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Some Epistemological Concerns About Dissociative Identity Disorder and Diagnostic Practices in Psychology

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In this paper we argue that dissociative identity disorder (DID) is best interpreted as a causal model of a (possible) post-traumatic psychological process, as a mechanical model of an abnormal psychological condition. From this perspective we examine and criticize the evidential status of DID, and we demonstrate that there is really no good reason to believe that anyone has ever suffered from DID so understood. This is so because the proponents of DID violate basic methodological principles of good causal modeling.

> When every ounce of your concentration is fixed upon blasting a winged pig out of the sky, you do not question its species' ontological status.
>
> James Morrow, *City of Truth* (1990)

1. Introduction

Recently both the public and professional fascination with a variety of psychological disorders involving recovered memory reached something of a crescendo, and this fascination is a dangerous obsession by any standards of conduct, professional or private.[1] On the professional side, one can see this fascination merely by browsing through the DSM-IV. In doing so it quickly becomes clear to even the casual reader that there are numerous disorders that are explicitly characterized by some form of memory-related dysfunction or other. For better or worse, the symptoms and etiological assumptions behind the theories of dissociation, repression and recovered memory are employed in the diagnosis and clinical treatment of a variety of

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prominent psychological disorders, including the cluster of what are known as the dissociative disorders.

As we shall see, however, there are serious doubts about the ontological status of these disorders and the mechanisms in terms of which they are defined. Given such doubts the fact that clinical treatments are based on these models raises serious additional ethical issues.[2] Our main concern here will neither be to focus on the ethical dimensions of this problem in any great detail, although such concerns will be briefly addressed in section 8, nor will we be concerned with anything like sociological explanations as to why the models of these disorders have been accepted into the body of theoretical psychology given that they have such a questionable epistemological status. Rather, we will focus primarily on the epistemological status of claims concerning the existence of these disorders. In doing so we will show that there is little, if any, good evidence to suggest that theses models are instantiated in our world. For the purposes of this paper we will specifically focus on the case of what is known as dissociative identity disorder, or DID.[3]

2. Dissociation, Repression, and the Failure of Freud

In order to set the stage, we think that it is important to look at the context in which the debate concerning the ontological status of DID and the putative mechanism of memory repression has arisen. The flurry of professional books and articles published from the 1980s onward dealing with these causal mechanisms as important conceptual elements of psychoanalytic theory, psychoanalytic therapy, and the recovered memory movement in general, is clear testimony to the *au currant* focus on this particular issue in psychology. However, in examining the epistemic status of psychoanalytic theory, Adolph Grünbaum, Edward Erwin, Frederick Crews et al. have leveled a variety of criticisms at the Freudian theory of repression. As many philosophers of science are well aware, these critics have undermined the epistemological foundations of psychoanalysis, and, at very least, they have thrown into question the evidentiary status of several of the key foundational and clinical claims associated with the theory of psychoanalysis.[4] In doing so, they have also served to bring the more general theories of recovered memory into the realm of critical epistemological discourse. Many of the criticisms that they have raised in the context of the debate concerning psychoanalysis can be extended to the causal models and clinical theories proffered by the recovered memory movement. As one would expect, especially given the (at least superficial) conceptual similarity between the Freudian theory of repression and that of dissociation and their controversial assumption about the nature of memory,[5] there has been a growing perception on the part of some critics that the whole theory of recovered memory is scientifically illegitimate.[6] To be brief, what the defenders of the theory of dissociation must show, if they are to avoid committing both serious epistemological and ethical wrongs, is that dissociation, repression, and memory recovery are actual psychological mechanisms.[7] We will argue that they have not, in fact, done so.

3. Models and Causal Mechanisms

The theoretical accounts of the various dissociative disorders, or more properly, the theoretical *models* of the dissociative disorders, depend heavily on three related causal mechanisms; specifically, dissociation, repression and memory recovery. In fact, these causal mechanisms are definitive elements of those models, and they play an absolutely central role in the nosological categorization of numerous psychological/mental disorders.[8] Consequently, our acceptance of the existence of this cluster of disorders stands or falls based on the evidence for the existence of the causal mechanisms that have been incorporated, *as a matter of definition*, into those models. In other words, our acceptance of the validity of these models depends on the evidence that we have to support claims concerning the instantiation and operation of the causal mechanisms in terms of which they are defined.

In considering questions concerning the existence of particular causal mechanisms we might sensibly adopt the following general, almost trivial, methodological principle; what we might call the 'no mechanism, no model' principle. This principle will be understood as follows:

> (NMP) If we have no good evidence for the existence of a particular causal mechanism, then we have no good evidence for the existence of disorders defined in terms of that causal mechanism.[9]

It might turn out that we have good reasons to believe that some theoretically posited causal mechanisms are not instantiated, and, as a matter of course, given the NMP we should question whether that particular model is, in fact, instantiated. This is simply one aspect of healthy scientific methodology concerning our entitlement to make existential claims about the causes and operation of phenomena. We can, and should, apply the NMP in the case of the dissociative disorders and question whether the mechanisms of dissociation, repression and recovered memory are real mechanisms for which we have good evidence.

As we shall soon see, the conclusion that there is good independent empirical evidence for the existence of the dissociative, repressive, and recovered memory mechanisms is dubious. So there is no good reason to believe that the disorders which are defined in terms of that causal mechanism are real and this follows from the NMP when conjoined with another methodological principle that will be introduced in section 5, the causal modeling principle. With respect to the particular case of DID and the main source of evidence for that disorder (i.e. various diagnostic interviews), these epistemological norms have not been satisfied. Standard diagnostic interviews provide no good reason to believe that instances of this particular disorder are instantiated in our world. In so far as the only other evidence typically offered in support of the existence of DID is merely anecdotal we have no good reason to believe that it is true that anyone has ever suffered from a dissociative identity disorder.

To expand on these themes it will be useful to consider the following analogous case. We ought to reject the concept of DID in the very same way we have rejected the reification of the concept of phlogiston. The problem is not exactly that phlogiston

does not exist, but, rather, that phlogiston just is oxygen with a few theoretical bells and whistles attached. Put simply, Stähl and Priestly's phlogiston does not exist as they construed it. The phenomena that the theory of phlogiston was designed to explain do not operate by the causal mechanisms that Stähl and Priestly proposed. Of course we all know that we have good evidence to support the claim that the modern mechanical model of oxygen developed by Lavoisier is instantiated in our world, and we have good evidence to the effect that the phlogiston model is not instantiated in our world. However, to reject the DID model in this way is not necessarily to say that the persons characterized as having DID are not sick. These patients are, of course, people with profound and real problems. Nevertheless, this simple recognition does not vitiate the claim that the theoretical account of the illness is itself methodologically flawed. Having acknowledged this analogy, it is important to emphasize there is an important disanalogy between the case of phlogiston and DID; viz., phlogiston theory did not play any role in the clinical treatment of patients. In any case, we need to begin looking at the scientific evidence for DID if we are to bring any serious methodological criticisms to bear, but first we need to look at the structure of theories of this sort.

4. The Nature of Mechanisms and Existence Claims

Recently, a considerable body of work has appeared attempting to provide a better understanding of mechanisms and their role in causal explanation. Specifically, the work of Carl Craver, Lindley Darden, and Peter Machamer (MDC) on mechanical models in neuroscience and cognitive psychology provides us with a convenient methodological framework in terms of which psychological mechanisms like DID can be lucidly discussed.[10]

The MDC theory of mechanisms is closely related to the notion of causal explanation defended most notably by Wesley Salmon.[11] The essential significance of specifying the mechanism responsible for a phenomenon is that in doing so we provide an explanation of how that phenomenon occurs, what is generally known as a 'how possible' explanation. In order to do this we must construct and then decompose the mechanical process in question into the entities and activities involved in the regular change that constitutes the phenomenon from its starting state to its completion state. As a result, we find that mechanisms are complex stable arrangements of entities and activities that constitute some regular *functional* process.

Consequently, mechanical explanations of phenomena are a species of functional explanation. However, MDC are clear that not all mechanical models employed in these sorts of causal explanation are complete, and they emphasize that incompleteness can occur in one of two ways. On the one hand, *mechanism schemata* are abstract descriptions of mechanisms the parts of which must be filled in to capture the sub-steps of a causal process, but which are continuous from start to finish. *Mechanism sketches*, on the other hand, are abstract descriptions that are discontinuous from start to finish and so involve one or more gaps that must be

treated as black boxes in the standard sense of the expression. Black box sub-steps are thus regarded as parts of phenomena in need of further mechanical explanation. A phenomenon is completely explained only when we can offer a full mechanical model for that phenomenon.[12] MDC are then careful to point out that mechanisms are discovered or constructed in a piecemeal fashion and so typical mechanical models offered as explanations are incomplete. In other words, most mechanical explanations really take the form of mechanism sketches, and so they are incomplete explanations.[13]

One aspect of this approach to mechanical explanation that is in need of more detailed attention is how one verifies that a given mechanical model, or sub-mechanism that is part of a larger mechanical model, is actually instantiated in some system in the world. Initially, such explanations can only be regarded as 'how possible' explanations, but what we really desire are 'how actual' explanations.[14] What needs to be appended to the MDC account of mechanical/causal explanation is an account of how the associated existential claims are to be confirmed so that competing mechanical models can be differentially compared as more or less correct explanations of a given phenomenon. In what follows we propose some basic conditions that must be satisfied in order to justifiably claim that a 'how possible' explanation is a 'how actual' explanation, and these principles have important implications for the epistemological status of the DID model as noted above.

The DID Mechanical Model and Related Disorders

Having adopted the MDC view of explanation here, we can now turn our attention to the specific details of the DID causal/mechanical model and its epistemic status. In point of fact we can now ask what exactly DID is supposed to be, and once we become clear about the nature of the mechanism(s) which define DID we can then ask what, if anything, is epistemically wrong with the model of this disorder.

Put simply, dissociation is supposed to be a phenomenon involving the burying, or repression, of traumatic memories in such a way that leads to the compartmentalization of those memories, in some cases to the degree that additional distinct personalities emerge. Generally, however, the dissociated memories are introspectively inaccessible to the patient, but these memories do cause overt behavioral symptoms, e.g. spells of radical amnesia. Moreover, these buried memories are supposed to be recoverable through therapeutic intervention typically involving hypnosis. So, dissociation is the causal mechanism by which such behavior modifying traumatic memories are made cognitively inaccessible to the patient, and which can later be made accessible to the patient through hypnotic therapy.

The DSM-IV model of DID indicates that those afflicted with this disorder exhibit the following symptoms:

> A. The presence of two or more distinct identities or personality states (each with its own relatively enduring pattern of perceiving, relating to, and thinking about the environment and self).

> B. At least two of these identities or personality states recurrently take control of the person's behavior.
> C. Inability to recall important personal information that is too extensive to be applied to ordinary forgetfulness.
> D. The disturbance is not due to the direct psychological effects of a substance (e.g., blackouts or chaotic behavior during Alcohol Intoxication) or a general medical condition (e.g., complex partial seizures). Note: In children, the symptoms are not attributable to imaginary playmates or other fantasy play. (DSM-IV, p. 487)

And, more interestingly, we are told that,

> Individuals with Dissociative Identity Disorder frequently report having experienced severe physical and sexual abuse, especially during childhood. Controversy surrounds the accuracy of such reports, because childhood memories may be subject to distortion and *individuals with this disorder tend to be highly hypnotizable and especially vulnerable to suggestive influences.* (DSM-IV, p. 485, our italics)

So, it is clear that the DSM-IV itself raises the possibility that DID is iatrogenic in origin, and we are further told both that,

> Controversy exists concerning the differential diagnosis between Dissociative Identity Disorder and a variety of other mental disorders, including Schizophrenia and other Psychotic Disorders, Bipolar Disorder, With Rapid Cycling, Anxiety Disorders, Somatization Disorders, and Personality Disorders. (DSM-IV, p. 487),

and that,

> Dissociative Identity Disorders must be distinguished from Malingering in situations in which there may be financial gain or forensic gain and from Factitious Disorder in which there may be a pattern of help-seeking behavior. (DSM-IV, p. 487)

So, at least one can detect a healthy air of skepticism and caution in the DSM-IV account of the disorder. The real question then becomes one of the probative force which the experimental evidence has with respect to the existence of the DID phenomenon and, thus, whether it should be in the DSM-IV at all.

We can now explicate the main features of the *theoretical* DID model in terms of the basic concepts of causal/mechanical models introduced in section 4. The DID mechanical model is constituted by a set of fundamental entities, persons (i.e. minds and bodies) and behaviors, and a set of activities including traumatic experience, memory encoding, memory repression, and dissociation. How the phenomenon of DID is supposed to occur is by the operation of instances of the following mechanism sketch:

> (D1) P has a traumatic experience, e_t.
> (D2) P's traumatic experience, e_t, is encoded in memory as $Mem(e_t)$.
> (D3) $Mem(e_t)$ is repressed due to its traumatic character.
> (D4) The production of $Mem(e_t)$ causes P to dissociate and the presence of $Mem(e_t)$ causally sustains this state, thus resulting in the splitting of P into one or more distinct and relatively isolated personalities, Q, R, ...
> (D5) The co-existence of Q, R, ... cause the relevant aberrant behaviors, B.[15]

What we have then is a causal/mechanical account of the behaviors exhibited by those supposed to have DID in terms of the activities, entities and processes referred to in

D1–D5. This is only a mechanism sketch because, at very least, there is no more detailed mechanical account of how any of D2, D3 or D4 occur. In any case, it is clear that the proponents of this model believe that the presence of $Mem(e_t)$ in a patient causally brings about and sustains the set of aberrant behaviors *B* noted in the DSM-IV.

In terms of therapeutic application, the theoretical DID model is assumed to be instantiated and the following overlapping mechanism, what we will call the *therapeutic* mechanism sketch, is taken to be correct:

> (T1) Aberrant behaviors, *B*, are caused by the co-existence of *Q*, *R*, . . . in the patient.
> (T2) the co-existence of *Q*, *R*, . . . is caused by the process described in D1-D4; i.e. by the presence of $Mem(e_t)$.
> (T3) Hypnosis and other related therapies eliminate the repression of memories like $Mem(e_t)$, thereby eliminating the cause of dissociation by effectively integrating the patient's fragmentary personalities *Q*, *R*, . . . into *P*, thus eliminating aberrant behaviors, *B*.

The therapeutic model opens the way for the elimination of the aberrant behaviors through integration of the dissociated mind via the 'un-repressing' of $Mem(e_t)$. As in the case of the theoretical DID model, this is a mechanism sketch because there is no seriously accepted deeper mechanical account of how dissociation, hypnosis therapy, and other related therapies work.

Two Methods of Generating Mechanical Models

Mechanical models of physical processes, phenomena, can be generated by two distinct methods.[16] The first method (method-l) is to build a particular mechanical model of a phenomenon from the laws of a more basic theory by using analytic methods. Through the use of this method we arrive at what we might call a mechanical model of a physical process or a 'candidate phenomenon'.[17] Such hypothetical processes or entities are deductively inferred merely by appeal to theory and the tools of deductive logic and mathematics. The second, perhaps more dangerous, method (method-2) is to generate candidate models by analyzing experimental data without the aid of any real prior theoretical considerations, except, perhaps, some background assumptions. One simply looks for regularities in the data and extracts some general causal/mechanical characteristics of the phenomenon directly from the data via statistical analysis or other related methods. Typically the inferences made in this procedure are inductive, and, hence, are less secure than the deductive inferences employed in the first method of mechanical model construction. But, candidate models, arrived at either way, cannot be legitimately said to be part of the actual causal structure of our world without passing some well-established, reliable, empirical tests aimed at confirming the actual instantiation of the model in question.

In effect, if we adopt the general motivation behind the causal/mechanical view of explanation, this is simply the confirming of the corresponding existential claim

associated with the particular mechanical model. But, this appears to pose a problem for the case at hand because DID is not accompanied by the kind of predictive/explanatory theory which is required for the construction of normal testing procedures. In any case, with respect to DID, it seems that the second method of phenomena generation mentioned above has been employed, i.e. method-2. As we suggested, it is not clear that there really is anything like a fundamental theory behind the phenomenon of DID in the manner in which, say quantum mechanics explains the quantum Hall effect or the manner in which singularities are explained by general relativity. One might be tempted to suggest that the theory in question just is a modified Freudian psychoanalytic theory, but, as noted at the outset, due to the criticisms which have been leveled at the theory of psychoanalysis, it would lend little support to the claims concerning DID to appeal to psychoanalytic theories, Freudian or otherwise. Moreover, this would be historically inaccurate, as it is a matter of historical record that Freud rejected the view that there really were any dissociative phenomena or *bona fide* multiple personalities. Rather, as Breuer & Freud's *Studies on Hysteria* demonstrate, Freud took the view that such phenomena were most likely cases of transference involving patients with hysteria.[18]

The current situation with respect to the DID phenomenon is something more like the case of the photoelectric effect discovered by Hertz in 1877. The causal mechanism we call the photoelectric effect occurs when an ultraviolet light is shined on certain metals thus generating a steady current. But, at the time of its discovery, there really was no adequate, 'deep', theoretical explanation of the photoelectric effect. All that was noticed was that there was a stable connection between shining light on the substance and the generation of a current. The explanation of the photoelectric effect was provided years later by Einstein when he discovered the photoelectric equation, $E = h\nu - \Phi$ (where 'E' is energy, 'h' is Planck's constant, 'ν' is the frequency, and 'Φ' is the work function), which related the photoelectric effect to electrodynamics. The relevant point here is that DID, like the photoelectric effect in 1877, was not derived analytically from some high level theory. Rather, it is alleged to be a mechanism that has been discovered by looking at specific cases, i.e. by looking at data. So, the relevant, closely related, epistemological questions that we must consider are: (1) whether or not the inference which has been made from the data to the mechanical model passes epistemological muster, and (2) has the existential claim concerning the actual instantiation of the DID model been sufficiently confirmed? These questions are especially important in cases of method-2 model generation, as there is no deeper theory from which the model is derived and from which the model can parasitically accrue confirmation.

5. Differential Diagnosis, Causal Modeling and Empirical Claims

Before proceeding, it is important to acknowledge that there has been some debate about whether or not claims made in the context of psychoanalysis and related fields ought to be held to the epistemological standards associated with the natural sciences.[19] Clearly, however, we can at least suggest that claims for the instantiation of

any particular mechanical model can only be regarded as confirmed when all reasonable alternative accounts of the data have been eliminated as realistic possibilities. It is simply not epistemically acceptable in any scientific discipline to infer that the existential claim concerning a particular model is true from data that are compatible with several such models. In any case of causal modeling one must differentially eliminate all reasonable competing models of the data in order to establish some model as the correct representation of a given phenomenon.[20] This may provide a way to avoid the problem posed above concerning the inability to construct theoretically grounded tests for the existence of a phenomenon.

If, due to our background knowledge, we believe that a particular set of data, d, could possibly be evidence for three extant incompatible phenomenological models, M_1, M_2 and M_3, we can only legitimately assert the existential claim that d is an instance of process M_1 (or a sub-process of M_1), by falsifying the existential claims that d is an instance of process M_2 and that d is an instance of process M_3. What this highly artificial example suggests is that in order to confirm the existential claim concerning a given model and a given data set, one must falsify, or at least significantly disconfirm, all of the existential claims concerning all plausible alternative models of the data in question.[21] We will refer to this methodological principle as the causal modeling principle, and it will be (initially) understood as follows, where 'd' is an appropriately formatted body of relevant data, '$\mathbf{E}_k$' is a set of claims of the form '$(\exists x)\ M_i x$', and '$\mathbf{M}_n$' is the set of all relevant models M_i.

> (CMP) Given a body of data d and existential claims $\mathbf{E}_k$ associated with models $\mathbf{M}_n$, where $n > 1$,[22] we are entitled to assert that the causal mechanism(s) definitive of M_i, where $M_i \in \mathbf{M}_n$, are instantiated in d, if and only if, it is not the case that there is a plausible model M_j, where $M_j \in \mathbf{M}_n$ and $i \neq j$, for which the associated existential claim has not been ruled out relative to d, and, the existential claim associated with M_i is itself plausible.[23,24]

This is neither an exceptionally strong nor controversial epistemological criterion, and it seems reasonable that any acceptable existential claim ought to satisfy CMP. This is just part and parcel of good statistical reasoning in the experimental sciences.[25] Controlling for alternative and confounding causes by ruling out relevant alternative causal models is a ubiquitous scientific practice. If d turns out, unambiguously, to be evidence for M_1, because we have ruled out all other serious alternative empirical claims as being very likely, then we can legitimately assert with some degree of confidence that mechanisms like those posited in model M_1 exist.[26]

Respecting the probabilistic nature of such methods we can reformulate CMP as follows:

> (CMP′) Given a body of data d and existential claims $\mathbf{E}k$ associated with models $\mathbf{M}_n$, where $n > 1$, we are entitled to assert that the causal mechanism(s) definitive of M_i, where $M_i \in \mathbf{M}_n$, are likely instantiated in d, if and only if, it is not the case that there is a plausible model M_j, where $M_j \in \mathbf{M}_n$ and $i \neq j$, for which the associated existential claim has not been shown to be unlikely relative to d,[27]and, M_i is itself plausible.

So, if there are no clear cases at all where the data supports a particular model unambiguously, then we ought to be very suspicious about the existential claim concerning that model. Furthermore, if this is true, then the data the model attempts to unify as being of a piece may, in fact, turn out to be the result of a heterogeneous set of mechanisms. In other words, it may not be the case that any of the individual members of the partition of plausible theories $\mathbf{M}_n$ accounts for every element of the data used in constructing a particular phenomenon. In effect, in such cases it may turn out that the data is not evidence for any one specific phenomenon or model.[28]

6. Test Batteries and Diagnostic Evidence for DID

We must now look directly at the evidence offered up in support for the existential claim associated with the DID model. Obviously, due to lack of space, an exhaustive consideration of all of the evidence relevant to DID cannot be made, and so we will examine the major source(s) of evidence offered up to support the existential claim associated with DID. This evidence is alleged to support, or at least aid in, the kind of differential confirmation of the existence of DID in accord with the causal modeling principle introduced above.[29] The Minnesota Multiphasic Personality Inventory (MMPI and MMPI-2), the Structured Clinical interview for Dissociative Disorders (SCID-D) and the Dissociative Experiences Scale (DES) are all supposed to be diagnostic screening tools which psychologists can use to help establish that patients have certain disorders.[30] In what follows we will look closely at the MMPI and the MMPI–2, and at the SCID–D and the DES.[31] We have chosen to focus on these particular devices as they have been employed frequently in test batteries intended to help in the diagnosis of DID.[32] The typical approaches to diagnosis of DID then take the form of batteries of such tests that, in conjunction with the subjective clinical experience of the psychologist, are supposed to offer the best available evidence for the accurate diagnosis of DID. This hybrid strategy that incorporates both the subjective judgment of the psychologist and various diagnostic instruments is, without question, the most common method of diagnosis employed in orthodox contemporary psychology.[33]

The profiles generated by administering these instruments are supposed to offer good, but not necessarily irrefutable, evidence that a particular patient has a particular disorder. The profile of a patient is compared against the profiles of typical disorder sufferers. Matching, within reasonable error, one of the abnormality profiles is supposed to be confirming evidence that the patient has the disorder in question. As different disorders are supposed to be characterized by different profiles, such surveys provide a means to help to make differential diagnoses with respect to patients, provided that they are both judicious and aware of the limitations of the diagnostic instruments.[34] In terms of the methodological principles developed earlier, matching a particular abnormality profile is then supposed to be some objective evidence for the empirical claim that the patient instantiates the model of that disorder, but the probative force of the evidence from these sources depends essentially on the methodology employed in the creation of such tools.

The various instruments employed in the hybrid approach can be constructed on the basis of either of two distinct methodologies, the empirical keying approach and the logical keying approach.[35] The empirical keying approach involves empirically determining some criteria or criterion which differentiates those afflicted with some disorder from those not so afflicted. The logical keying approach involves the *a priori*, subjective, selection of some criteria or criterion which is supposed to allow for such differentiation. The MMPI and MMPI-2 were developed using the empirical keying approach, and the SCID-D and DES were developed using the logical keying approach. As we shall see, both test construction methodologies are characterized by serious epistemological problems that ultimately violate the principles of causal modeling introduced in 5 and so the instruments developed in these ways fail to provide evidence sufficient for reasonable differential diagnosis.[36]

The MMPI, MMPI-2 and the Empirical Keying Approach

Instruments based on the empirical keying approach are typically constructed in a very peculiar way. Specifically, they do not involve much, if any, theory. Rather, as we shall see, they are generated empirically. Being aware of this, it is interesting to look at how MMPI and MMPI-2 are structured. The MMPI is a self-report questionnaire composed of several scales designed to indicate the presence and magnitude of certain psychological traits relevant to common psychological disorders. The original scales in the MMPI include (l) hypochondriasis (HS), (2) depression (D), (3) hysteria (HY), (4) psychopathic deviate (PD), (5) masculinity–femininity (MF), (6) paranoia (PA), (7) psychasthenia (PT) (8) schizophrenia (SC), (9) hypomania (MA), and (0) social introversion (SI). These individual trait scales are supplemented by the lie scale (L), the correction for defensiveness (K), and the infrequency scale (F), which are individually and jointly designed to control for various kinds of faking. The individual trait scales, some of which involve correction factors, sub-scales and weightings, are supposed to reflect normal and pathological measures of each such trait.[37] So, profiles that typify certain well-defined disorders have been generated based on these scales in reference to the profiles of psychologically normal samples of the general population. The typical MMPI profiles for DID, borderline personality disorder, and Briquet's syndrome, are reported and graphically presented in North, et al., 1993, and they are all startlingly similar, if not simply indistinguishable.

For the purposes of this discussion this is a sufficiently detailed conception of the MMPI. What is important is that this tool is supposed to provide an evidential aid for differential diagnosis, but such an instrument is only reliable if the manner in which the tool was developed is methodologically acceptable.[38] As we shall see, due to the manner in which these sorts of psychological testing devices have been constructed, it is not clear that they provide any basis for confirming the existential claims associated with the models of psychological disorders for which they are supposed to be relevant.[39] Diagnostic tools based on the empirical keying approach have been explicitly developed by the application of statistical methods to empirical data, and, hence, resemble an instance of method-2 phenomena generation

(see section 4). As such, the process by which these tests were constructed suffers from some of the inductive problems that plague method-2 procedures. We will find that the MMPI has little or no probative force with respect to the existential claim associated with DID. More specifically, the tests do not satisfy the CMP′ in the case of DID. It is clear that such interviews do not provide anything like a way to make reliable differential diagnoses of DID (or even contribute to the evidence in favor of that diagnosis), and, as a result, they do not provide any evidence that the existential claim about DID is true.

The MMPI and MMPI-2 scales were designed by selecting a set of true/false questions that were, subsequently, given both to patients who were supposed to clearly exhibit pathology with respect to the trait in question and to control groups composed of normal people presumed to exhibit normal behavior with respect to that trait. The resultant data were analyzed in order to find questions that were answered by those with the disorders, the criterion group, in characteristically different ways from the normal people in the control groups. Questions which did not reliably distinguish the group with the disorder from the normal group in terms of the trait for which that scale was constructed were discarded. The significance of the difference in measured by applying the following equation:

$$z = p_1 - p_2/\sqrt{pq}[(1/n_1) + (1/n_2)]$$

Where 'p' is the proportion of true responses in the total group, 'p_1' is the proportion of true responses in the first sample, 'p_2' is the proportion of responses in the second sample, 'q' is $1 - p$, 'n_1,' is the number of persons in the first sample, and 'n_2' is the number of persons in the second sample. Items were retained if their z value was greater than $+2$. As Greene explains,

> In selecting items for a specific scale (e.g. Hypochondriasis), they [Hathaway and McKinley] used an empirical approach. The items had to be answered differentially by the criterion group (e.g., hypochondriacal patients) as compared with normal groups. Since their approach was strictly empirical and no theoretical rationale was posited as the basis for accepting or rejecting items on a specific scale, it is not always possible to discern why a particular item distinguishes the criterion group from the normal group. Rather, items were selected solely because the criterion group answered them differently than other groups. (Greene, 1991, p. 5)

Subsequently, scores on the set of questions for each scale were subjected to statistical analysis, weighting, and other corrections, and scores indicative of the presence of pathological and normal behaviors were computed in accord with the equation above. This type of procedure was used to construct the ten trait scales on the MMPI.[40]

So, the question sets were tested empirically against what were taken to be well-established reference groups that were properly partitioned into 'normal' and 'abnormal' classes. Answering the questions like those in the criterion group, who were supposed to have the pathological version of the trait, is supposed to be good evidence that the person being tested has some disorder that is characterized by

that trait. But, this presumes that the members of the criterion group really did have a disorder characterized by the trait in question. Green tells us that,

> The first step in developing Scale 1 was to select an appropriate criterion group. Using diagnostic classification as the basis for the criterion group selection was logical since McKinley and Hathaway's intent was to develop an inventory to aid in differential diagnosis. They defined hypochondriasis as an abnormal neurotic concern over bodily health, excluding the symptomatic occupancy of hypochondriacal features in psychotic individuals. Using this definition, they selected 50 cases of pure, uncomplicated hypochondriasis as their criterion group. (Greene, 1991, pp. 5–6)

A similar procedure was used with respect to all of the scales. But, surely we cannot simply assume that the patients in the 'abnormal' partition of the reference class just have the disorder and the pathological trait in question, even if they are simple cases. If we cannot directly observe the disorders, then we must infer that these people are so afflicted from some data.[41] Good evidence of the sort necessary to assure that the criterion group all really had the pathological trait cannot be provided by any interview generated in the manner that has just been outlined. That would just cause the problem to arise all over again in terms of that further diagnostic test. Disorders may be defined in terms of the traits that are used to construct the MMPI scales, but unless we can reliably assert that the criterion group instantiates that mechanical model, then it is not clear what the particular trait scale is a measure of. With respect to the general interview, we find a similar problem arises.

Profiles associated with specific disorders are constructed by the whole test to a normal group and to what are taken to be clear, simple, cases of patients who instantiate the disorder in question. The profile which results from this procedure is supposed to provide a means of differential diagnosis, and, hence, tests like the MMPI allege to provide evidence for the empirical claims concerning various psychological disorders by indicating the presence of certain abnormal traits associated with these disorders. The interviews are supposed to be tools for differentially detecting disorders by detecting these relevant traits, but the tests *presuppose* that the disorders are already instantiated and that all the members in the 'abnormal' partition of the reference class used in constructing the profile for a disorder, in fact, instantiate the disorder in question. It may turn out to be the case that the subjects in the 'abnormal' partition of the original reference class are not homogeneously characterized by any one pathological disorder, unless there is some other source of objective and unambiguous evidence to establish that the subjects in the 'abnormal' partition do, in fact, all have the disorder in question.

In the case of some of these psychological disorders, like schizophrenia, physiological evidence can serve this vital function as such evidence is publicly accessible and repeatable. But, what about the cases where there is no reliable physiological indicator for an alleged disorder? In such cases the tests we have been considering do not seem to be tests for the presence of such disorders at all. Rather, they are tests aimed at establishing that subjects are sufficiently like or unlike the subjects in the 'abnormal' partition of the reference class, and so the legitimacy of these

tests depends crucially on the assumption that the member(s) of the 'abnormal' partition do instantiate the disorder in question. This must be independently confirmed through legitimate, publicly accessible and repeatable, testing procedures. If the members of the 'abnormal' partition of the reference class do not all instantiate the disorder in question, then the interview will not be a legitimate test for that disorder.

With respect to DID the situation is slightly different because there is no specific trait scale designed for dissociation, and the MMPI was constructed long before DID entered the arena of legitimate psychological disorders.[42] However, we will find that the same problem noted above arises in the case of DID, albeit in a slightly different way. The fact that there is no dissociation scale on the MMPI does not really pose a problem as the profiles for all disorders are specified in terms of all of the trait scales, and not just in virtue of any one of them. This is simply a result of the fact that most psychological disorders are polysymptomatic. The specific trait scales were selected because these traits are supposed to be the backbone of psychological pathology in general. They are the basic traits with which abnormal psychology is primarily concerned. Standard profiles are given in terms of all of the scales because many disorders share significant overlap in terms of the symptoms that characterize them, and as we have seen, disorders are defined in terms of these mechanisms, because such mechanisms are built into the models of these disorders.

We indicated in earlier sections that in the case of the construction of the particular trait scales, we find that standard profiles for specific disorders are generated by giving the interview to a group of subjects who are supposed to be clearly partitioned into 'normals' and 'abnormals'. So, when a subject has a profile sufficiently similar to the standardized 'abnormal' profile, this is supposed to be good evidence that he or she instantiates the model of that disorder. But, if we have no independent evidence to confirm that the criterion group in question actually has the disorder in question, then we have no real reason to suspect that the test reliably indicates the presence of the phenomenon. Such testing procedures are *only* reliable if the members of the criterion group are afflicted with DID, but that they are must be established by some means other than through diagnostic instruments which presuppose that the phenomenon does occur in our world.

The DES, SCID-D and the Logical Keying Approach

The DES and SCID-D, unlike the MMPI and MMPI-2, were specifically created as aids for the diagnosis of dissociative disorders, including DID. As noted earlier, they were also developed by means of a very different, but more traditional, method; the logical keying approach. The DES takes the form of a 28 item self-report instrument scored on a scale of 0–100 and it was developed by constructing questions that were supposed to be intuitively indicative of the dissociative disorders.[43] For example, question 3 states that,

> Some people have the experience of finding themselves in a place and having no idea how they got there. Mark the line to show what percentage of the time this applies to you. (Bernstein & Putnam, 1986, p. 733)

Subjects were asked to determine the degree to which descriptions of certain experiences like this applied to them by marking a slashed line on a continuous graphic interval marked 0–100% for each question, and the DES score for the patient was made for each question by measuring the distance of the slash from left to right for each question to the nearest 5 mm. The total DES score for the patient was then the average of the scores on the individual questions. The questions were generated by appeal to the clinical experience and understanding of DID by those practitioners who constructed this particular questionnaire; i.e. Bernstein and Putnam.

Subsequently, they gave the questionnaire to patients with various disorders and normals, thus generating DES profiles of those disorders. For example, the median score for those afflicted with DID was measured to be 57.06, the highest median score for any group. In employing this *a priori* approach to the construction of a diagnostic instrument, they assumed that matching a particular response profile, for example scoring say near to 60, really is at least some evidence that the patient has DID. However, it has been subsequently noted that while higher DES scores correlate with suffering from DID, in that it is more likely that those who score high on the DES have DID, the DES is not, when taken alone, a fully reliable diagnostic instrument as relatively few of those who scored high on the DES scale actually had DID.[44] However, attempts have been made to demonstrate the validity of the DES as a diagnostic aid by administering it to those alleged to suffer from DID and by reference to other tests, specifically the SCID-D.[45] In any case, even according to the proponents of the DES, the DES is more properly regarded only as a screening device to aid in the diagnosis of dissociative disorders and to determine the severity of dissociative experiences in the context of a more sophisticated test battery.

The evidential support that the DES might provide, as in the case of the MMPI and MMPI-2, is only good if the methodology on which that test has been constructed and verified is rationally acceptable. Given the brief characterization just offered of the methods used to construct and validate the DES, it should be apparent that there are serious epistemological problem with the DES *qua* its role in psychodiagnostics. First, as Graham (1990) and others have noted, the chief reason behind the introduction of the empirical keying approach in psychology was the perception that the logical keying approach is utterly subjective. The profiles supposed to be indicative of disorders and the questions designed to detect such disorders have no immediate validity in the sense that, other things equal, they provide objective reasons to believe that matching a certain profile really is evidence that a patient suffers from that disorder.[46] Second, the attempt to validate the DES by administering it to patients that are supposed to be afflicted with DID,[47] in order to determine how well it detects DID, suffers from a flaw similar to that which characterizes the MMPI and MMPI-2. Specifically, such a validating procedure presupposes that the patients against which the DES was tested really are, in fact, afflicted by DID. Absent some independent and objective reasons to accept this, it is nothing more than a test that shows that a candidate answers the proposed questions much like someone assumed to have DID. Thus, such a validating procedure will be able to ground the existential claim that the patient really has DID only if the patients

in the validating procedure actually had DID. Finally, the attempts to validate the DES by testing it against the SCID-D, are reliable only if the SCID-D is itself a reliable instrument for the detection of DID, and we shall see that it is not evidentially reliable in this way.[48] Given such problems we cannot claim that the DES provides adequate reasons to assert that anyone really has DID as characterized in the DSM-IV.

The SCID-D, developed by Steinberg, Rounsaville and Cicchetti (1990), was also constructed on the basis of the logical keying approach and it was specifically designed to aid in the diagnosis of dissociative disorders, but it is a rather different kind of instrument. Unlike the DES, the SCID-D is not a self-report questionnaire, and the SCID-D takes the form of a structured psychiatric interview. The SCID-D is made up of 200 questions, which are aimed at revealing information about five symptoms: amnesia, depersonalization, derealization, identity confusion, and identity alteration. The questions are structured and begin with very general questions concerning these symptoms, followed by more detailed follow-up questions that are supposed to elicit more specific information from the patient concerning the degree and frequency of certain symptomatic experiences.[49] A manual is available for scoring the SCID-D, and scoring is supposed to be made by a clinician trained in the use of the instrument. The clinician rates the patient in terms of his/her responses, especially concerning severity, and then is supposed to use the manual to generate an overall compiled score. The questions were generated via the logical keying approach and so were generated on the basis of the developer's intuitions that certain answers are indicative of certain disorders. As in the case of the DES, the SCID-D was validated by administering the interview to a variety of patients assumed to have various disorders and a number of control subjects.

As should be obvious, problems similar to those that afflict the MMPI, MMPI-2 and DES arise here. First, as the SCID-D was developed on the basis of the logical keying approach, it suffers inherently from the first and most general flaw noted with respect to the DES. Specifically, there is no objective reason to believe that the questions chosen and the profiles constructed on the basis of those questions are really indicative of the relevant disorders. Moreover, in the case of the SCID-D we have the added complication that the scores reported are the result of the subjective evaluation of the clinician in question and there is no reason to suspect that such score reports really reflect the actual severity of symptoms of the patient. Finally, as in the cases of the MMPI, MMPI-2 and DES, the method used to attempt to validate the SCID-D begs the obvious question concerning whether or not the test subjects employed in the validating process really suffered from the disorders they were assumed to have.

To summarize the results of this section we think that it suffices to point out that the kinds of instruments employed in orthodox psychodiagnostic test batteries as aids to differential diagnosis of psychological disorders are not even remotely reliable, and so provide little or no objective evidence for the existential claims that patients actually have the disorders that the instruments are supposed to help detect. This leaves us with the obvious and pressing question concerning what other

possible sources of evidence might be available to justify the existential claim concerning DID.

Physiological Evidence and the Judgment of Psychologists

One crucial problem that DID faces with respect to such diagnostic instruments over and above those it shares with other psychological disorders is that there does not seem to be any independent psychological or physiological evidence for the empirical claim associated with DID that might ground the legitimacy of the MMPI, MMPI-2, DES, SCID-D, etc. with respect to DID. This problem is further exacerbated because, as we have now seen, the symptoms which characterize DID are similar, if not identical, to those which characterize numerous other disorders, malingering, and iatrogensis.[50] This is made clear by the DSM-IV model of DID; the only symptom which is uniquely associated with DID is patient reports of fragmented personalities. Certainly this evidence cannot serve as a reliable differential indicator of DID unless suggestion, faking, and malingering are ruled out in the sense described in earlier sections. The CMP′, in fact requires, that we do so. We must also, in a similar manner, rule out all alternative existential claims associated with the other competing causal models (many of which are quite plausible because, unlike DID, we possess a much deeper understanding of the underlying physical mechanisms that they are associated with) if we are to be confident that a particular causal mechanism is, with some likelihood, at work in the phenomena in question.[51]

Consequently, if there are no legitimate and objective ways to differentially establish the existential claim concerning DID other than by appeal to diagnostic instruments and physiological indicators, then the existential claim concerning DID cannot satisfy the CMP′.[52] Both sources of evidence are currently incapable of providing evidence that would satisfy the CMP′, and the mere subjective judgments of psychological practitioners alone cannot do so either. If this is, in fact, the case, then there really is no epistemological basis for the claim that the DID model is, or ever has been, instantiated in our world. This is because if the accounts of DID do not satisfy the CMP′, then by NMP we can conclude that there is no good reason to believe that the DID model is instantiated. Such a psychological disorder might, in point of fact, exist, but, at least as things stand at this point in our discussion, we have no good reason to believe that it does. Things are, however, rather worse for the proponents of DID as we shall see in the following section. In point of fact, there is some reason to believe that the account of memory assumed in the theoretical and therapeutic models of DID is false and that the memories that are referred to in those theories are really iatrogenic in origin. If this is true in addition to the points made above, then not only is there no reason to believe that DID is instantiated but also there is some reason to believe that it is *not* instantiated in the relevant patients.

7. Memory, Suggestion and Guided Imagination

It is not hard to see that the DSM-IV DID model presupposes a rather commonsense, folk psychological, account of the nature of memory wherein memories are taken to

be accurate stored traces of past experiences that can either be accessible, if they are non-traumatic, or inaccessible, if they are traumatic and repressed.[53] On this view, barring any unusual causal interference, a memory that is ordinarily recalled or one that is recalled by 'un-repressing' that memory, is taken to be accurate; that is to say, unless there is some special reason to doubt the verity of a memory it should be taken to be true.[54] Finally, as should be clear from the theoretical model of DID, such memories, including those that are repressed, are taken to have significant causal roles in the production of overt behaviors. As our previous discussion indicates, the DID models are deeply dependent on the accuracy of this conception of memory. However, there is a significant and growing body of literature in support of the claim that this model of memory is utterly mistaken.[55] Here only a brief mention of some of the evidence against this account of the nature of memory will be needed to show that there is significant disconfirming evidence for some of the crucial theoretical and therapeutic (causal) claims constitutive of DID.

The work on memory relevant to this issue began with studies focusing on the veracity of eyewitness testimony. These studies were specifically designed to show that appealing to the memories of eyewitnesses is an unreliable source of evidence in the context of the court system.[56] For example, in a compelling study Loftus and Hoffman report that people are susceptible to what is called the 'misinformation effect'. This effect is exhibited when a memory of an event becomes polluted or is transformed into another memory as the result of exposure to misinformation provided by other witnesses to an event, interrogators, the media, and various other sources of information.[57] In such cases eyewitnesses can become thoroughly convinced of the truth of apparent memories that are demonstrably false. In fact, Loftus and Hoffman see what they refer to as a 'Watsonian' future for the misinformation effect.[58] Consider the following challenge that they issue:

> Give us a dozen healthy memories, well-formed, and our own specified world to handle them in. And we'll guarantee to take any one at random and train it to become any type of memory that we might select—hammer, screwdriver, wrench, stop sign, yield sign, Indian Chief—regardless of its origin or the brain that holds it. (Loftus & Hoffman, 1989, p. 103)

All bravado aside, Loftus (1993) demonstrates that much more evidence concerning the ubiquity and depth of the misinformation effect in memory recall has been gathered, but even more interesting for our purposes here are the related studies that have been done on false memory implantation as a result of suggestion and imagination.

Loftus (1993) reports the results of a study done by Loftus and one of her colleagues concerning the implantation of false memories of childhood events. Loftus and her colleague took a fourteen year old subject and had several of the subject's family members ask the subject to recall a time at the age of five when he was lost in a shopping mall, an event that had, in fact, never taken place. At first the subject could not remember the event, but after persistent questioning the subject 'recalled' the

event. The subject was then asked to try to remember details of the event after two weeks of suggestive questioning coupled with activity of the subject aimed at trying to 'remember' the event. In this case the subject reported a false memory of being lost in a shopping mall with great confidence and in surprising detail.[59] As a result of this experiment and other related findings we can conclude that it is clearly possible that false memories of traumatic events can be recalled after suggestive questioning and guided imagining. This is especially relevant to the case of DID when we recall that we are told in the DSM-IV that:

> Individuals with Dissociative Identity Disorder frequently report having experienced severe physical and sexual abuse, especially during childhood. Controversy surrounds the accuracy of such reports, because childhood memories may be subject to distortion and *individuals with this disorder tend to be highly hypnotizable and especially vulnerable to suggestive influences.* (DSM-IV, p. 485, our italics)

So, we have what is clearly some disconfirming evidence against the theory of memory assumed by the proponents of DID, and it indicates that repressed memories may well not be authentic and may not be memories at all. To some degree, these findings implicate the general view that memory involves the storage of accurate representations of events, and supports the view that what we call memory may be more a matter of our constructing events by the use of complex psychological mechanisms. Less generally and less controversially, these studies *clearly* show that memory is highly malleable and subject to suggestion. If this is true, then we have some good reasons to suspect that the theory of memory on which DID is based is mistaken and that DID is probably not an actual disorder. Given these empirical results about suggestibility and imagination, the practices of therapists who treat and diagnose DID patients look to be highly suspicious, and when we look at the particular details of their therapeutic methods and at the nature of suggestion it becomes even more clear that DID might simply be iatrogenic in origin.

Some factors that are known to influence the suggestibility of false 'memories' are the strength of the memory, the authority of the source of information, and the delay before the presentation of post event information.[60] Why might we believe that DID patients are really the victims of suggestion? First, we have reason to suspect this because the childhood memories of the patients are naturally quite old and very weak. Second, the information sources, the therapists, are in a position of authority relative to the patient, and this sort of problem is well-known to Freudians and can result in relations of dependence and transference between patient and therapist. Finally, there is typically a very long delay between the actual event and the presentation of post event information, namely the information inherent in the therapist's (often leading) questions. All of these factors are often present in the discourse between DID patients and therapists, thus indicating that there is a serious potential for memory suggestion and the ensuing construction of detailed false memories in the treatment of DID patients.

The other practice characteristic of therapists who are proponents of DID, asking their patients to imagine that they have been the subjects of traumatic childhood sexual abuse, is done because this guided imagining is supposed to help trigger the recall of heretofore repressed memories.[61] However, as the results of Garry, Manning, Loftus and Sherman (1996) demonstrate, this technique is often bound to cause the confident construction of false repressed memories by patients. As Garry et al. report, if a subject counterfactually imagines an event to have happened, then the confidence that the subject will have in reporting the actual occurrence of the event in question is significantly heightened.[62] Subsequent to imagining the event to have occurred, subjects become much more likely to believe that fictitious events did in fact occur and that they are in fact correctly remembering the event. What Garry et al. have exhibited in this and related studies is that guided imagining is another mechanism capable of generating confident beliefs about events that never actually occurred, and this technique is a typical component of the therapies employed in clinical contexts by the proponents of DID.

Together, these various studies suggest that many of the 'memories' that are supposedly dredged up concerning the past of DID patients are quite plausibly the result of iatrogenic suggestion and that the practice of recovering repressed memories is fundamentally misguided as it is based on a highly dubious account of how memory works.[63] In any case, these studies further undermine the evidential status of the existential claim associated with DID as not only does the methodological practice of the proponents of DID violate the CMP′ in the sense that there are viable alternative explanations of the behaviors of the patients that have not been eliminated and for which there is significant physiological evidence, but also in the sense that the DID explanation *cannot even plausibly be taken to be on an evidential par with those alternative explanations*.

8. The Therapeutic Mechanism and the Ethics of Treatment

These critical points concerning the DID mechanical model are of great methodological interest in the context of the epistemological evaluation of psychology, but they also have much deeper ethical implications. Recall that the therapeutic mechanism introduced as a means of curing patients who allegedly suffer from DID assumes the veracity of the DID mechanical model. The process described by T1–T3 makes sense only if D1–D5 is correct. However, given that there is little confirming evidence and some compelling disconfirming evidence that the mechanisms mentioned in D1–D5 are real, there is little or no reason to believe that the therapeutic mechanisms mentioned in T1–T3 will have any efficacy in eliminating the aberrant behaviors exhibited by the patients in question.

What is *ethically* disturbing in the case of DID is that the treatment of patients diagnosed with DID is based upon the DID model. If DID is really fictitious in the sense that we saw that phlogiston is fictitious, then the patients in question may not be receiving the treatment that they sorely need. Continuing to treat patients based on the assumption that they have DID may constitute a serious violation of the

therapeutic obligation that doctors have with respect to their patients. It is for this reason that the question of the existence of DID is important, and, recalling the discussion of the phlogiston model in 3 and the NMP, it should be clear at this point why this is so. The acceptance of DID by clinicians (as understood in D1–D5), given the criticisms raised here with respect to orthodox test batteries and the inadequacy of subjectivity of clinician judgment to ground reliable differential diagnosis, entails the illegitimate assumption that DID is a post-traumatic dissociative phenomenon that results in the creation of actual fragmented personalities rather than some other phenomenon. This is not a trivial matter either as establishing the actual etiology of the symptoms exhibited by those who appear to have DID will have important implications concerning the vastly different therapeutic and preventative measures appropriate given that particular causal model.[64]

The failure of the defenders of DID to subject the existential claim associated with the mechanical model of that disorder to appropriate tests and to respond adequately to apparently disconfirming evidence constitutes a serious violation of both epistemic and moral obligations. The therapeutic obligation states, roughly, that, medical practitioners must treat their patients in a way that yields the best chance of recovery.[65] Presumably this requires of medical practitioners that they verify to a reasonable degree the claim that not only is the particular patient suffering from a particular disorder, but also that it is a real and distinct disorder. To fail to do so is, in violation of the therapeutic obligation, to treat a patient with a therapy that the practitioner cannot *possibly* have good reason to believe yields the best chance of recovery as that practitioner has no good reason to believe the disorder is even real, let alone that the particular therapy being employed is efficacious. Notice that this behavior need not necessarily be an intentional failure on the part of the medical practitioner in order to constitute a violation of the therapeutic obligation. More often than not, we suspect that such behavior is the result of simple negligence, but it is still behavior that violates the therapeutic obligation nonetheless and so acting in this way is a serious moral wrong. This is a clear case where violating an epistemological imperative constitutes an ethical wrong whether it is done by engaging in epistemic negligence, by self-deception, or by engaging in a more sinister form of intentional failure to satisfy good methodological practice.

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Notes

[1] The dissociative disorders include dissociative amnesia, dissociative fugue, dissociative identity disorder, depersonalization disorder, and dissociative disorder not otherwise specified. Our criticisms concerning the evidential status of DID should be extendable to any disorder defined in terms of dissociation or repression.

[2] For more or less recent professional consideration of clinical treatments for DID/MPD see Braun (1993), Fine (1993), Kluft (1983, 1993a, b), Loewenstein (1993), Brassfield (1983), and Horevitz (1983).

[3] It is interesting to note that DID is the new name for multiple personality disorder (MPD) introduced in DSM-IV. The controversy over the status of MPD as specified in DSM-III led to some minor emendations in DSM-IV, including renaming. But, for the purposes of this paper, they are indistinguishable.

[4] See Grünbaum (1984, 1993), Erwin (1996) and Crews (1995).

[5] See Ross (1997) (especially p. 31) and Hacking (1995) for historical perspectives on Freud's attitude toward dissociation and how it both differs from and is similar to repression. We do not intend to suggest here that dissociation is Freudian in origin, only that it bears some resemblance to Freudian repression. Given this resemblance, the current, highly critical, attitude towards Freudian psychology also suggests that the practices and theoretical assumptions of the proponents of dissociation should be scrutinized along similar lines.

[6] Elizabeth Loftus is perhaps the most well known critic of the mechanisms of memory repression and memory recovery. Loftus has labored to show that people do not store complete records of events in their minds to which they later have access. Loftus' work on memory and suggestion tends to support the view that many of the 'repressed' or 'dissociated' memories are iatrogenic in origin. See, for example, Loftus (1980a, 1992, 1993, 1994), Loftus and Ketcham (1994), Loftus and Banji (1989), Spanos (1994), and Lilienfeld, Lynn, Kirsch, Chaves, Sarbin, Ganaway and Powell (1999) for discussion of iatrogenesis, suggestion and recovered memories. The methodological implications of these studies will be given a more satisfactory treatment in 7.

[7] The argument presented here will most closely resemble those presented in Grünbaum (1984, 1993).

[8] Disorders characterized in terms of dissociation are not, however, limited to the dissociative disorders proper. Acute stress disorder, posttraumatic stress disorder, and somatization disorder are also all characterized by dissociation.

[9] Of course, the converse of the NMP is not true.

[10] See, for example, Machamer, Darden and Craver (2000), Craver (2000), Craver and Darden (2001), and also Bechtel (2001) and Glennan (1996, 2000) for related approaches.

[11] See Salmon (1984) and Machamer et al. (2000), p. 7.

[12] See Glennan (2000), p. S347.

[13] See Shaffer (2000) for a more or less complete account of partial explanation.

[14] The point is noted in Machamer et al. (2000), p. 21, and is implicit in the discussion of the adequacy of mechanical models in Darden and Craver (2001).

[15] The DID model so sketched assumes that DID is post-traumatic in origin, and this is the typical assumption that has been made by proponents of DID as a legitimate psychological disorder (Boon & Draijer, 1993, ch.s 1, 2). As we shall see, however, this is not the only possible causal model of DID. See, e.g., Spanos (1994, 1996) and Lilienfeld, et al. 1999 for elaboration of the so-called sociocognitive model of DID. On this model, DID is not the result of a real trauma induced splitting of personalities, but rather is only the iatrogenic, socially conditioned, play-acting of the patient as if he or she had multiple personalities. This need not be intentional, but it is iatrogenic. In what follows, when we refer to the theoretical model of DID, we are explicitly referring to the post-traumatic model as characterized by D1-D5. In our opinion, it seems to be something of a mistake to even treat the sociocognitive model of DID as a bona fide model of DID, for the sociocognitive model denies that symptom A. of the DSM-IV characterization really ever occurs. Moreover, insofar as the sociocognitive model treats DID as largely (if not wholly) iatrogenic in origin, to treat such cases as real DID would be rather like categorizing false pregnancies as real pregnancies simply because these two phenomena are characterized by very similar symptoms. We hope that, for obvious reasons, this is methodologically inappropriate. If it turns out that all the

symptoms exhibited by supposed DID patients are iatrogenic in this manner, then the patients in question are not really suffering from DID as it is typically understood. So, pace Lilienfeld et al. (1999) we do not accept that the issue of the existence of DID is a pseudo-issue that is tangential to the debate about the etiology of DID. The question of the etiology of a disorder is, in an important sense, just the question of the existence of a disorder *caused in such-and-such a manner*.

[16] This distinction is closely related to that discussed in Bechtel (2001).

[17] We are here borrowing the term 'candidate phenomenon' from Humphrey and Dennett (1989).

[18] See Ross (1997), ch. 2, and Lilienfeld and Lynn (2003).

[19] See Fine and Forbes (1984) for the suggestion that psychoanalytic theories ought to be judged more like theories in somatic medicine, and see Erwin (1996), ch. 2 for a forceful response.

[20] This point is closely related to Nancy Cartwright's conception of inference to the best cause. See Cartwright (1983), p. 92. To clarify, by 'eliminate' we mean only that competitor hypotheses must be shown to be significantly less explanatory, or less likely.

[21] The sort of reasoning going on in such cases is similar to inference to the best explanation as described in Harman (1965). It might be best understood as inference to the best causal model. In such reasoning we appear to be attempting to select the best causal model with respect to the data. As such, it is not really strictly required that we falsify all alternative existential claims, only that we make it clear that one of the set of plausible existential claims associated with the competing models is more likely than its competitors. The main point is that one must at least take into account all alternative models that have existential claims with non-marginal likelihoods.

[22] This condition is designed to rule out so-called 'only game in town' cases. As such, epistemic support for existential claims is differential. See Erwin and Siegel (1989).

[23] Again, as suggested in fn. 21, this principle will need to be weakened somewhat. We need not require that all plausible alternative existential claims be ruled out in the sense of being falsified. But one might be tempted to insist on the strong formulation of CMP qua strict falsification of such alternatives where the issue is a matter of the bare existence of some phenomenon.

[24] We take 'plausible existential claim' here to mean one that has a significant likelihood. How high we fix this requirement at is a partially contextual issue. See Shaffer (2000) for discussion and elaboration on this sort of theme.

[25] See Mayo (1996) for extensive discussion of such practices.

[26] Of course, all such reasoning is nonmonotonic, and so any conclusion arrived by such a procedure is revisable.

[27] As in the case of the simpler formulation of the CMP, we must interpret unlikely here to mean of sufficiently low likelihood, and, again, we believe that this is partially a contextual matter.

[28] This, of course, is a common problem in science in general.

[29] As we have suggested above, there is little in the way of other evidence for the existential claim associated with the mechanical model of DID, and, more, specifically with the existential claim asserting the instantiation of the mechanism of dissociation. First, as a survey of the literature shows (see the references in fn. 50), there is little or no physiological evidence for DID, and there do not seem to be any tests for DID other than these sort of test batteries. All of the evidence for the existence of DID is the result either of (frankly specious) physiological tests or from the components of test batteries. If the former evidence is, in point of fact, as weak as it appears to be and conflicts with our background knowledge concerning the nature and functioning of human memory (see the references in fn. 53), then unless the test batteries provide good evidence for the existence of DID, we have no good evidence for the existence of DID.

[30] There are others as well, e.g., the Dissociative Disorders Interview Schedule (DDIS).

[31] For a detailed explanation and evaluation of the DES and SCID-D see North, Ryall, Ricci and Wetzel (1993) and Boon and Draijer (1993).

[32] See, for example, Armstrong (1996), North et al. (1993), Boon and Draijer (1993), and Putnam (1989).

[33] See, for example, Butcher (2002a), Exner and Erdberg (2002), and Acklin (2002).

[34] See the various articles in Butcher (2002b).

[35] See Graham (1990), ch. 1.

[36] The criticisms raised here are similar to those raised in, e.g., Lilienfeld et al. (1999), Lilienfeld & Lynn (2003), Merskey (1992), and Spanos (1994). However, we believe that our criticisms are, nevertheless, novel in focus.

[37] For details of the MMPI and MMPI-2, see North et al. (1993), and, especially, Greene (1991).

[38] We employ the terms 'reliable' in the sense of epistemological reliabilism, whereby a method is reliable, if and only if, it tends to produce true beliefs rather than false beliefs.

[39] The criticisms we raise in this section are not entirely new as, for example, reading of Graham (1990), Levitt (1989), and Levitt and Gotts (1995) illustrates. However, despite general criticism of the MMPI and MMPI-2 as truly reliable diagnostic instruments in the sense intended by its authors, Starke Hathaway and J. C. McKinley, it has nevertheless continued to be employed with startling frequency in psychodiagnostic test batteries. This is made especially clear in the case of DID, for example, in North et al. (1993), ch. 4, Putnam (1989), ch. 4, Ross (1997), ch. 7, and Armstrong (1996).

[40] See Greene (1991), ch. 1, Graham (1990), and Colligan et al. (1983) for historical accounts of the MMPI, the MMPI-2 and the rationale behind their construction.

[41] This is especially troubling in the case of psychological phenomena that may not, in principle, be publicly observable.

[42] It is interesting to note that this seems to be one of the reasons behind the construction of the DES and the SCID-D, and these instruments were supposed to provide for more reliable differential diagnosis of DID and other dissociative disorders. But, we shall see that these instruments suffer from related, but somewhat different methodological flaws.

[43] See Bernstein and Putnam (1986), p. 729.

[44] See Carlson et al. (1993).

[45] See Boon and Draijer (1993).

[46] In spite of the caveats issued concerning the inability of the DES to ground differential diagnoses, Ross, for example, still claims that, '. . . it is the best self-report instrument for measuring dissociation available (1990), p. 173).'

[47] This is the method of validation adopted by Bernstein and Putnam (1986).

[48] See Boon and Draijer (1993) for extensive discussion of this attempt to validate the DES.

[49] For examples of SCID-D questions and structures, see Steinberg et al. (1990).

[50] See Merskey (1992), Piper (1997), Spanos (1994, 1996), Lilienfeld et al. (1999), and Lilienfeld and Lynn (2003) for defense of the view that DID is not post-traumatic in origin, but rather is likely iatrogenic in origin.

[51] An examination of the relevant literature on the physiological aspects of dissociation shows that there is some anecdotal evidence concerning the physiological basis of dissociation, as well as a strained effort to mount a kind of parity argument between the phenomenon of hypnosis and the phenomenon of dissociation. But, as demonstrated in 4 there is no real deep mechanical understanding of how dissociation is supposed to work. Consideration of the physiological basis of dissociation is addressed in Ludwig (1983), Miller and Triggiano (1992), Braun (1983a, b). For extensive discussion of the attempt to ground the existence of dissociation by parity with hypnosis see Ludwig (1983), Bliss (1983), Bartis and Zemansky (1986), Strass (1986), Speigel (1986), Yapko (1994), Bloom (1994), Ewin (1994), Loftus et al. (1994), Lynn et al. (1994), and ch. 6 of Aldridge-Morris (1989).

[52] One alternative possibility for improving the situation might be to appeal to the bootstrapping methods as understood in Cronbach and Meehl (1955) in order to improve the reliability of the instruments we have discussed. Via bootstrapping we are supposed to be able to develop tests with greater validity than the criterion on the basis of which it was originally developed. For example, our primitive test for heat by the use of our tactile senses is not very reliable, and has been replaced by far more sophisticated and reliable operational methods for determining temperature, i.e. thermometers, that correlate (roughly) with human temperature sense. So, initially fallible criterion can be replaced by conceptual enrichment/change and lead to the development of much more reliable instruments that test for the property in question. In effect, one pulls one's self up by the bootstraps. While this suggestion is perhaps useful here, it, of course, depends on the development of those more highly enriched tests and our showing that they correlate with our prior, less reliable, tests for that property. However, bootstrapping can only occur if the original property that one is trying to develop an improved criterion for actually exists. Absent any independent reasons to suspect that the original test for the criterion is at least partially reliable, there is no purchase for such bootstrapping. Such bootstrapping in no way provides the kind of evidence needed to make existential claims of the sort with which we are concerned.

[53] Of course, the inclusion of memory repression in this theory is a Freudian influence.

[54] In other words, memory is taken to be a reliable source of beliefs about the past.

[55] Representative literature on the topic includes Loftus (1980a, 1992, 1993, 1994), Loftus et al. (1994), Loftus and Banji (1989), Loftus and Hoffman (1989), Loftus and Ketchum (1994), and Thomas and Loftus (2002).

[56] See Loftus (1979).

[57] See Loftus and Hoffman (1989), p. 103.

[58] They are, or course referring to the behaviorism of Watson (1939).

[59] See Loftus (1993).

[60] These factors are detailed in Toland et al. (1991), pp. 237-239.

[61] As the technique of Maltz illustrates, ' [s]pend time imagining that you were sexually abused, without worrying about accuracy, proving anything, or having your ideas make sense' (quoted in Garry et al., 1996, p. 209).

[62] Garry et al. (1996), p. 213. This includes even pseudo-memories that are extremely bizarre. See Thomas and Loftus (2002).

[63] Additional positive evidence in favor of this view is collected in Lilienfeld and Lynn (2003).

[64] Here we differ somewhat in our assessment of the importance of the existential question concerning DID from, for example, Lilienfeld et al. (1999) and McHugh (1993). We take the existential question to be inextricably intertwined with the etiological question for the reasons just given.

[65] See Gifford (1994) and Marquis (1983) for further discussion of the therapeutic obligation.

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