The Loss of Grief: Science and Pseudoscience in the Debate over DSM-5's Elimination of the Bereavement Exclusion

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Abstract Was the DSM-5 revision process based on careful evaluation of scientific evidence, as the DSM-5 Task Force repeatedly claimed? The counterfeit of science is pseudoscience, the systematic motivated deviation from basic canons of rational scientific evaluation of evidence to create the false appearance of scientific support for a favored hypothesis. In this chapter, I consider the arguments that were used to support the DSM-5's controversial decision to eliminate the bereavement exclusion (BE) to major depressive disorder (MDD). I consider three central arguments: that the BE had to be eliminated for reasons of consistency; that the BE excluded cases from MDD that would respond to treatment; and that the BE leads to missing suicidal cases. The analysis reveals forms of rhetoric by which the question at issue was obfuscated or misconstrued, and the scientific evidence sidelined, rendered impotent, or outmaneuvered to make it seem to support elimination, despite strong evidence to the contrary. I conclude that the arguments for elimination of the BE were largely pseudoscientific and the BE's elimination unwarranted by the evidence.

Was the DSM-5 (American Psychiatric Association 2013) revision process scientifically based? The DSM-5 Task Force repeatedly asserted that the changes in the manual would be made on the basis of scientific evidence. In this chapter, focusing on DSM-5's decision to eliminate the bereavement exclusion (BE) to major depressive disorder (MDD), I explore the scientific quality of some of the arguments used in the debate over a proposed DSM-5 change. I argue that key assertions by those favoring the BE's elimination ("eliminationists") were in crucial respects pseudoscientific.

Like many others, I believe that the BE (explained below) was a sensible way to protect against overpathologizing the grief process. In fact, as the debate over the BE proceeded, the scientific support for the BE's validity and thus for its retention in DSM-5 became increasingly persuasive, anchored in multiple epidemiological studies replicated across several major data sets (Gilman et al. 2012, 2013; Mojtabai

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2011; Wakefield and Schmitz 2012a, b, 2013a, b, c, d, 2014; Wakefield et al. 2007, 2011a, b). The proposed elimination was criticized in editorials in leading medical journals, such as *Lancet* (Editorial 2012) and *New England Journal of Medicine* (Friedman 2012). Claims were made and tested, and a clear, scientifically satisfying outcome emerged supporting the validity of the BE (Wakefield 2013a, b, c).

Yet the BE was eliminated. For those bewildered by the BE's elimination, the post-mortem question arises of how this came to pass. Was it a matter of scientific debate and attention to the nature of the scientific evidence, as the DSM-5 Task Force insists? If not, by what manner of argument or forms of rhetoric was the question at issue obfuscated, diverted, or misconstrued, so that the scientific evidence was sidelined, rendered impotent, or outmaneuvered? I explore these questions by considering three central arguments from the DSM-5 BE debate: that the BE had to be eliminated for reasons of consistency; that the BE excluded cases from MDD that would respond to treatment; and that the BE leads us to miss suicidal cases.

Terminology is challenging in a discussion like this. Because the issue is whether certain periods of sadness are psychiatric disorders or normal reactions, and these two domains tend to involve quite different terminology, the discussion can be become quite tortured as one flips from one vocabulary to another, and one's choice of vocabulary can appear to beg the very question being disputed. In this paper, for convenience I adopt the standard terminological convention in discussions of the DSM-5, which is that I often use the medical vocabulary to describe the conditions whose status is being debated, but these terms are neutral in such contexts in that they do not imply disorder. Thus, terms generally associated with disorder such as "symptom," "depression," and "diagnosis" are used descriptively, such that normal grief has "symptoms" such as sadness and insomnia, "depression" is sometimes part of normal grief, and one can "diagnose" a normal condition. Obviously, the use of these terms does tend to medicalize the discussion, but properly understood, it need not bias the outcome. I also use the phrase "depressive episode" neutrally to denote any condition that satisfies the DSM's symptom and duration criteria for major depressive disorder (i.e., at least 5 symptoms for at least two weeks), but again it remains open to dispute whether such depressive episodes are sometimes part of normal grief or always instances of depressive disorder. In fact, "depression" has long been common as a description of both pathological depression and normal experiences of depression due to life's vicissitudes (e.g., Clayton et al. 1974).

The DSM-III Through DSM-IV Bereavement Exclusion

Sadness is a biologically designed emotional reaction to loss and stress seen in other species as well, a view put forward by Darwin (1872) and further explored and supported in a vast theoretical and empirical literature on the nature and evolution of sadness (e.g., Bowlby 1980; Ekman and Friesen 1971; Freud 1917; Horwitz and Wakefield 2007; McGuire et al. 1997; Nesse 2000, 2009; Nettle 2004; Price 1967;

Price et al. 1994; Sloman et al. 1994; Watson and Andrews 2002; Welling 2003). The intensity of sadness generally tends to be roughly proportional to the magnitude of a loss and to ameliorate over time as the individual reconstructs his or her meaning system and adapts to the changed situation. However, the intensity and duration of the reaction and the nature of the specific events that trigger the reaction vary to some extent across cultures and among individuals within a culture. The precise functions of sadness remain a matter of scientific investigation, but negative emotions like sadness may be analogous to physical pain in focusing our attention on addressing a challenge, and evidence suggests that the withdrawal and rumination that occurs during grief may help us to readapt to a changed environment (Andrews and Thompson 2009; Horwitz and Wakefield 2007).

Throughout medical history, physicians have observed that intense normal sadness in response to life events can include many of the same symptoms of distress as occur in depressive disorder (Horwitz and Wakefield 2007). They have also observed that loss can trigger pathological depressive reactions that go beyond the range of normal response to loss and that continue in a morbid trajectory without adaptation and with severe symptoms (Parkes 1964). Thus, when diagnosing intense sadness with its associated symptoms, physicians traditionally have asked: all considered, is there a sufficient cause in the individual's circumstances to explain the individual's condition as likely a normal reaction to loss or stress, or is the condition so severe or enduring or independent of context to be better explained as a pathological failure of normal mood regulation?

Starting with the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III) (American Psychiatric Association 1980), diagnosis of major depressive disorder (MDD) was based on operationalized descriptive diagnostic criteria that largely ignored the context of symptoms, essentially requiring that someone must experience 5 or more out of 9 specified symptoms for at least 2 weeks to be diagnosed with MDD. There was one exception, however, to the DSM's context-free approach to MDD diagnosis. Paula Clayton et al.'s (1968) classic longitudinal investigation of normal grief, using a nonclinical sample of relatives of individuals who had died, empirically established for the specific context of bereavement what the physicians had observed since antiquity, that normal grief after the death of a loved one routinely includes many of the symptoms that the DSM was using to diagnose MDD. Among the DSM's symptoms of depression, Clayton et al. distinguished those that are also common manifestations of normal general distress reactions, such as sadness, insomnia, fatigue, difficulty concentrating, decreased appetite, and loss of interest in usual activities, from the symptoms that are more patho-suggestive and tend to be distinctive of depressive disorder. For example, in Clayton's normal sample, in the first weeks post-loss, 87 % of bereaved subjects reported depressed mood, 85 % sleep disturbance, 79 % crying, and about half reported each of diminished interest in usual activities, difficulty concentrating, and lessened appetite, all of which appear among the DSM symptoms used to diagnose depression. Such bereavement-related episodes containing only general-distress-type depressive symptoms remitted on their own over time and did not cause the kind of marked impairment that frequently leads to psychiatric consultation

and care. In contrast, such symptoms as psychotic ideation, self-condemnation, suicidal ideation, and psychomotor retardation were rare in these normal cases but more common in a comparison group of pathological cases of depression unrelated to bereavement (Clayton et al. 1974). Some degree of withdrawal and thus role impairment was common among the normally bereaved, whereas severe impairment or very prolonged course were rare.

Clayton's research yielded an empirically supported approach to distinguishing between normal and disordered reactive depressions triggered by bereavement. In effect, the symptoms used to identify depression fell into two categories noted above, namely, those that occur in depressive disorder but are not distinctive of disorder and are present in normal distress responses as well, and those that are more distinctive of pathological episodes. The critical point was that with major depression having a 5-symptom threshold for diagnosis, one could have enough of the general distress symptoms during normal bereavement that one could be misdiagnosed as having a depressive disorder. Indeed, many individuals among nonclinical grievers – 42 % in Clayton's original studies (Hensley and Clayton 2013) – do reach the DSM's 5-symptom threshold for MDD at some point in the first weeks after loss, and almost all of the rest have some subthreshold depressive symptoms as part of their normal grieving.

Thus, to prevent a massive number of false positive diagnoses among normally grieving individuals, an exclusion clause was added to DSM-III criteria for MDD. This clause became known as the "bereavement exclusion" (BE) and, although changing over time in its details, it was retained in similar form through to DSM-IV. It specifies which depressive episodes during bereavement should be presumed to be normal and which are likely pathological. In its most recent incarnation, the BE specifies that, when a depressive episode satisfies the DSM's symptom and duration criteria for MDD but follows the death of a loved one, it can be considered a depressive disorder only if:

> E. The symptoms are not better accounted for by Bereavement, i.e., after the loss of a loved one, the symptoms persist for longer than 2 months or are characterized by marked functional impairment, morbid preoccupation with worthlessness, suicidal ideation, psychotic symptoms, or psychomotor retardation. (American Psychiatric Association 2000, p. 356)

The BE states that a depressive reaction should not be diagnosed as MDD if it can be better explained as part of an intense normal reaction, and it offers guidelines as to which sorts of symptoms generally suggest that it cannot be so explained. Thus, in effect it says that to qualify for exclusion from MDD diagnosis, a bereavement-related DSM-defined depressive episode must meet six tests: (1) no psychotic ideation (2) duration of no more than 2 months, by which point it must remit; (3) does not cause severe impairment in role functioning; (4) no suicidal ideation; (5) no psychomotor retardation (i.e., no general and observable slowing down of thought and movement); and (6) the bereaved individual must not suffer from a morbid preoccupation with his or her worthlessness as a human being. Episodes during bereavement that meet these six requirements are considered "uncomplicated" and

classified as normal. All other depressive episodes during bereavement that fail one or more of these six tests are classified as "complicated" and are diagnosed as MDD despite the recent loss.

The Definition of the BE and the "Eliminate or Extend" Argument

You cannot have a rational scientific debate about whether a claim is true if there is lack of agreement or lack of clarity about the nature of the claim. Of course, as sociologists of science would no doubt observe, illogical arguments can sometimes lead to scientific progress. However, that is selective attention; mostly, confusion about what is being disputed leads to nonsense. Scientific discourse generally starts with consensually agreed and precise statements of the propositions for which evidence is being marshaled so that the evidence can be rationally brought to bear on a fixed target claim over time. Illogic can be fertile at times, but generally not when the question is specifically whether the evidence logically supports a publicly stated and reasonably precise claim.

This commonsense principle, that a target claim under dispute should be accurately and nontendentiously stated to advance scientific understanding, was repeatedly violated by the BE eliminationists, making rational discussion of the BE's validity virtually impossible. In particular, the eliminationists frequently misstated the meaning of the BE itself in ways that made it appear less valid or more open to counterargument than it was. Misstating one's opponent's position for argumentational advantage is called the "straw-man fallacy," and it is one of the most common forms of pseudoscientific argument. I focus my discussion on only the most egregious examples of misstatements by those who had unusual authority in the discussion of the BE and thus potentially did the most damage to the scientific integrity of the dispute.

Professor Kenneth Kendler was without doubt the most scientifically eminent of the staunch eliminationists. He is a world-renowned research psychiatrist specializing in the genetic underpinnings of psychiatric disorder, an expert on depression, and a sophisticated analyst of the conceptual foundations of psychiatry. He was also one of the few individuals to have actually done empirical research on the BE. Consequently, when in 2010 the proposal to eliminate the BE was criticized in an opinion piece in the New York Times by Allen Frances (2010), the DSM-5 mood disorders work group, which was responsible for the proposal, turned to Kendler, who was at that time a member of the work group, to write an official rebuttal and explanation for why the BE should be eliminated. Kendler's (2010) official statement was placed on the DSM-5 website, and remains the most detailed and serious attempt by the work group to officially state its reasoning. (Kendler later left the work group and ascended to Chair of the Scientific Review Committee reviewing DSM-5 proposals from all the work groups for their scientific merit, a committee whose proceedings and findings remain secret to this day [see Demazeux, this volume])

Kendler begins his statement by explaining that misconceptions about the proposal to eliminate the grief exclusion from DSM-5 have been presented in the media, so he aims to provide "some insight into thinking behind the proposal." He begins his argument proper with the point that the BE did not exist in earlier diagnostic systems, in the course of which he provides a definition of the BE:

> First, the grief exclusion criterion – which states that someone who has experienced a recent bereavement is not eligible for a diagnosis of major depression – was not present in the two major psychiatric diagnostic systems that formed the basis for the DSM-III – the diagnostic manual that is the immediate precursor of our current DSM-IV. (Kendler 2010)

Kendler (2010) asserts that no BE existed before the DSM-III, presumably suggesting that the DSM's exclusion was a novel and potentially arbitrary deviation from the baseline of medical thought. However, Kendler's assertion that such exclusions were not present in earlier systems of diagnosis is simply untrue (Wakefield 2011). The two pre-DSM-III diagnostic systems to which Professor Kendler refers are the Feighner (1972) criteria (named after the first author of the paper reporting them) and the subsequently refined Research Diagnostic Criteria (RDC) (Spitzer et al. 1975a, b, 1978). With respect to the Feighner criteria, the BE was not stated in the formal diagnostic criteria but was included in the instruction to raters. We know this because when Eli Robins, a coauthor of the Feighner paper, worked with Robert Spitzer and Jean Endicott on the RDC, he explained to them that Feighner-criteria raters had been routinely cautioned not to diagnose an individual as having a depressive disorder if the individual had recently suffered the loss of a loved one, even if the individual met full Feighner criteria for depression (Jean Endicott, personal communication, October 15 2009).

Consequently, Spitzer, Endicott, and Robins took a similar approach in the RDC criteria, and placed the BE in manuals of instructions to raters (Robert Spitzer, personal communication, October 29, 2010; New York State Department of Mental Hygiene 1980). Kendler's account is contradicted by the fact that the BE is cited as a requirement of the RDC and applied in major pre-DSM-III epidemiological studies of depression. Weissman and Myers (1978) state, for example, that "Because of the overlap in presenting symptoms, there is an effort in the RDC to separate clinical depression from normal grief reactions secondary to the death of a 'significant other'- termed grief . . . .Symptoms that lasted more than a year were considered symptomatic of major depression" (p. 1306).

Kendler's claims are not just inaccurate regarding the Feighner and RDC criteria but misleading about the earlier history of psychiatry as well. To take one salient example, Emil Kraepelin, the preeminent nineteenth-century diagnostic theoretician who inspired the DSM approach to depression and who Kendler (1990) elsewhere cites as the seminal thinker about depression, believed that intense normal sadness in response to grief and other reverses in life circumstances could symptomatically look just like major depressive disorder but have a different prognosis. The clinician's diagnosis, according to Kraepelin, depends on an examination of the context of the symptoms:

> Several times patients have been brought to me, whose deep dejection, poverty of expression, and anxious tension tempt to the assumption of a circular [pathological] depression, while it came out afterwards, that they were cases of moodiness, which had for their cause serious delinquencies and threatened legal proceedings... [T]he slighter depressions of manic-depressive insanity, as far as we are able to make a survey, may wholly resemble the well-founded moodiness of health. (Kraepelin 1917)

However, historical inaccuracies aside, what is most stunning about the passage in Kendler's statement quoted above is his characterization of the BE. He asserts that "the grief exclusion criterion... states that someone who has experienced a recent bereavement is not eligible for a diagnosis of major depression." That is, Kendler starts his discussion by defining the BE as excluding *all* bereaved people from MDD diagnosis.

This, as we saw, is a mischaracterization of the BE, which excludes a limited subgroup of those experiencing bereavement-related depression who must satisfy six demanding criteria that indicate a normal-range depressive response to loss. Kendler's incorrect characterization of the BE is not random error but rather highly tendentious. It makes the BE look like a much broader exclusion than it is, thus potentially making it look less reasonable than it is and setting up a straw-man position as a target for criticism. As far as I know, no one in the debate over the BE held that all bereaved individuals (or even all bereaved individuals with shorter than 2-month episodes) should be excluded from MDD diagnosis. As noted, it has been well-known since antiquity and confirmed repeatedly in modern studies that some people do develop a depressive disorder during an extreme grief reaction to the loss of a loved one, sometimes even to the point of sinking into chronic psychotic melancholia. So, if the BE claimed that "someone who has experienced a recent bereavement is not eligible for a diagnosis of major depression," it would be manifestly invalid. Having misstated the BE at the outset and set up a weak straw-man position that nobody holds as the target for his critique, Kendler's official statement becomes potentially irrelevant to the question of whether the real BE is valid.

Is it possible that Kendler's initial characterization of the BE is just an abbreviated introductory explanation that is elaborated later in the statement? No, nowhere later in his statement does Kendler accurately restate the BE. To the contrary, he reasserts the same interpretation in his pivotal argument. I (Wakefield et al. 2007) had earlier proposed that the BE should be expanded to apply to uncomplicated depressive reactions to losses and stressors other than bereavement (e.g., marital dissolution, job loss). Kendler argues against this proposal by stating that if the BE were extended to other stressors, then "no depression that arises in the setting of adversity would be diagnosable." This statement presupposes (like his initial statement) that the BE excludes all bereavement-related depressive episodes, so if extended to other stressors it would similarly exclude all stressor-related episodes. But, of course, that is not how the BE works; it only excludes episodes that meet certain stringent conditions. So, the distortion in the initial statement was not a transient approximation but rather the target of Kendler's entire analysis. (I return below to the argument about extending the BE.)

Is it possible that Kendler, an expert on depression diagnosis, never noticed or grasped the details of the BE's diagnostic criteria that he is attacking? No, lack of a clear understanding of the BE cannot be the explanation because in a scientific paper he senior-authored in the prestigious *American Journal of Psychiatry* just 2 years before issuing his official statement (Kendler et al. 2008), Kendler had quite correctly defined the BE, including, and even specifying, the features of the BE that he could not apply in the analysis:

> [W]e operationalized the DSM-IV "normal grief criterion" as a duration of $\leq$2 months and an absence of psychomotor retardation, suicidal ideation, and severe work impairment. We could thus determine whether each depressive episode met these proposed criteria for a normal grief response. Our interviews did not inquire about psychotic symptoms related to bereavement or "morbid preoccupation with worthlessness," the other DSM-IV criteria for normal grief. (Kendler et al. 2008, p. 1450)

Clearly, Kendler knew that the BE says nothing remotely like "someone who has experienced a recent bereavement is not eligible for a diagnosis of major depression." But perhaps the statement of the correct criteria at the beginning of the 2008 research article was just paying lip service to the correct criteria for scholars, and the research article then went on to use the broader notion that appears in Kendler's statement in the analyses? No, in the research article, the correct definition of the BE is systematically and consistently used throughout. In the article's Table 1, in a column listing analyzed variables, there is a sublist of variables titled "DSM-IV exclusion criteria" that lists the features relevant to evaluating the BE's applicability: "Duration of >2 months"; "Psychomotor retardation"; "suicidal ideation"; and "Severe work impairment" (Kendler et al. 2008, p. 1451). At the end of the list, there is a summary variable that is in part, 'Meets criteria for 'uncomplicated bereavement-related disorder . . . '." The interaction analysis performed in the study specifically uses these correct "normal grief" criteria.

Even if Kendler knew and applied the correct BE exclusion, perhaps he was under the impression that virtually all bereavement-related depressive episodes in fact do satisfy the BE's six requirements for exclusion? If so, then he would have believed that in effect, as a practical matter, the BE eliminates virtually anyone whose depressive episode followed loss of a loved one, and his statement's definition would be only technically incorrect.

However, Kendler knew to the contrary that the BE eliminates only a minority of bereavement-related depression, and that his mischaracterization was not even approximately correct. We know this because one of the major results of Kendler's et al. (2008) study was that only a minority of bereavement-related depressive episodes is excluded by the BE. In Table 1, the row labeled partly "Meets criteria for 'uncomplicated bereavement-related disorder" indicates that only about a quarter (28 %) of all bereavement-related depressive episodes actually qualified for exclusion (p. 1451). Could this one row in a large table have gone unnoticed by Kendler, lost among the many analyses he performed? No, that is impossible too, because Kendler specifically commented on this result in his Discussion section: "A low percentage of individuals with bereavement-related depression met

criteria for symptoms and a course of illness consistent with 'normal grief'" (p. 1453). The finding of Kendler's own study showed that the BE excludes a modest "low percentage" of bereavement-related cases, a finding radically different from Kendler's claim in his Statement that all such cases are excluded.

One might still ask: did the misstatement of the BE really matter to Kendler's argument? Some of his points, such as the claim that no BE appeared in earlier diagnostic manuals, are relatively independent of the precise content of the BE and would not have been affected by his mischaracterization.

The answer is that Kendler's misstatement of the BE is not a side issue but is directly implicated in his most important argument for eliminating the BE. Kendler's central argument in his statement, and the argument that ended up being most influential in the BE debate, was based on findings that if you apply the BE's six criteria to depressive reactions to other stressors, you get a similarly benign-looking group. Wakefield et al. (2007) were the first systematically to apply the BE's criteria for normal depressive episodes to reactions to losses other than death of a loved one, such as relationship problems, marital dissolution, job loss, financial ruin, negative medical diagnosis in oneself or a loved one, loss of possessions in a disaster, and other such stresses and losses that are known to trigger depressive feelings. Using a series of 11 validators that indicate the degree to which a condition is likely pathological versus normal, ranging from measures of service use to recurrence and duration, they found that the excluded bereavement-related cases so closely resembled the other-stressor cases that satisfied the same BE criteria that they could not be statistically differentiated. Moreover, both these groups scored much lower on level of pathology than the episodes – both bereavement and other-stressor triggered – that did not satisfy the BE criteria. These results were replicated both in Kendler et al.'s (2008) subsequent study as well as in later follow-up studies that were methodologically more rigorous (Wakefield and Schmitz 2012a, 2013a, b, c, d, 2014). Thus, it is inconsistent with the evidence to exclude uncomplicated bereavement-related episodes but not to exclude uncomplicated other-stressor related episodes. How this inconsistency is resolved – by eliminating the BE or by extending its rules to the exclusion of depressive reactions to other stressors – was framed as a central issue by Kendler.

Kendler consistently dismissed out of hand the alternative of extending the BE, but demanded consistency with the evidence showing that the BE must be eliminated or extended, thus reduced to absurdity the notion of retaining the BE. This central argument emerged in his statement as follows:

> [A] broad range of evidence agreed to by both sides of this debate shows that there are little to no systematic differences between individuals who develop a major depression in response to bereavement and in response to other severe stressors... So the DSM-IV position is not logically defensible. Either the grief exclusion criterion needs to be eliminated or extended so that no depression that arises in the setting of adversity would be diagnosable. This latter approach would represent as major shift, unsupported by a range of scientific evidence, in the nature of our concept of depression as epidemiologic studies show that the majority of individuals develop major depression in the setting of psychosocial adversity. (Kendler 2010)

Certainly no one wants to eliminate all adversity-triggered episodes from MDD. Also, it is correct that by far most episodes of MDD in community studies are adversity-triggered. However, when he penned this passage, Kendler knew that the argument's dramatic pivotal claim, that extending the BE to other stressors would eliminate *all* adversity-triggered episodes from MDD so that "no depression that arises in the setting of adversity would be diagnosable," and that therefore depression as we know it would mostly disappear from psychiatric diagnosis except for untriggered engoneous depression, was untrue. Table 1 of his own 2008 study of the BE and its extension shows that, of all adversity-triggered MDD episodes (including both grief and other triggers), only about a quarter (25.7 %) are excluded by an extended BE (28.1 % of bereavement-related episodes and 24.6 % of other-stressor triggered episodes) (Kendler et al. 2008, p. 1451). Moreover, Kendler highlighted the low percentage of other-stressor cases that would be excluded by an extended BE in his paper's Discussion section. There, immediately after noting that "a low percentage" of bereavement-related episodes satisfied the BE's exclusion requirements, he observes that "the same percentage" of other-stress triggered episodes satisfied the extended BE (p. 1453).

However, Kendler's *reductio ad absurdum* argument is no longer valid if, as his and other research establishes, only a minority of other-stressor-triggered MDD is eliminated by extending the BE. The *reductio* step is no longer compelling because, to many observers of the extraordinarily high community prevalence rates of DSM-defined MDD, it is not at all absurd on its face that some percentage of DSM-defined MDD cases are in fact intense normal sadness reactions to various losses other than death of a loved one (Clayton et al. 1968; Maj 2011a,b; Regier et al. 1998). If this issue is taken seriously, then rather than a simple armchair reduction to absurdity, the question of the BE's status becomes the scientific challenge of understanding the nature of other-stressor-triggered uncomplicated depression cases, and of looking at the proper status of uncomplicated triggered depressions overall. This is an issue that has never been addressed by any DSM review group, and was never even broached by the DSM-5 work group. It was left to others to address the question empirically.

So, there is no longer an automatic reduction to absurdity of retaining and extending the BE once the implications are accurately stated. Yet, in all likelihood at least partly due to Kendler's influence, the notion that extending the BE to other stressors would lead to a diagnostic disaster in which virtually all cases of depression as currently diagnosed would no longer be diagnosable became firmly presupposed by the eliminationists and the DSM-5 work group, so that the work group offered no serious analysis at all of the evidence for and against this option. It instead took the pseudoscientific path of insisting that its own position was supported even as it refused to address the major alternative hypothesis. Even when directly challenged (Wakefield et al. 2009), Kendler refused to provide analyses from his 2008 article's data that would address the validity of extending the BE by comparing all uncomplicated to all complicated triggered cases (Kendler and Zisook 2009). The "extend" option was ruled out of bounds based on the made-up idea that it would depathologize most MDD, then the "consistency" argument that demanded either to extend or to eliminate was used to perform the *coup de grâce* on the BE.

Kendler wrote one way, with scientific accuracy, when addressing his colleagues in research journals, then wrote in an entirely different and contradictory way when arguing for the elimination of the BE in the context of the DSM-5 revision. His research correctly states the BE criteria, the low percentage of bereavement-related cases that the BE excludes, and the comparably low percentage of other-stressor reactions that an extended BE would exclude, but his official DSM-5 website statement supporting the elimination of the BE offers radically different and incorrect versions of all these facts and definitions. That gap reveals the failure of the DSM-5 revision process, and specifically the elimination of the BE, to be scientifically defensible.

In fact, the evidence now overwhelmingly supports the validity of the BE itself and of the "extend" option. Study after study has shown that both BE-excluded cases and the cases excluded under an extended BE do not have the core features of MDD, such as recurrence and suicide attempts (Mojtabai 2011; Paksarian and Mojtabai 2013; Gilman et al. 2012, 2013; Wakefield 2013a, b; Wakefield and Schmitz 2012a, b, 2013a, b, c, d, 2014; Wakefield et al. 2007, 2011a, b). Indeed, given Kendler's great concern about consistency, he might consider the following inconsistency. In a classic article on validation of disorder categories, Kendler (1990) pointed out that "for a number of disorders, one or two [validators] are implicitly more important than the others, because they reflect the key defining features or 'construct' of the disorder" (p. 970). He points out that this is particularly the case when it comes to the recurrent course of mood disorders: "For Kraepelin, the 'construct' of ... manic-depressive insanity assumed a relapsing disorder without deterioration" and thus "course and outcome would be the most important validators" (p. 970). The notion that course is a critical validator was also expressed by other eliminationists who, on the basis of no relevant data at all (Wakefield and First 2012), generally claimed that BE-excluded uncomplicated bereavement-related depressive episodes had the same course and outcome as other major depression (Pies 2009; Zisook and Kendler 2007; Zisook et al. 2007). However, once appropriate studies were done, it consistently emerged that adversity-triggered uncomplicated depression cases, whether bereavement- or other-stressor related, do not have elevated rates of recurrence over background population levels of depression incidence among those who have never had a depressive disorder, whereas other depressive disorder has highly elevated recurrence rates, as the Kraepelinian formulation would predict. Thus, the extended BE excludes cases that, judged by Kendler's own analysis performed outside the politics of the DSM-5, do not satisfy the crucial validating criterion for MDD.

Kendler is by no means the only one to engage in brazen straw-man tactics. Such tactics were routinely used by those defending the BE's elimination to exaggerate the impact of the BE and make it seem unreasonable. For example, in a *Scientific American* blog published just a few months before DSM-5 was published, Professor Sidney Zisook, the primary consultant to the DSM-5 mood disorders work group on the BE to whom the committee outsourced much of its work on the BE (and who had been arguing for the elimination of the BE since 1991), explained:

> The "exclusion" essentially detailed a two-month period of "normal grief" that people would experience after the loss of a loved one. During this period, it was all but forbidden to diagnose a patient with major depression—even if the individual had all the symptoms (which are, in important and sometimes life-threatening ways, different from grief). (Zisook 2013)

Zisook's claim that, according to the BE, diagnosis of depression during the first months of grief was "all but forbidden," is of course incorrect. Indeed, he was a coauthor of the study senior-authored by Kendler that showed that the BE actually excluded from MDD only a "low percentage" of bereavement-related depressive episodes overall. It is instructive to consider what the 2008 study of which Zisook was a coauthor actually showed about the first 2 months in particular. According to that study's Table 1, about 67 % of bereavement-related depressive episodes lasted 2 months or less, but only 28 % of bereavement-related episodes satisfied criteria for BE exclusion. So, of bereavement-related episodes lasting 2 months or less, 42 % qualified for exclusion, whereas 58 % were diagnosable as MDD despite the recent loss. That is quite different from diagnosis being "all but forbidden" by the BE in the first 2 months. One might argue – and I believe the research shows quite convincingly – that in fact the extent of misdiagnosis of normal reactions as major depression was underestimated by the BE and that the threshold for diagnosis should be even higher. For example, the research suggests that a much more generous durational limit for normal depressive episodes after loss, closer to between 6 months and 1 year, is more valid than the DSM-IV 2-month limit (Wakefield et al. 2011a, b). However, that is another matter. The point here is that even the BE in its overly constrained DSM-IV form was misrepresented.

Particularly egregious instances of misleading statements of the BE are those issued by the DSM-5 Task Force and asserted by its leaders, thus giving an official imprimatur to such distortions. An example is the authoritative explanation offered by the Chair of the DSM-5 Task Force himself, Professor David Kupfer, in a short video featured on the DSM informational website aimed at explaining the exclusion's elimination. Kupfer (2013) states: "[A]fter reviewing the literature and having a number of our advisors go over all the material that was available, we decided to remove the fact that clinicians should not make a diagnosis of clinical depression in anybody who has suffered a loss before two or three months." But, of course, the BE states no such thing. Similarly, in the DSM-5 itself, in the appendix chapter presenting the highlights of the changes to the manual, it is explained that "In DSM-IV, there was an exclusion criterion for a major depressive episode that was applied to depressive symptoms lasting less than 2 months following the death of a loved one (i.e., the bereavement exclusion)" (DSM-5, p. 811).

A further official "fact sheet" on the bereavement exclusion from the DSM-5 Task Force that is posted on the DSM-5 website explains:

> Using DSM-IV, clinicians were advised to refrain from diagnosing major depression in individuals within the first two months following the death of a loved one in what has been referred to as the 'bereavement exclusion.' By advising clinicians not to diagnose

> depression in recently bereaved individuals, the DSM-IV bereavement exclusion suggested that grief somehow protected someone from major depression. (American Psychiatric Association 2013b)

However, the BE, we have seen, classified most depression during bereavement, and even most depression during the first 2 months after loss, as MDD. The Fact Sheet's misstatement that the BE prohibited diagnosis of MDD during the first 2 months yields a bogus *reductio* argument because it is implausible that there is no pathology among depressive episodes early in grief. The BE is constructed so as to identify those early episodes based on five symptom and impairment criteria.

The Medication Responsiveness Argument

One argument repeatedly put forward for eliminating the BE is that there is evidence that medication works with excluded cases, therefore the cases should be considered depressive disorders. When Jan Fawcett (2010), Chair of the DSM-5 mood disorders work group, initially presented the work group's proposed changes, he credited treatment responsiveness as the sole reason for eliminating the BE: "The Mood Disorders Workgroup has decided to remove the bereavement exclusion from the major depressive episode diagnosis based on data indicating that when a patient meets the criteria for a major depressive episode, the response to treatment is identical to that for any major stressor preceding a major depression" (p. 536). Fawcett (2012) repeated this claim in a later commentary on why the BE was eliminated: "People who develop major depression from or after bereavement respond the same way to treatment, even to medications, as people who develop depression that comes out of nowhere."

The medication response claim is dubious from the start as an argument for considering a condition to be pathological, since many psychotropic medications have an effect across a large swath of normal and disordered conditions. So, it would not be particularly surprising for there to be a treatment response even if BE-excluded depressive feelings are perfectly normal. For example, stimulants make everyone, not just those with ADHD, more focused and alert, including, say, those getting tired while staying up all night studying for college exams, but that does not support the claim that the inability to stay up all night studying for college exams without getting tired is a disorder. It is true that sometimes the fact that two similar conditions do not respond to the same medication may suggest that they are different disorders, an approach to validation known as "pharmacological dissection." However, there is no similar basis for a "pharmacological assimilation" thesis that when two conditions do respond to the same medication, they are likely the same disorder, because a given drug can influence an array of normal and disordered conditions.

Has the claim that medication is helpful in BE-excluded depression at least been scientifically demonstrated by standard empirical methods, as Fawcett's claims

imply? Despite the logical flaw pointed out above in the argument against the BE based on medication effectiveness, the scientific status of such claims about treatment effectiveness remain important. Aside from the fact that people want to know the answer to this question due to the extremely painful nature of grief, in the BE debate there are many who would be swayed by a pragmatic argument that we know that medication helps, therefore we might as well provide a way to diagnose it. The frequency with which this argument was put forward by the eliminationists testifies to its perceived persuasive power.

Fawcett's support for his claim can be traced back to Zisook and Kendler's (2007) mention of one earlier paper by Zisook et al.: "The only treatment study of individuals who the DSM would diagnose with 'bereavement' rather than MDD based on time since death also found [bereavement-related depression] to respond to antidepressant medication similar to other studies of SMD (Zisook et al. 2001)" (Zisook and Kendler 2007, p. 789). There have been no relevant studies since, and the same single reference is cited by several other authors to support eliminating the BE (e.g., Gilman et al. 2012; Shear 2011).

However, the Zisook et al. study is so weak as to be scientifically meaningless, and it would not be taken seriously as scientific evidence in any other medical specialty. Zisook et al. (2001) treated 22 bereaved individuals who satisfied DSM-IV MDD criteria at about 6–8 weeks post-loss on average. Only the BE criteria requiring no psychotic or suicidal ideation were applied. Subjects were treated for 8 weeks, and 13 out of the 22 subjects (59 %) experienced a reduction of $\geq$50 % in symptom scores on a standard inventory of depressive symptoms. The study's fatal flaw is that it contains no control group, so the sample's modest "response rate" is impossible to interpret. This is because the medication was administered during a period – roughly between 6 and 14 weeks post-loss – in which, without treatment, normal bereavement-related depressions have precipitous drops in symptoms anyway, at roughly the same rate as was observed in the study. Zisook et al. themselves acknowledge that "because of the open, uncontrolled design, it is impossible to be sure that the observed changes were due to the effects of bupropion SR . . . " (p. 229).

For comparison purposes, in Clayton et al.'s (1968) prospective study of normal bereavement, in the period from the first month to on average about 3 months after loss, roughly comparable to Zisook et al.'s period of treatment, the percentages of Clayton's sample having six high-prevalence depressive symptoms decreased as follows: depressed mood, 87–12 %; sleep disturbance, 85–27 %; crying, 79–12 %; difficulty concentrating, 47–27 %; loss of interest in TV, news, friends, 42–19 %; anorexia and/or weight loss, 49–27 %. Virtually none of these subjects (3 %) were taking medication. In Zisook's own earlier study of depression in bereavement (Zisook and Shuchter 1991, 1993), out of about 75 non-recurrent depressives who had early depressions triggered by a loss, 30 (40 %) were no longer depressed by 2 months, almost all without medication, and Zisook himself observed the need for placebo controls in medication studies. Zisook's bupropion data are thus quite consistent with trajectories of resolution for depressive symptoms during early grief without medication.

The Suicide Risk Argument

There is a great emphasis in contemporary discussion of depression on this disorder's potential for suicide. Nonetheless, one would think that making statements about heightened suicide risk without appropriate evidence, apart from being antithetical to reasoned scientific discourse, is inappropriate as a way to score points in a dispute over a DSM-5 proposal. Yet, this is precisely what happened in the BE debate. Without supportive evidence to back up their assertions, proponents of eliminating the BE repeatedly raised the specter of suicide in individuals excluded from MDD diagnosis by the BE. In doing so, they carefully avoided addressing or even mentioning the fact that the BE is constructed to prevent any such error, since the BE exclusion requires absence of suicidal ideation. The suicidal ideation criterion directly challenges the plausibility of the eliminationists' claims, so it was effectively banished from their discussions. This was pseudoscience, in which reliance on emotions of fear and selective attention to facts replaced careful assessment of evidence.

For example, in a review paper, Zisook, Shear, and Kendler (2007) cited the risk of suicide as a reason for not waiting for BE-excluded depressive feelings to subside on their own without early treatment. To support their point, they noted that "a recent study demonstrated that both lack of a partner and time in depression were significant predictors of suicidality among people meeting criteria for MDE" (p. 104). The study that Zisook et al. cited (Sokero et al. 2005) concerned largely severely pathological inpatient subjects, many of whom had prior suicide attempts. For example, "preceding the follow-up phase, 15 % of the cohort had attempted suicide during the index episode and 24 % before that" (pp. 316–317). This is far cry from a sample with only general-distress symptoms and no suicidal ideation, and it is a sample wholly irrelevant to predicting behavior by individuals with uncomplicated bereavement-related depressions that satisfy the six demanding conditions required for exclusion. Zisook, Shear, and Kendler's logic seemed to be that depressed people without partners are generally more likely to commit suicide, and bereaved individuals (if the loss was of a spouse) do not have partners, thus depressed bereaved individuals are at elevated risk for suicide, thus BE-excluded cases – which are bereaved and depressed – are at elevated risk for suicide. This reasoning commits the elementary fallacy of ignoring a crucial fact that influences the outcome of interest, namely, that the BE-excluded individuals are specifically screened to avoid suicide risk by prohibiting suicidal ideation and other risky symptoms such as a sense of worthlessness.

Zisook (2010), in arguing for elimination of the BE, declared on public radio that "I'd rather make the mistake of calling someone depressed who may not be depressed, than missing the diagnosis of depression, not treating it, and having that person kill themselves." He failed to mention to the public that cases are screened for suicidal ideation as part of the assessment for exclusion.

Shear et al. (2011), in explaining why they agree with Zisook (who was a coauthor of the paper) that the BE should be eliminated, argue that early intervention

with depressive feelings even during acute grief is warranted because "bereavement may increase the risk of suicide" (p. 111). Shear et al. fail to mention that BE exclusion requires that there be no suicidal ideation. They cite two references to support their contention that the BE poses a suicide risk (Ajdacic-Gross et al. 2008; Stroebe et al. 2005). Both concern increased suicide risk in general during bereavement, and neither address uncomplicated cases or cases in which suicidal ideation is absent, rendering them irrelevant to assessing suicidal risk in BE-excluded, uncomplicated cases.

In these citations of supposed support for eliminationist claims, there is a general lack of thoughtfulness about whether the findings of the study are pertinent and revealing in the way claimed about the question at issue. For example, Stroebe et al.'s entire study uses suicidal ideation as a proxy for suicide, concluding that: "Bereaved persons are at excess risk of suicidal ideation compared to nonbereaved people. Heightened suicidal ideation in bereavement is associated with extreme emotional loneliness and severe depressive symptoms" (p. 2178), and Stroebe et al. remark that "ideation would seem a precursor to suicidal acts" (p. 2178). Yet, suicidal ideation is by definition not present in excluded cases. Moreover, the heightened suicidal ideation Stroebe et al. found in bereavement was associated specifically with severe depressive symptoms but not with more moderate depressive symptoms, whereas excluded cases are generally mild or moderate, because severe symptoms tend to be the pathosuggestive symptoms that block exclusion according to the BE. The findings of the cited study do not fit the context of their citation by Shear et al.

In a further article, Shear (2011) defends the elimination of the BE by asserting that the standard DSM depression criteria have been developed by experts and stood the test of time and thus should be applied during bereavement (thus refusing to consider the hypothesis that uncomplicated cases are distinct), and that "among the things we have learned are that MDD is associated with a high mortality from suicide (as many as 15 % of people with severe MDD die by suicide)." No citation is provided for these startling claims about suicide potential. However, the source of the 15 % rate is well-known (Guze and Robins 1970) and has long been repudiated (Bostwick and Pankratz 2000). It was the rate for a very severe population of hospitalized depressed individuals, many of whom had bipolar disorder, and the statistics were biased in several other ways as well. Shear simply assumes that whatever is known about MDD in general applies to BE-excluded cases as well, yet the homogeneity of uncomplicated cases with standard MDD is precisely what is denied by the BE. In the guise of a scientific paper, she is simply asserting her authority on the question based on no specific evidence pertaining to BE-excluded cases.

There is a basic point of scientific logic involved here. The claim regarding the BE is that excluded, uncomplicated episodes are a different and less pathological kind of condition from standard MDD. Consequently, citing general points about the seriousness of standard MDD to argue for the elimination of the BE, as eliminationists tended to do, begs the question at issue of whether BE-excluded cases are different from standard MDD. This sort of argument is in effect simply a way of refusing to seriously consider an alternative hypothesis, and that is the

mark of pseudoscience. Ignoring the fact that the BE includes a requirement for no suicidal ideation, which is directly relevant to suicide risk and thus renders irrelevant most studies of standard MDD suicide risk, is a further way of avoiding a scientific evaluation of an opposing view.

What is the truth about suicide risk in uncomplicated depression? The only published evidence regarding suicide risk in uncomplicated depression comes from my own studies with Mark Schmitz of uncomplicated depression. The evidence is overwhelmingly reassuring and supportive of the BE's validity (Wakefield and Schmitz 2014). We used four major epidemiological data sets to explore the relationship between uncomplicated depression and suicide attempt risk among those who experienced a period of sadness. Our results decisively falsified the claim by the eliminationists that exclusion of uncomplicated depression in an extended BE would risk missing suicidal MDD cases. In terms of both concurrent risk during the uncomplicated episode and predictive risk over 1- and 3-year follow-up periods, our results indicated that those depressive episodes that satisfy the demanding requirements for being uncomplicated depression predict rates of suicide attempt that are no more than, and often less than, those of the general population.

In our study, the "no suicidal ideation" component of the uncomplicated criteria by itself reduced suicide attempt rates to background population levels. However, the additional uncomplicated criteria reduced the rate further. It turned out to be simply false that excluding uncomplicated depression from MDD diagnosis poses a risk that the elevated rates of suicide among MDD patients will lead to missed suicidal cases, because uncomplicated depression does not have the elevated suicide attempt rates that are a major feature of standard MDD. In terms of concern about suicide risk, one might as well be concerned about the average person on the street as BE-excluded cases, because the BE-excluded rates are the same as or lower than background population levels in those who have never had MDD. Proponents of BE elimination in effect engaged in fear mongering in raising the specter of suicide without any relevant evidence.

Concluding Remarks

My analysis has led me to the conclusion that the arguments for elimination of the BE were largely pseudoscientific and the BE's elimination unwarranted by the evidence. In considering the line of argument leading me to this conclusion, three caveats are important to keep in mind. First, in arguing that pseudoscientific thinking was decisive in eliminating the BE, I do not deny that there may have been considerable nonsense and pseudoscience on the part of those defending the BE's retention as well. However, as is indicated in a sketchy way in the course of the discussion, the difference is that the scientific evidence supported retention. Second, although I have perhaps been rather harsh in criticizing the assertions of several colleagues for their unscientific performance in the BE debate, these same colleagues have provided many admirable and scientifically invaluable

accomplishments as well, and the concerns expressed herein on the specific issue of the BE in no way reflect a blanket judgment about any individual investigators or their work. Finally, the debate over the fate of the BE was long and complex and multifaceted, and unraveling fallacious reasoning is often much more demanding than putting forward the fallacious reasoning in the first place. Consequently, the debate could not be fully excavated in this one chapter, and many points and arguments made by the eliminationists and those defending the BE could not be addressed. Instead, I settled for three pivotal illustrative examples that offer a précis for a longer analysis of the debate hopefully to come.

The usual indicator that pseudoscience has replaced science is the systematic motivated deviation from rational assessment of evidence by a community in arguing for a claim that the community insists is scientifically supported but which in reality is not supported by the evidence. In pseudoscience, the basic canons of scientific discourse are systematically violated in such as a way as to attempt to create the false appearance of scientific discourse. Moreover, those putting forward the bogus arguments must be in a position to more appropriately evaluate the evidence but fail to do so. It is this sort of suspension of the usual scientific rules that I argue occurred on the part of the eliminationists in the debate over DSM-5's elimination of the BE.

When I say that support for the elimination of the BE was pseudoscientific, I mean this quite literally, and not as hyperbole for a vaguer assertion that the arguments for elimination were weak or incorrect. There was systematic abandonment of basic scientific and medical canons of reasoning. In my view, the arguments by the eliminationists were intellectually no sounder than claims that we routinely dismiss as pseudoscientific, such as the claims of astrology.

It is of course a potentially questionable and even desperate strategy to label one's opponents as pseudoscientists and rhetoricians, and to suggest that a process claimed to be scientific was merely a masquerade of science. It is generally better to get on with the difficult scientific work necessary to advance understanding, and this work has been undertaken by investigators cited above. Nevertheless, I believe "pseudoscience" is the accurate description of what happened in the BE debate, and it deserves to be labeled as such. This was after all a debate about whether millions of people experiencing grief should or should not be considered by psychiatry to have a mental disorder. Pretending that there was a scientific dispute that went one way rather than another as opposed to a gross deviation from scientific standards of reasoning would be to distort reality and to further betray the individuals newly subject to diagnosis.

A hidden cost of the BE debacle is that, in spuriously disputing the non-disorder classification of the cases identified by the BE and placing those cases within major depression, the DSM-5 work group effectively prevented serious exploration of whether other, less immediately obvious candidates might also be normal sadness or grief rather than depressive disorder. Surely not everyone who experiences suicidal ideation or feels worthless is mentally disordered, yet a wider-ranging discussion of the boundary between major depression and normal sadness never occurred once the most scientifically supportable cases of normal sadness were spuriously reclassified.

Recognizing the pseudoscientific status of the BE decision potentially opens up further discussion of other boundary questions.

The quality of the BE debate potentially has broader implications. The proposal to eliminate the BE was one of the most controversial in the DSMs history. This issue was "in the radar" of the Task Force Chair and Vice-Chair, who were confronted with a tidal wave of public challenges regarding this proposal. Thus, the quality of the BE discussion tells us something about the quality of the overall regulation of the DSM-5 revision process and the sorts of arguments that were taken as adequate for making decisions on proposals, although the quality of the scientific reasoning underlying DSM-5 revision proposals no doubt varied with the work groups and the individuals involved.

Science is an achievement that requires discipline of the mind's unruly elements, and pseudoscience is the use of the frame and vocabulary of science without the developed discipline of science. It is precisely the unruly antiscientific elements that are tempted to emerge in the DSM political process. Given emotionally charged issues such as grief and suicide risk for which many potential patients seek solace from mental health professionals, lack of quality control from a strong Chair may allow the revision process to spin out of control towards pseudoscience rather than the scientific evaluation of alternative hypotheses. The DSM-5 process in the case of the BE debate is a prototype and a cautionary tale of how a manual revision can thus quickly move psychiatry from science to pseudoscience, undermining rather than enhancing its status in the long run.

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