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A Psychiatrist's Perspective on Using Drugs

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By Kelly Brogan, MD

When I see new patients, I do not prescribe medication for them. Patients who come to me know that I plan to help them understand "why" they are experiencing "what" they are going through.

Once I have tapered patients off of medication, we use alternatives if symptoms crop up again.

Knowing my basic orientation around the issue of psychiatric prescribing doesn't seem to stop some patients from asking for what they believe will be a quick fix in an antidepressant pill. Where did they learn to make these treatment requests of providers?

Perhaps they are a reflection of the 49% of requests for drugs prompted by "direct-to-consumer" (DCA) advertising by pharmaceutical companies.¹ Fully 7 out of 10 times, doctors prescribe based on these requests made by patients who learned from advertising that they have an "imbalance" that must be fixed with a pill.

In a 10-year period from 1999 to 2008, DCA tripled from 1.3 to 4.8 billion dollars devoted to educating patients about their need for psychiatric medication. The "mass provision" of SSRIs to the public is not a reflection of their well-understood mechanism, of their efficacy, or of their safety. In fact, it flies in the face of all three.

As stated by Professor of Neuroscience, Elliot Valenstein:² *"What physicians and the public are reading about mental illness is by no means a neutral reflection of all the information that is available."*

Reasoning Backwards: What Are We Treating?

If you were to ask the average person on the street what the biology of depression relates to, they would very likely parrot, "serotonin deficiency." This hypothesis, referred to as the monoamine hypothesis, grew out of observations of mood-related side effects in the treatment of tuberculosis patients with iproniazid,³ which has some inhibitory impact on the breakdown of monoamines.

From this accidental observation and double talk about reserpine's role in inducing and treating depressive states, a theory was born. Six decades of subsequent studies in never-medicated depressed patients have been conflicting, confusing, and inconclusive, and a critical review of the hypothesis concludes:⁴

"... there is no direct evidence of serotonin or norepinephrine deficiency despite thousands of studies that have attempted to validate this notion."

Similarly conclusive is a *New England Journal of Medicine* review on Major Depression,⁵ which stated:

"... numerous studies of norepinephrine and serotonin metabolites in plasma, urine, and cerebrospinal fluid as well as postmortem studies of the brains of patients with depression, have yet to identify the purported deficiency reliably."

Story at-a-glance

Contrary to popular belief, depression is not typically the result of 'low serotonin' levels, nor is it an imbalance that needs to be fixed with antidepressant drugs

Eleven billion dollars are spent each year on antidepressant medications, pharmaceutical companies have 625 lobbyists, and they underwrite more than 70% of FDA trials

There are no studies that show a better outcome in those prescribed antidepressants long term, while side effects are well documented; long-term antidepressant treatment even compromises the known benefits of exercise

Prior to the widespread use of antidepressants, the National Institute of Mental Health told the public that people regularly recovered from a depressive episode, and often never experienced a second episode

We need to identify vulnerabilities, modifiable exposures, and support basic cellular function, detox, and immune response to effectively treat depression

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Even in the pursuit of this appealingly reductionist idea of a chemical deficiency, we are unable to measure central nervous system quantities, to account for the inner workings of 14 different types of serotonin receptors,

Also for the vast projections of serotonin trafficking neurons, and for the delicate interplay between the 100 some neurotransmitters that we know to be active in the brain. Dr. Daniel Carlat, author of *Unhinged*, writes:

"We have convinced ourselves that we have developed cures for mental illnesses...when in fact we know so little about the underlying neurobiology of their causes that our treatments are often a series of trials and errors."

How Do These Meds Work?

Even if we were to accept the premise that these medications are helpful, extrapolating a medical etiology from this observation would be the same as saying that shyness is a deficiency of alcohol, or migraine a deficiency of codeine.

And to my holistic and integrative colleagues who are very excited about tryptophan and 5HTP in medication-naïve patients, I will remind them that the only time that tryptophan depletion has correlated with low mood is in those patients previously treated with SSRIs.

We have been taught to associate serotonin with feeling good, but the fact is that high serotonin has been associated with feeling bad, including carcinoid syndrome, Alzheimer's, autism, and schizophrenia.

Low serotonin metabolite (5H1AA) is indicative of turnover of serotonin, and is the eventual result of increased serotonin in the synapse. This has been associated with suicide, violent crime, alcoholism, bulimia, and exhibitionism! Clearly, we are not dealing with a simple more is better, or even a "looking for the right balance" type of scenario.

Chasing this pattern and seeking to alter "levels" is like trying to connect a pile of scattered dots into a long straight line – you have to ignore the ones that don't fit. *What about genetics? Wasn't I born with this defect?*

Despite the continued efforts to identify "the gene," a false start in 2003,⁸ which suggested that those with a variant in the serotonin transporter were 3x more likely to be depressed, was later mowed over by a meta-analysis of 14,000 patients that denied this association.⁷ Dr. Insel, head of the NIMH, had this to say:

"Despite high expectations, neither genomics nor imaging has yet impacted the diagnosis or treatment of the 45 million Americans with serious or moderate mental illness each year."

Carlat goes on to say: *"And where there is a scientific vacuum, drug companies are happy to insert a marketing message and call it science. As a result, psychiatry has become a proving ground for outrageous manipulations of science in the service of profit."*

Pharma Weaves an Irresistible Tale

Eleven billion dollars are spent each year on antidepressant medications,⁸ pharmaceutical companies have 625 lobbyists,⁹ and they underwrite more than 70% of FDA trials. They court physicians,¹⁰ give them samples, tell patients to "ask their doctor," pay consultants to speak at scientific meetings, advertise in medical journals, fund medical education, and ghostwrite, cherry pick and redundantly submit data for publication. Psychiatric studies funded by pharma are 4x more likely to be published if they are positive,¹¹ and only 18% of psychiatrists are disclosing their conflicts of interests when they publish data. Their studies allow:

- *Placebo washout* (getting rid of those who are likely to respond to placebo before the study to strengthen the perceived benefit)
- *Replacement of non-responders*
- *Breaking blind* by using inert placebos so that subjects know that they have received the treatment
- *Use of sedative medications* concurrent to study medications

A now famous 2008 study in the *New England Journal of Medicine*¹² by Turner et al sought to expose the extent of data manipulation. Through valiant efforts to uncover unpublished data, they determined that from 1987 to 2004, 12 antidepressants were approved based on 74 studies. 38 were positive, and 37 of these were published. Thirty-six were negative (showing no benefit), and 3 of these were published as such while 11 were published with a positive spin (always read the data not the author's conclusion!), and 22 were unpublished.

Since two studies are required by the FDA for approval, you can see how these companies are tossing the coin repeatedly, until heads comes up, and hoping no one is looking when it's tails. Per Robert Whitaker, author of *Anatomy of An Epidemic* and *Mad In America*, references, these practices undermine the accuracy of data and deliver information that corrupts physician's delivery of care and endangers patients.

The costs of this manipulation of information is the loss of true **informed consent** – physicians cannot adequately share with

patients the risks and benefits if the benefits are fabricated and the risks are not uncovered (by 5-6 week trials) or are unacknowledged.

Placebo Effect – Why They "Work"

Despite Pharma's efforts, the truth about these brain bombs is emerging. In 1998, Dr. Irving Kirsch, an expert on the placebo effect, published a meta-analysis¹³ of 3,000 patients who were treated with antidepressants, psychotherapy, placebo, or no treatment and found that 27% of the therapeutic response was attributable to the drug's action.

This was followed up by a 2008 review,¹⁴ which invoked the Freedom of Information Act to obtain access to unpublished studies, finding that, when these were included, antidepressants outperformed placebo in only 20 of 46 trials (less than half!), and that the overall difference between drugs and placebos was 1.7 points on the 52 point Hamilton Scale. This small increment is clinically insignificant, and likely accounted for my medication side effects strategically employed (sedation or activation).

He found that severely depressed patients were less placebo responsive, generally, potentially accounting for the impression of some increased benefit, such as that found by Fournier et al.¹⁵ When active placebos were used, the Cochrane database¹⁶ found that differences between drugs and placebos disappeared, given credence to the assertion that inert placebos inflate perceived drug effects.

In response to 2005 recommendations from the National Institute for Health and Clinical Excellence that SSRI medications be first line treatment recommendations for depression, Drs. Kirsch and Moncrieff pointed out¹⁷ that the NICE data, itself, demonstrates a 1 point difference on the 52 point Hamilton Scale between placebo and drug groups, and that it was not in more severely depressed patients that this was found.

The finding of tremendous placebo effect was also echoed in two different meta-analysis by Khan et al¹⁸ who found a 10% difference between placebo and antidepressant efficacy, and comparable suicide rates. The largest, non-industry funded study,¹⁹ costing the public \$35 million dollars, followed 4000 patients treated with Celexa (not blinded, so they knew what they were getting), and half of them improved at 8 weeks. Those that didn't were switched to Wellbutrin, Effexor, or Zoloft OR "augmented" with Buspar or Wellbutrin.

Guess what? It didn't matter what was done, because they remitted at the same unimpressive rate of 18-30% regardless. Only 3% of patients were in remission at 12 months.

So what if it's placebo effect? It's working at least some of the time, so who cares? Here's why I, and other concerned psychiatrists and practitioners, care: I first became aware of the habit forming nature of these medications when I tapered a patient off of Zoloft in anticipation of a pregnancy in the coming year, and she experienced about 6 months of protracted withdrawal that began at about two months after the last dose. This was nothing I was prepared, by my training, to deal with.

What are these medications actually doing?! The truth is, we have very little idea. We like to cling to simple explanations, but even the name of the various antidepressants, *selective serotonin reuptake inhibitors* and *norepinephrine reuptake inhibitors* is misleading.

They are far from selective. An important analysis²⁰ by the former director of the NIMH makes claimed that antidepressants "create perturbations in neurotransmitter functions" causing the body to compensate through a series of compensatory adaptations which occur after "chronic administration" leading to brains that function, after a few weeks, in a way that is "qualitatively as well as quantitatively different from the normal state."

Changes in beta-adrenergic receptor density, serotonin autoreceptor sensitivity, and serotonin turnover all struggle to compensate for the assault of the medication.

Andrews et al²¹ calls this "oppositional tolerance," and demonstrate through a careful meta-analysis of 46 studies demonstrating that patient's risk of relapse is directly proportionate to how "perturbing" the medication is, and is always higher than placebo (44.6% vs 24.7%). They challenge the notion that findings of decreased relapse on continued medication represent anything other than drug-induced response to discontinuation of a substance to which the body has developed tolerance. They go a step further to suggest:

"For instance, in naturalistic studies, unmedicated patients have much shorter episodes, and better long-term prospects, than medicated patients (Coryell et al., 1995; Goldberg et al., 1998; Posternak et al., 2006). Several of these studies have found that the average duration of an untreated episode of major depression is 12–13 weeks (Coryell et al., 1995; Posternak et al., 2006).

Since acute ADM management of major depression minimally requires several weeks to reduce symptoms, the duration of untreated episodes is much shorter than the recommended duration of ADM therapy. This suggests that ADM therapy may delay resolution of depressive episodes."

Harvard researchers²² also concluded that at least fifty percent of drug-withdrawn patients relapsed within 14 months. In fact:

"Long-term antidepressant use may be depressogenic . . . it is possible that antidepressant agents modify the hardwiring of neuronal synapses (which) not only render antidepressants ineffective but also induce a resistant, refractory depressive state."²³

Buyer Beware

Here we come to the little disclosed poor outcomes associated with long-term treatment. We won't focus on the risk of suicide and violence, bleeds, or even suppressed libido and sexual dysfunction, indifference (or "medication spell-binding" as Dr. Peter Breggin calls it), or weight gain and dysglycemia. Let's just focus on what the data shows on how your ability to function, long-term, in the world with depression is significantly sabotaged by treating that first episode of depression with medication.

This was famously explored by Robert Whitaker, and can be summarized with the following studies, as a primer. Longitudinal studies demonstrate poor functional outcomes for those treated with 60% of patients still meeting diagnostic criteria at one year²⁴ (despite transient improvement within the first 3 months). When baseline severity is controlled for, two prospective studies support a worse outcome in those prescribed medication:

One in which the never-medicated group experienced a 62% improvement by six months, whereas the drug-treated patients experienced only a 33% reduction in symptoms,²⁵ and another WHO study of depressed patients in 15 cities which found that, at the end of one year, those who weren't exposed to psychotropic medications enjoyed much better "general health;" that their depressive symptoms were much milder;" and that they were less likely to still be "mentally ill."²⁶

I'm not done yet. In a retrospective 10-year study²⁷ in the Netherlands, 76% of those with unmedicated depression recovered without relapse relative to 50% of those treated. Unlike the mess of contradictory studies around short-term effects, there are no comparable studies that show a better outcome in those prescribed antidepressants long term.

Perhaps most concerning to a holistic physician is data²⁸ that suggests that long-term antidepressant treatment actually compromises the known and evident benefits²⁹ of exercise! Benefits of exercise treatment of depression were comparable to Zoloft and were diminished when combined with Zoloft where patients relapsed at higher rates than they did with exercise alone.

Selling Sickness

Whitaker helps us to remember: Prior to the widespread use of antidepressants, the National Institute of Mental Health told the public that people regularly recovered from a depressive episode, and often never experienced a second episode.³⁰ Now we have skyrocketing rates of disability in the setting of skyrocketing prescriptions. Whitaker has compiled and analyzed data demonstrating that days of work lost are increased by medication treatment as is long-term disability (19% vs 9%),³¹ 3-7 times the incidence of loss of "principal social role" and "incapacitation,"³² with treated illness, and that 85% of unmedicated patients recover in a year, with 67%³³ doing so by 6 months – an enviable statistic.

What has happened here? Since its 1952 inception and notorious inclusion of homosexuality as a diagnosable syndrome, the Diagnostic and Statistical Manual has now ballooned to more than 300 diagnoses in its fifth edition, all arrived at through general consensus of a committee consisting of practitioners with conflicts of interest³⁴ and pharmaceutical enmeshments. Allen Frances at Columbia states:

"Wholesale imperial medicalization of normality that will trivialize mental disorder and lead to a deluge of unneeded medication treatment – a bonanza for the pharmaceutical industry but at a huge cost to the new false positive patients caught in the excessively wide DSM-V net."

We need to break the populace out of its spell, reject the serotonin meme, and start looking at depression (and anxiety, and bipolar, and schizophrenia, and OCD, etc) for what they are – disparate expressions of a body struggling to adapt to a stressor. We need to identify vulnerabilities, modifiable exposures, and support basic cellular function, detox, and immune response. This is personalized medicine, where these abstract labels become meaningless because they only address the "what" of the symptoms" in an impressionistic, non-specific manner. One as helpful as saying the fever is the disease, and Tylenol the cure. Psychiatry's swan song has been sung...listen for its plaintive wail.

About the Author

Dr. Brogan is boarded in Psychiatry/Psychosomatic Medicine/Reproductive Psychiatry and Integrative Holistic Medicine, and practices Functional Medicine, a root-cause approach to illness as a manifestation of multiple-interrelated systems. After studying Cognitive Neuroscience at M.I.T., and receiving her M.D. from Cornell University, she completed her residency and fellowship at Bellevue/NYU. She is one of the only physicians with perinatal psychiatric training who takes a holistic evidence-based approach in the care of patients with a focus on environmental medicine and nutrition. She is also a mom of two, and an active supporter of women's birth experience, rights to birth empowerment, and limiting of unnecessary interventions which is a natural extension of her experience analyzing safety data and true informed consent around medical

practice. She is the Medical Director for Fearless Parent, and an advisory board member for GreenMedInfo.com and Pathways to Family Wellness. She practices in NYC and is on faculty at NYU/Bellevue.

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