sophisticated epidemiological research on mental disorders every undertaken in the United States (1997, p.243) that has been heralded by scientists as outstanding scientific research. Five research teams interviewed 20,000 randomly selected adults using the DIS (Diagnostic Interview Scale), searching for the presence of specific mental disorders, employing broad *DSM*-III criteria published just before the study was initiated. Immense amounts of data were released from the study contending that Americans have a 32% chance of having a mental disorder in their lifetime and a 20% chance at any given time. The study also produced extensive data of mental illness "risk" constructed along categories of sex, age, race, and other definers. The study contended that a higher lifetime prevalence of mental illness existed among men (32%) than women (20%), among the young (37%) than old (21%), and among the financially dependent (47%) than the wealthier (31%) (Kutchins & Kirk, 1997, p. 243). Kutchins and Kirk are concerned that the study used methods untested for accuracy and reliability and that little meaning was given to the statistics in the tables. However, because the researchers and NIMH (the sponsoring organization) have grand status in the field, Kutchins and Kirk assume that the study will constitute "the definitive word on psychiatric disorders for years to come." Groups that want to document the prevalence of a particular disorder can simply *refer* to the report and *extract* numbers that support their cause (Kutchins & Kirk, 1997, p. 244).

media, consumer groups and pharmaceutical companies.[46] The affinity between economic and mental health is naturalized by the discourse of neuropsychiatric science, which, repeated across cultural spheres, appears reasonable and natural to consumers. Epidemiological and neuroscientific discourses strategically reiterate the mantra that science can locate, explain and treat depressive disorders to improve both personal functioning and national productivity. Through repetition these discourses normalize the polarization of camps of knowledge, making it seem natural for consumers to choose and trust a "side" in science.[47] As such, consumers come to embrace a single depression knowledge paradigm, employ its terms, and follow its advice. Consumers thus feel comfortable in embracing what is unfamiliar—we trust authors, diagnosticians, policymakers and scientists to name depressed symptoms, link them with the diagnosis of depression, and recommend biotechnical treatments. The marketing of these framings in quick soundbites and with ubiquitous celebration of neuroscience and psychopharmaceuticals sells psychiatry and the expert as the solution to what—two decades back—were common, everyday changes in mood.

NAMI tells us "Depression is an illness." Eli Lilly announces, "Welcome Back," compliments of Prozac, and invites consumers to use Sarafem, offering "Think its PMS? Think Again . . . It could be PMDD." GlaxoSmithKlein consoles us that "There's Hope for Depression" (via Wellbutrin). Soundbites headline the mass media celebration of neuroscience and distorted reports of scientific harmony. Soundbite discourses sell improved personal and economic health as gratifying, strongly recommending biopsychiatric diagnosis and treatment as efficient activities that appropriate consumers and citizens should undertake. These phrases—depression is illness, antidepressants work—convey a confidence in the neuroscience experts, and are designed to speak to consumer anxieties regarding productivity and work. The first phrase is marketed by the common discourses of consumer manuals, research reports, and mass media recommending uncontextualized standard diagnosis and treatment information. The idea that depression is an illness and drugs work becomes an everyday script more than a trusted one—what is vastly repeated seems "right." In turn, productivity—the product of diagnosis and treatment, is circulated through the discourses of other venues. NIMH health policy and Surgeon General reports make consumers keenly aware that there are standards of acceptable consumer productivity, that productivity indicates mental

[46] For example, the Web pages of the National Institute of Mental Health (www.nimh.nih.gov), the American Psychiatric Association (www.psych.org) and the National Alliance of the Mentally Ill (www.nami.org), all define depression as a brain illness and highlight pharmaceutical treatments as the first line of treatment alone or in combination with psychotherapy.

[47] Hayden White uses the theory of strategies or "tropes" to shed light on how speech can mediate between our supposed oppositions. White argues that speech works like discourse itself to mediate "between our apprehension of those aspects of experience still 'strange' to us and those aspects of it which we 'understand' because we have found an order of words adequate to its domestication" (1978, p. 21). In other words, the repetitive structuring of depression discourse makes it seem familiar and reasonable to consumers.

wellness and vice versa, and finally that neuropsychiatry is a logical expert to comment on questions of citizen productivity. At the same time, neuroscience discourses clearly emanate from the assumption that modern levels of productivity are normal, and are ideals of citizen behavior. Though the pieces of the equation "surveillance + treatment = productivity" emanate from the (somewhat) separate institutions of research, policy and pharmaceutical production, consumers take in the discourses of State and psychiatric research in tandem, linking surveillance with treatment, and with productivity. In the cultural sphere of media consumption, consumers logically bundle the depression discourses of neuroscience, health policy and pharmaceutical industry as a cogent narrative. Taken together, the discourses are powerful—they translate to consumers that the loss of mental health can mean the loss of employment and income, or even the loss of the normal self. Conversely, productivity losses are said to be signs of possible decline into full-blown major depression. In turn, soundbites, meant to target desire or anxiety with a product dressed in legitimacy, sell quick fixes to those troubled by sadness or grief—appealing to a fairly large audience, indeed.

Embracing this scenario seems not only logical, but the reasonable act of the ideal citizen. The good consumer-citizen is expected to passively embrace the link between mental health technologies of surveillance and treatment, accept biotechnologies as the *solution* to productivity lapses, and to leave critique to the policy and science experts.[48] In the process, citizens of democratic States come to respect a regime of governmentality where activities of the social sector and consumer culture delimit the individual's possibilities for action, in this case, actions associated with distresses of mood (Foucault, 1976).[49] The framings of mass media and consumer groups suggest that where health and productivity are virtues, reasonable citizens are expected to repair depression (that is, productivity problems) according to expert recommendations, without needing to inquire of the scientific theories or shortcomings, or to scrutinize generalizations.

CULTURE, MEDIA, SCIENCE, AND CHOICE

The internal self-replicating and regulating capabilities, and broadly repeated rules and truth claims of biopsychiatric depression research ultimately coerces consumers. While consumer acquiescence to discursive coercion might be viewed

[48] For more discussion of citizenship and governmentality, see Nicholas Rose, specifically *Governing the Soul* (1994), which provides essential thoughts on how individuals agree to govern themselves by accepting a host of self-scrutinizing technologies. The term consumer-citizen is adapted from Miller (1992) and Cruikshank (1999), who each explain the late capitalist phenomenon requiring a citizen-subject to be dually active and passive—politically docile but fiercely competition in enterprise culture. Their work, in turn, is indebted to the seminal writings of Nicholas Rose (1990) on the topic of how citizens are encouraged to govern their own mental health, thereby largely relieving government of the burden.

[49] Foucault showed that, as such, citizens of democracies are ruled through ingested information, rather than overt coercion.

as an act of choice, the possibilities for informed choice are obscured by literature that collapses contradictory discourses into a homogenous script that blames and fixes the body in isolation. With the broader diagnosis of depression and increasing antidepressant use comes increased respect for biopsychiatry, increased profits for pharmaceutical interests and increased consumer acquiescence to broad spectrum diagnoses in order to improve their productivity. The myth of a single, knowable depression repairable by biopsychiatric treatments is thus useful to the State in managing crises of low consumer productivity. The myth can also be deployed to confront social problems including youth rampage murders, suggesting that diagnosis can rehabilitate both wayward individuals and economies.

NAMI's soundbite proposition "Treat it, defeat it," cogently illustrates enterprise culture, where consumers are willing to accept that depression cures can indicate cause, and demand little research, logic or limitations. Because little skepticism of biopsychiatric process is to be found in mass media, consumer literature, advocacy groups, the pharmaceutical industry, or governmental policy, depressed consumers are left to choose among popularized (drugs) and less popular, belittled psychotherapies. Constantly reiterated by health policy, practitioners and mass media, the depression script becomes difficult to think beyond. Far from representing a studied review of the field, consumer depression literature twists scientific process in the name of some *other* logic intent on marketing biopsychiatry and its products. Altogether, the activities of the psychopharmaceutical industry, researchers and consumer manual authors mitigate against truly expansive democratic discourses that offer consumers the possibility to make fully informed choices based on all of the currently conducted research. Exchanging health for citizenship in our late modern, post-industrial culture, the consumer-citizen has little desire to theorize the trade.

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