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NOONDAY DEMONS AND MIDNIGHT SORROWS

BIOLOGY AND MEANING IN DISORDERED AND NORMAL SADNESS

Abstract: Andrew Solomon's *The Noonday Demon* and his article in this issue (on which we focus our commentary) present an eloquent, wide-ranging, and non-reductionist portrayal of depression as a disorder of both biology and meaning. Solomon illustrates the contributions that psychodynamic and pharmacological therapies alike can make to the treatment of depression, emphasizing the interplay between exploration of meaning and biological manipulation of the brain. We argue that the link between biology and meaning may help to explain the notion that depression yields distinctive truths; depression can yield truths or suggest meanings that otherwise are hidden from us by the normal biology of our meaning systems. We also identify several limitations of Solomon's discussion. First, in light of recent evidence, Solomon is occasionally overenthusiastic about the benefits of medication. Second, he overemphasizes the role of depression in creating negative social circumstances when most evidence indicates that the condition is more a consequence than a cause of social environments. Finally, in light of our recent exploration of how psychiatry has confused intense normal sadness with depressive disorder (Horwitz and Wakefield, 2007), we examine Solomon's views on the distinction between sadness and depressive disorder. We argue that, although Solomon recognizes the importance of distinguishing normal sadness from depressive disorder, and even astutely characterizes the difference as anchored in evolutionary psychology, at times he mischaracterizes likely cases of normal sadness as depressive disorder. Overall, however, Solomon provides an exceptionally well-drawn portrayal of the nature, causes, and treatments of depression.

Key Words: depression, sadness, antidepressant medication, harmful dysfunction, psychiatric diagnosis, evolutionary psychology

ANDREW SOLOMON has performed an enormous and distinctive service to those who suffer from depressive disorder, as well as to the mental health professionals that attempt to help them. The magnitude of his achievement is manifested in the prominence his book, *The Noonday*

Demon: An Atlas of Depression (2001), has attained within the nation's discussion of depression. It lies in the power of his prose to portray depression in a vivid and compelling way, his consequent ability to bring public attention to this disorder, and the fact that his own success with treatment of severe depression serves to instill hope in readers who suffer from this disorder.

But there are other talented writers who, before and since, have contributed similarly by telling their personal stories. In our view, the particular distinctiveness and timeliness of Solomon's contribution lies in what he himself emphasizes in his book and in his article in this issue (we focus our comments on the latter), namely, his integration of disparate approaches to depression. He says what needs saying but what professionals often hesitate to say, and he says it with the air of obviousness that it deserves. First, depressive disorder is generally a disorder of both biology and meaning simultaneously rather than one or the other: "The intense fashion for biological explanations of depression seems to miss the fact that chemistry is a different vocabulary for a set of phenomena that can also be described psychodynamically. Neither our pharmacology nor our analytic insight are at the time advanced enough to do all the work." (Solomon interestingly observes that the divergence between the physician Hippocrates's and the philosopher Plato's approaches to melancholia was a forerunner of the kind of divergence we see today between biological and meaning-related accounts.) Of course, it is equally true that there is a biology underlying normal reactions of sadness, and it is the malfunctioning of these biological underpinnings of normal sadness that is one source of depressive disorders.

Second, for many cases an approach that addresses both the biological and the meaning levels is likely to be preferred to treatment that dogmatically pursues one approach. Solomon writes:

I am a staunch believer in approaching the problem with multiple strategies. For me, the medication was an absolutely necessary step to assuage the destructive emptiness of my terrible depressive episodes. Once I had begun to return to some reasonable facsimile of myself, though, there was the need for a different kind of work. I had to figure out what triggered my episodes and how to control them. This I did with my analytically trained therapist, with whom I had begun working after ending my treatment with the analyst who had failed to help me in my initial crisis. Our work drew mostly on psychoanalytic thought, even though it incorporated some aspects of cognitive-behavioral models.

Solomon's clear-headed and intellectually open-minded approach adds to a growing discussion of the need for integration of psychotherapeutic/psychodynamic and pharmacological approaches to mental disorders that challenges the standard fragmented and dogmatically one-sided approaches. The mental health professions—to a shameful degree and to a degree potentially harmful to patients' welfare—tend to talk past each other. Rather than drawing a distinction among kinds of cases of depression, debates occur about which type is the real, universal phenomenon. Each view or theory or school of thought tends to inflate its portion of the truth about depression into the whole truth. Thus, for some, depressive disorder is always a biological defect calling for medication; for others it is always a deep-rooted psychodynamic affliction; for others it is a matter of distorted cognition; and for still others it is a matter of deficiency of positive reinforcement due to lack of activation of behavior, and never a real disorder at all.

Even a perfunctory review of the many kinds of cases of depressive disorder reveals that such expansive views are implausible on their face. Depressive disorder is an outcome of dysfunctions that can occur at several levels of brain and mental functioning singly or simultaneously; it is likely one of the most multietiological categories of disorder in the DSM. In some instances, onset of profound and enduring depression is bewilderingly unrelated to any environmental event; in other cases, possibly for psychodynamic, cognitive, or biological reasons, people react to a real loss and then get "stuck" in that response in a way that is no longer linked to the realities of the situation or proportional to the loss, nor self-healing over time. Universalistic claims about depression serve the self-esteem and ideological needs of the clinician and have no legitimate place in a scientifically based service profession.

Depressive disorder is often a disorder of biology, but it is a disorder of the biology underlying meaning formation and transformation. Thus, biological and psychodynamic or cognitive approaches can access the multitiered dysfunction. Solomon emphasizes how the interplay between meaning exploration and biological manipulation can be helpful. Compared with the Babel of overgeneralized depression theories, the reasonableness of Solomon's integrative view is refreshing and illuminating. It is all the more compelling because it is from a patient's perspective and because Solomon describes, first, how overly dogmatic treatment was initially harmful; second, how each component of integrative treatment had its benefit; and, third, how the combination of treatments interacted

synergistically to create an overall better outcome. These are truths that embarrassingly continue to elude many mental health theoreticians, researchers, and clinicians. Solomon also understands a central point that is often obscured by professionals' myth of effecting a "cure": by and large, whatever approach one takes, mental problems are unlikely to be cured in the strict sense. Rather, people learn to distance themselves from their problematic feelings and urges, which are thus rendered incapable of exerting the power that they had to possess those individuals. As Solomon puts it, "That ability to tolerate your own depression is what allows you, I believe, to achieve some resilience."

Certain strands from Peter Kramer (2005) offer a useful counterpoint to elements of Solomon's article. Kramer presents a case history of a patient with whom he explored meanings and to whom he also provided medication. Kramer reports that, when the patient remitted, she accused him of having unjustifiably given credence to her expressed feelings and thoughts during her depression, as if they expressed her meanings; after all, she argued, that was not she, that was the biological disorder talking. Although a practitioner of integrative treatment himself and one who views both medication and psychotherapy as having an important role in treatment of depressive disorder, Kramer focuses insistently on the biological roots and wonders if, indeed, he erred in taking his patient's depressed self's meanings so seriously. He seems inclined to assume, as he argued in an earlier book (Kramer, 1993), that the depressed self is not the real, essential self and that medication makes oneself into one's real self—the essence of oneself having been obscured by the disorder.

In contrast, Solomon expresses appropriate puzzlement about this entire area:

Once you have been depressed, and particularly once you have allowed medication to reshape your mental states, you need to understand who you are at the most fundamental level. You need to sort out the chemical facts of depression from the experiential; you need to gain insight into the patterns that depressive tendencies doubtless forged in your earlier life. You need to examine the relationship between love and depression in your own experience. You need to make sense of the idea that you are on medication and determine whether the medication has made you more truly yourself or has shifted you into being someone else.

Which self is the real self? Solomon, assuming that in principle there is a real self (i.e., someone "who you are at the most fundamental level"),

leaves open the question of whether the medicated self is more or less real. We see patients worried about this question with regard to themselves, their spouses, or other loved ones who have changed owing to medication. Obviously, integrity of identity—aside from relief from suffering—is of great interest and importance to people. However, although personalities may be conceived as having essences consisting of some set of ultimate, biologically driven, stable, enduring dispositions, it has been apparent from at least the time of the “self-actualization” movement that the notion of which of the various potentialities is the “real” self is a murky concept to say the least. It certainly is one about which we ought to reserve judgment and should approach with a heavy dose of skepticism. Talk of the “real,” or “essential,” self seems to appeal to something objectively privileged and yet may simply be a camouflaged preference or value judgment heavily shaped by social desirability.

Another important topic on which Solomon diverges from Kramer's (2005) doggedly biological approach to the nature of depression is the potential benefit from a depressive episode. Solomon says, “It is not that depressions are wonderful and everyone should have one, but if you've gone through this experience, there is a great deal of insight that you can get from it.” This statement is in contrast with two points Kramer pursues with great polemical vigor. One is his rejection of the notion that people might learn something from their depression, over and above the sorts of lessons everyone learns from struggling with adversity and from intense suffering of the kind common in a variety of physical disorders, mental disorders, and horrific life events. That is, Kramer rejects the notion that depression has anything *distinctive* to teach us, other than strategies of how to get over it and a general virtue of courage in facing adversity. (Even Solomon says of his selection of informants, “I did not choose them as people whose illness was more or less severe but, rather, as people who I thought were in some way courageous in handling their own illness.” He thus suggests a generic criterion that would apply to many conditions.) Second, Kramer notes the long tradition going back to Aristotle that holds that depression has some distinctive role in creative endeavors. Kramer insists that no such creative benefit is likely, despite his admission that there may be such a relationship between bipolar disease and creativity (he argues that any such benefit lies entirely in the mania) and that there has been little study of the relationship between unipolar depression and creativity.

Here we think that Kramer's position lacks *prima facie* persuasiveness and that Solomon does better. Surely Kramer is right that depressive disorder

should not be glamorized, and all commentators agree that it is an undesirable condition, whatever mitigating occasional uses it might have. Nonetheless, Kramer seems to take too simplistic a view here of the link between biology and meaning in depression. He seems to suggest that because the meanings generated are the product of disorder, they must be false; and, if they are false, there is nothing distinctive and illuminating to learn from their content that represents anything useful. The flaw in Kramer's argument is simply that, even if depressive disorder is indeed a biological disorder of brain functioning, it is a biological disorder concerning how the brain determines meaning. Thus, by definition, the disorder provides access to meaning states that the subject would otherwise likely not experience—and meanings that could represent or indirectly suggest truths.

Solomon, in contrast to Kramer, assumes that depression does teach something and can have value in life in general and in the creative life. Without data, we, like Kramer and Solomon, are reduced to speculation. But, as a speculative judgment, it seems to us quite possible that something unique is learned from depression and that depression might have a special relationship to creativity as well. The first should be obvious; in other cases where biological agents change the meaning-formation functions of the brain (e.g., LSD, marijuana, alcohol), we do seem capable of learning new things from the altered meanings that we generate, even though the meanings are not part of our natural functioning. The very possibility of certain induced experiences changes our perspective on our normal experience. Moreover, we can expect that a depressive illness's assault on people's meaning system would likely be in some respects an extension of the feelings they had when they suffered extreme real losses and experienced normal sadness. Thus, exploration of their experiences, both normal and under disorder, might each shed light on how the other manifests itself. As Solomon suggests, "You need to know what grief is all about, where it is lodged in you, and how it overlaps with depression as an illness."

Consider one possible example of a lesson depression might teach. The French phenomenologist Maurice Merleau-Ponty (1945) suggests that, when we grieve the loss of a love, the entire world seems reduced in or devoid of meaning; the loss of the lover has drained from us an entire way of seeing the world that shapes our experiences and is in the background even when we are alone, and the lover's loss thus yields a sense that every experience is missing its full meaning. Now, this is a normal experience. But, if one effect of depression is, under conditions in which no extreme loss has been sustained, to generate such meaning-loss in our experience

nonetheless, then surely we must learn something that may well inform our experience and sense of life forevermore: the degree to which meaningfulness does flow from our projection into reality of certain kinds of fantasies or meaning filters. Analogously, people commonly report that after the loss of a love it is difficult to fall in love again for awhile because, for one reason or another, they cannot project onto another person the idealized imagery that makes love possible; this experience can teach one how much love depends on one's ability to project such fantasies onto the other. Similarly, depressive episodes—even if induced by a biological malfunction of sadness-generating brain mechanisms—may empty the world of our usual projections and thus startle us with the degree to which the felt meaningfulness of our lives is dependent on what we ourselves can bring to the table in desire and fantasy. This is no small lesson to learn, and it is not inherent in the experience of physical disorders, or even most other mental disorders, but rather appears to be unique to depression. LSD perhaps provides a broadly analogous insight about our perceptual construction of reality.

As to the longtime belief in a link between depression and creativity, there are many ways that the two might be related. Perhaps the alterations of the meaning system we have alluded to yield a fluidity of creative association, or perhaps sufferers fight the intensity of despair with correspondingly intense attempts at creativity by which they hope to transcend themselves and create the meaning that is lost. But the most plausible link lies in the cycle of creative work and its vicissitudes. Blocked progress or lack of inspiration is frustrating, even frightening, and causes hopelessness and despair; self-esteem rides on the outcome of creative work that is beyond one's ability to control. All too often people feel that the result of their efforts is a humiliating lack of excellence and, as research amply demonstrates, humiliation is the surest way to despair and depression (Brown, Harris, and Hepworth, 1995). The cycle of creative work is thus potentially intimately related to periodic feelings of depression. Moreover, the person driven to create may be the sort of person who is also an intense responder to life's vicissitudes, a risk factor for depression, as Kendler and Prescott (2007) and other behavioral geneticists have shown. While neither Solomon's embrace of the traditional view nor Kramer's facile dismissal of it is based on solid evidence, the preponderance of plausible speculation must at least allow for the possibility of the linkages that Solomon suggests.

One feature of Solomon's description of his symptoms raises a historical and nosological question. He emphasizes the intense anxiety he felt as

part of his depression; indeed, he says that he could have endured the other depressive symptoms knowing they would eventually remit but he could not have endured persistent anxiety because it is so painful (“Then the anxiety set in . . . if someone said to me I had to have acute anxiety for the next month, I would kill myself, because every second of it is so intolerably awful”). In fact, from the ancient writings on melancholia to those of the early part of the 20th century, anxiety, fear, and nervousness were listed as standard and common symptoms of depressive disorder, along with sadness, insomnia, loss of appetite, and the rest. Solomon quotes the very first definition of depression from Hippocrates, which already reveals this historical usage: “The symptoms were sadness, anxiety, moral dejection, tendency to suicide, aversion to food, despondency, sleeplessness, irritability and restlessness, accompanied by prolonged fear.” Then, in an attempt to define a purer syndrome, the DSM-III (American Psychiatric Association, 1980) excluded anxiety from the possibly symptomatic indicators of depression. Recent epidemiologic studies, however, unsurprisingly have “discovered” that there is a high comorbidity of anxiety disorders—and anxiety generally—with depressive disorders. Thus we are left with a supposed scientific puzzle about why anxiety and depression should occur so frequently together (Kessler et al., 2005). A syndrome of anxious depression is now contemplated as a category in future DSMs, and the study of the relation of anxiety to depression has become a major industry in the mental health research community.

In light of Solomon’s matter-of-fact and wholly organic portrayal of the integral place of anxiety in his depressive syndrome, one can only wonder by what logic anxiety was ever excluded from depressive disorder’s symptoms. The common alternation of despair and desperation, of anxious fear of an expected future and hopeless submission to that future, seems to be built into our emotional mechanisms. Losses prototypically trigger sadness; and loss is both about the past (what is lost) and the future (which must be newly faced without the lost object); and anxiety, classically, is an expectancy of future danger. Loss and anxiety thus seem naturally to go together because of the meanings at their core and the reality of the links between events that trigger them. The paradox of anxious arousal in a syndrome of depression is more apparent than real, like the seemingly paradoxical symptom an early physician pointed to when he noted that some depressive patients are so afraid of death that they want to kill themselves. (Regarding the latter, note that this fear is a very different motive for suicide than the emptiness Solomon and one of his

informants describe: “It is that feeling of deadness that tempts people to achieve an actual deadness.”) The contemporary use of “comorbidity” to deal with these complex relations between anxiety and depression is misleading and needs substantial reworking in the DSM-V.

Solomon’s encouraging comments on the effectiveness of medication (and of treatment more generally as well) warrant a note of caution. Recent studies suggest that antidepressant medication does not work for as many people as was once thought (Moncrieff and Kirsch, 2005; Kirsch et al., 2008). Indeed, some studies warn that, aside from the most severe depressions, whether or not these medications have more than a placebo-level effect at all is unproven. While it seems that the latter fear is unlikely to be realized, and while Solomon is well aware of the limits of medication, he shares with Kramer the inevitable attitudinal bias of one who has either experienced a cure or watched it occur in patients. It remains possible that, despite all the hype, these medications are just not as good as either Kramer or Solomon thinks they are. The troubling fact is that controlled studies reveal an amazingly robust placebo effect when medication for depression is administered, an effect substantially larger than the placebo effect in other psychiatric disorders (Kirsch et al., 2008). Caution is warranted here lest false hopes be raised that we have for depression an equivalent of aspirin for a fever.

Solomon repeats and comments on an informant’s view—resonating with the “depressive realism” literature that Solomon cites—that the thoughts one has during a depression are in some sense true or reflect reality. The informant says:

You don’t think in depression that you’ve put on a gray veil and are seeing the world through the haze of a bad mood. You think that the veil has been taken away, the veil of happiness, and that now you’re seeing truly. You try to pin the truth down and take it apart. And you think that truth is a fixed thing. But the truth is alive, and it runs around. You can exorcise the demons of schizophrenics who perceive that there’s something foreign inside them. But it’s much harder with depressed people, because we believe we are seeing the truth. But the truth lies.

Solomon is much taken with the informant’s apparently paradoxical but fascinating notion that “the truth lies.” It does not seem to mean what common sense might dictate, namely, that even a statement of the truth can, by emphasizing one part of the truth over another in a given context, mislead one. The truths that emerge in a depression (assuming that truths

they are) do not seem misleading so much as fundamentally malformed despite their truth. The question is, What might this mean if translated into a more explicit and coherent assertion?

Here is Solomon's comment:

That formulation of a truth that lies is extremely powerful. When people are depressed, they tend to have a whole string of perceptions. Some of the perceptions are very easy to contradict, because they're actually inaccurate. Someone who is depressed will say, "No one loves me. There's just no point because no one loves me." And it's very easy to say to a person like that, "I love you. Your mother loves you. Your children love you. Your friends love you." You can come back with all those answers, at least for people who are, in fact, loved by somebody. It can also be the case that people who are depressed have accurate perceptions with exaggerated affect associated with them. So, people will say, "You know, whatever we do in this life, in the end, we're all just going to die anyway." Or they say, "There can never be any real union between two people. We're all trapped alone in our own single body." And you have to be able to say to them, "That's true. But let's focus on breakfast for right now." It's terribly difficult to handle that shift in affect. . . . What is extraordinary is that most of us are able to go right on with life without being distracted and disabled by those things. It is an evolutionary and a social advantage to have some degree of protective optimism that allows one to ignore the darkness of human experience. . . . If you have too optimistic a view, you obviously take idiotic risks, and it's very destructive. Mild optimism, however, is the state on which lives and societies are most successfully built. . . . [D]epressed people . . . will frequently say, "What I have here is not illness, it's insight." One has to be able to articulate that it is insight, that insight is illness, and the illness needs to be treated, assuming that the person is having trouble tolerating it.

There are two central insights lurking in Solomon's astute passage. The first concerns the varieties of malfunctions that can underlie depressive disorder. As one of us (Wakefield, 1998) has argued, there are in principle two kinds of depressive disorders, that is, two kinds of ways the mechanisms responsible for generating sadness responses can malfunction. These mechanisms appear to work by, first, monitoring cognitions concerned with loss and estimating the magnitude of the loss, and, second, generating an affect of sadness in rough proportion to the magnitude of a perceived loss. Each of these two steps can be disordered. First, then, there are disorders in which cognition has malfunctioned such that the perception of loss is no longer reasonably accurate (e.g., one sees every

slight loss as indicative of a major assault on self-esteem). Because the cognitions do not reflect the reality, one experiences the kinds of cognitions that normally lead to sadness (e. g., perception of major loss) even though no such loss has taken place. (Such false beliefs can be disputed, as in Solomon's example of the person who says, incorrectly, that no one loves her, but a true malfunction of belief is likely to be more difficult to change than a run-of-the-mill error.) Second, even if one's cognitions and perceptions of loss are accurate, such perceptions may trigger intensely exaggerated affective responses that are not remotely proportional to the magnitude of the perceived loss, or one may be unable to contain and control an affect.

But the most important point to be made about that passage is that Solomon's passing reference to evolutionary theory is exactly the right way to resolve the paradox of the truth that lies. The truth cannot literally lie, but it can represent something having gone wrong with the way the person's belief system is working nonetheless and thus lie to us to the extent that it is a truth that it is abnormal to believe and that, if we are normal, we ought not to believe. The trick here is that our biologically designed nature does not always dictate that we should believe the literal truth. We are, for example, likely to be more optimistic than circumstances warrant and are likely to think more highly of our spouse and our children than could possibly be objectively supported (Taylor and Brown, 1988). Biologically designed biases and illusions allow us to live with our mortality and to love others and to that extent are essential to our living a good life. It may well be that depressive disorder, among other things, disengages us from such positive distortions of reality and subjects us to something closer to the truth in a way we were never biologically designed to experience. Normality and the truth thus diverge, and the truth "lies" to the extent that it subverts beliefs essential to normality.

Despite his generally balanced tone and cautiousness, we note that Solomon on occasion tends to fall into the sort of exaggeration that takes an already admittedly horrible condition and tries to inflate its horror for literary effect. For example, the statement about depression, "For almost everyone who has it, it is a recurrent, lifelong condition" is just plain wrong. Of those who satisfy DSM criteria for major depressive disorder at some point in their lives, about 25% report they have had only one episode in their lifetime (Marcotte, Wilcox-Gox, and Redman, 1999). It may, however, be that many of these persons do not really have depressive disorders and are experiencing intense normal sadness (Wakefield et al. 2007).

A second place where Solomon's discussion is incorrect is his lengthy lament that we do not have any program aimed at identifying people in the general population who have depressive disorder: "It struck me as something of a humanitarian crime that we don't have outreach programs to identify these people and help them." In fact, prodded by findings in epidemiologic studies of large numbers of untreated persons in the community who appear to satisfy diagnostic criteria for depressive disorder, there has for some time been a major movement to screen for depression in the community (Horwitz and Wakefield 2007, ch. 7). Recently it has become not uncommon for depression-screening instruments to be administered in doctors' offices and schools, and they are readily available on the internet (e. g. depression.com).

Solomon also errs in his discussion of depression among the impoverished. First, he supposes that, when poor people are depressed, the depression is likely the cause of the poverty owing to its immobilizing effects: "But the reality, as I found when I began doing this research work, is not that people are in general depressed because they are so poor, but, rather, they are poor because they are so depressed. People who are depressed cannot hold down jobs, and they cannot sustain relationships, and their lives become terrible and bleak. In the popular perception, the cause and effect have been reversed." Solomon notes that he is reversing the commonsensical view of the direction of causality. However, the research suggests that it is Solomon who gets the direction of causality backward and that common sense is right, at least in the majority of cases (Johnson et al., 1999; Ritsher et al., 2001; Lorant et al., 2003). That is, it appears that the dire circumstances of the poor do cause them to be miserable. (Nor, we note in passing, is it entirely obvious, as Solomon presumes, that the genetic load for depression is evenly distributed among segments of the population: "Depression is caused by the intersection of a genetic vulnerability, which is presumably evenly distributed across the population and triggers external circumstances," but this is a complex matter with a long history of dispute that we will not pursue here (Dohrenwend et al., 1992). Moreover, Solomon suggests that the poor do not seek treatment because their dire circumstances mislead them into thinking that their reactions are normal responses to their situations rather than disorders:

If you have a life that is grim and brutal and terrible in every way, and you feel really horrible all the time, it does not occur to you that you might have an illness, because the way that you feel seems to be commensurate with

the way that your life is. And so impoverished people who are experiencing acute depression by and large do not seek treatment, because it does not occur to them that there is anything aberrant about what they're feeling.

One problem with that statement is that such reactions may be normal responses to people's situations, and their accurate perception of this fact may be why they do not seek psychiatric treatment. Another is that in recent years rates of psychiatric help-seeking among the poor have increased so that they no longer underutilize mental health treatment for depression (Olfson et al., 2002).

These confusions lead to the major weakness in Solomon's discussion of poverty and depression, namely, a potential confusion between normal misery and depressive disorder. No better example could be given of this problem and of the controversial nature of Solomon's judgments here than his own extended example of an abused woman who receives treatment and recovers from her depression:

I remember one woman who came from the inner city just outside Washington, DC. She had gone to a family planning clinic because she was in an abusive relationship with a very cruel man. She had seven children, and she was terrified of having more. While she was at the clinic, she was picked up by an academic who was doing the screening for a pilot study. This is how she described her situation: "Yes, I had a job, but I had to quit because I just couldn't do it. I didn't want to get out of bed, and I felt like there was no reason to do anything. I'm already small, and I was losing more and more weight. I wouldn't get up to eat or anything. I just didn't care. Sometimes I would sit and just cry, cry, cry. Over nothing. Just cry. I just wanted to be by myself. My mom helped with the kids. I had nothing to say to my own children. After they left the house, I would get in bed with the door locked. I fear when they come home, three o'clock, and it just comes so fast. My husband tells me I'm stupid, I'm dumb, I'm ugly. I'm tired. I'm just so tired. I've been taking a lot of pills, mostly painkillers. It could be Tylenol or anything for pain. A lot of it though. Anything I could get to put me to sleep."

She was included in a protocol that provided six months of treatment. She had group therapy and was prescribed Zoloft. At the end of the six months she had left the abusive man and had gotten a job working in child care for the U.S. Navy. She was set up in a new apartment. She was physically unrecognizable.

That is a moving example, but Solomon fails to acknowledge that its conceptual nature as a case of depressive disorder is ambiguous, to say

the least. The woman was impoverished, unemployed, caring for seven dysfunctional children in a dysfunctional family, constantly humiliated (“My husband tells me I’m stupid, I’m dumb, I’m ugly”), and terrified of being abused by her violent partner. Do we really need to add the construct of depressive disorder (i.e., some *malfunction* of her brain or mental mechanisms concerned with sadness responses) to explain why this woman felt hopeless and did not want to get out of bed? Her response seems consistent with normal human responses to horrific and demoralizing circumstances for which no way out is apparent.

A telling feature of the vignette, partly emerging in Solomon’s description subsequent to the foregoing passage, is that not only did the woman get treatment but the situational triggers for her feelings all changed in a relatively short time. She left her abusive partner, and she got a job. Her seven children were not only transformed with respect to their negative features that had previously caused her despair (“I talk to them about drugs . . . And they keep clean now. They don’t cry like they used to. And they don’t fight like they did”) but they became a source of pride and hope for the future (“My kids are so much happier. They want to do things all the time now. We talk hours every day, and they are my best friends. As soon as I come in the door, I put my jacket down, purse, and we just get out books and read. Doing homework all together and everything. We joke around. We all talk about careers. And before they didn’t even think careers. My eldest wants to go to the Air Force. One wants to be a firefighter. One a preacher. And one of the girls is going to be a lawyer”). Moreover, she can now live without terror (“I’m not afraid anymore. I can walk out the door not being afraid”) and thus can become more expressive of herself (“And things keep on changing. The way I dress. The way I look. The way I act. The way I feel”). Do we really need to add to what we know of normal human emotion the additional construct of “cure of a depressive disorder” to explain why, once these dramatic changes in her situation occurred, virtually immediately her depression fully disappeared? This appears on its face to be a case in which the depressive response was likely situation dependent and in which nothing necessarily had gone wrong with internal functioning. In other words, it is not at all clear from the vignette’s details that this woman in fact had a depressive disorder.

This brings us to an issue central to our own work, the distinction between depressive disorder and normal sadness (Horwitz and Wakefield, 2007). Solomon is concerned about this distinction because he believes that possible confusion might lead people to trivialize major depression.

We have emphasized the other side of the coin, that overly inclusive diagnostic criteria can yield incorrect pathologization of normal responses to negative life circumstances. Whichever danger one emphasizes, the same issue arises: how to make this distinction. Solomon makes several useful points in this regard:

[A] single word describes the spectrum of emotion that ranges from how a small child feels when it rains on his birthday to how people who have committed suicide in the most atrocious ways have felt. . . . [W]hen you say about someone, Oh, he has acute depression, people tend to think, Well, I get depressed, too, and I deal with it just fine. They don't understand the difference. And that is partly, I think, because there is an ongoing and significant question of the extent to which depression is part of the normal mood spectrum and is on a continuum with ordinary sadness or difficulty or despair, and the extent to which it is actually a separate clinical condition. . . . It serves a great evolutionary advantage for us to have a mood spectrum and to be capable of joy, of sorrow, and of anger and distress. It is when that mood spectrum moves beyond its useful extent and into some other arena that it becomes something else. . . . In clinical depression, you have something that is continuous with the normal emotional range but that nonetheless becomes categorically different when it gets to that extreme point.

There is in my view a very sharp distinction between depression and sadness. Sadness is highly important. . . . If you love someone, but your feeling is, Well, if she dies, I'll be fine, I'll meet someone else, it would not be love as we know it. . . . There is no way that you can have love without the potential for sadness, without that experience of sadness. On the other hand, there is a point at which you become so sad that you are utterly, totally paralyzed and useless to yourself and to the rest of the world and completely nonfunctional.

I am often asked to distinguish between grief and depression. Grief is explicitly reactive. Essentially, if something terrible happens and you experience grief, and a year later you are still feeling sad about it, but somewhat less sad, then what you are feeling probably is grief, which will eventually ameliorate. If something happens and you feel terrible about it, and six months later you feel worse and less able to function, and six months after that, you feel as though you cannot see beyond your own ego world, then that is depression. The trajectory, I think, is often the most important yardstick, more significant than the degree of affliction in any isolated moment.

Solomon recognizes the importance of normal sadness, and that sadness can be a crucial expression of what is most meaningful to us. He also

understands that at its core, the concept of normal sadness depends on the fact that humans are biologically designed to experience such feelings, that indeed we would be disordered if we were not capable of such feelings in response to deep loss. And he alludes to the ease with which normal and disordered sadness can be confused. His point that depressive disorder is “continuous with the normal emotional range” but “nonetheless becomes categorically different when it gets to that extreme point” seems to us to contain an important insight that is missed by the many researchers who argue that, because depressive feelings occur along a continuum, depressive disorder should be seen as occurring along a dimension of severity and the entire dimension should be considered disordered, as in subthreshold or minor depression (e.g., Judd, Akiskal, and Paulus, 1997; Kessler et al., 2003).

We do not believe that depressive phenomena are best conceived simply as a continuous dimension of severity, with mild, natural sadness lying on one end and severe depression on the other end of what should be considered a continuum of disorder. Instead, analogous to Freud’s (1926) distinction between realistic fears and anxiety disorders, there is a conceptually sharp (even if in application substantively often fuzzy) distinction between feelings of sadness when people have something to feel sad about and depressive disorders that either lack grounding in circumstances of loss or are disproportionate to the circumstances that initiated them or to the evolving situation. The symptoms of normal sadness can be extremely intense when they follow especially severe losses, such as the unexpected death of a child or the loss of a valued relationship, source of financial security, or profession. Conversely, depressive symptoms that are ungrounded in any context of loss can be mild. The “mild” versus “severe” distinction is not the same as the “normal” versus “disordered” distinction, for the latter has to do with something having gone wrong with the biologically designed functioning of emotion mechanisms (Wakefield, 1992a, 1992b, 1999), and normal and disordered responses alike can be mild or severe. Both normal sadness and depressive disorders are continuous in severity, but they do not fall on different ends of the same continuum.

There are three major differences that distinguish depressive disorders from natural sadness, all of which are hinted at to one degree or another in Solomon’s article. First, depressive disorders are not so clearly reactive to real loss in a proportional way as are normal reactions. Second, some particularly severe symptomatology, such as total, enduring immobilization

that goes beyond the normal grief response, are almost always indicators of disorder. For example, Solomon describes his initial episode as starting as follows: "From my bed I looked at the telephone on my night stand, but I could not reach out and dial a number. I lay there for four or five hours, just staring at the telephone." Finally, and above all, the trajectory of the normal response is that, assuming no additional losses and stressors, it will naturally tend to remit over time as the person adjusts, whereas the disordered condition may stay the same or even get worse as time passes.

Andrew Solomon has provided us with an eloquent, wide-ranging, and nonreductionist portrayal of depressive disorder. It is virtually unique in combining a searing depiction of the experience of depression with an enormous command of the historical, biological, and psychiatric literature on the subject. Moreover, his emphasis on the multifaceted nature of not only the causes but also the treatments for this condition is almost certainly correct.

Our book, *The Loss of Sadness*, does not contradict but complements Solomon's *The Noonday Demon*. Whereas Solomon's subject is depressive disorder, our focus lies on sadness that is naturally grounded in circumstances of loss but may be misconstrued as disorder. Such confusion is especially likely given that current DSM diagnostic criteria ignore the context of symptoms (except for certain instances of bereavement) and base diagnoses on symptoms alone, despite observations dating back to antiquity that as far as symptoms go, intense normal sadness and depressive disorder can look very much the same. Yet the relation to context is at the heart of the distinction between normal and disordered sadness.

Unlike depression, there is nothing wrong with becoming sad in the face of loss, as Solomon observes. Indeed, when people suffer extremely devastating losses, it is natural that they will develop correspondingly severe symptoms. Like any biologically shaped function, such normal sadness reactions can go wrong and people can become stuck in enduringly immobilizing responses that no longer represent a normal coping trajectory, yielding disorder. While depressive disorders represent a dysfunction of loss response mechanisms, sadness arises from the way that evolution designed people to respond to the losses that they suffer, and as long as it stays within those parameters it should not be misclassified as a disorder.

Yet the distinction between depressive disorder and normal sadness is not identical to the decision to treat or not treat a condition. Understanding the situation for what it is and thus understanding the likely positive prognosis, a patient might still decide to try to relieve painful feelings with

medication, and we see no reason for moralistic censure of such a decision. Rather, censure is appropriate when a professional ignores the context of a patient's feelings and meaning system and incorrectly classifies the patient's normal sadness as disorder, thus biasing the patient's decision toward taking medication rather than considering other options.

Thus conceived, the boundaries between normal sadness and depressive disorder are admittedly often elusive, so that in practice it is often difficult to distinguish the two in a range of borderline cases. However, as we have argued, current diagnostic criteria misclassify many clear cases of normal sadness as disorders (Horwitz and Wakefield, 2007; Wakefield et al., 2007). While it is difficult to draw any precise lines between natural and disordered conditions, important etiological, prognostic, and treatment issues turn on where we set this demarcation; clearly, getting the distinction right whenever possible is important. Solomon's justifiable quest to call attention to the immense suffering that depression entails and the ways in which treatment can help people to cope with this suffering sometimes appears to lead him into the trap of confusing likely instances of natural, contextually grounded sadness with depressive disorder.

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