INTRODUCTION

Psychiatry is the medical specialty that focuses on disorders of the mind, especially disturbances in thinking, behavior, and emotions. Psychoanalysis refers here not to a form of individual psychotherapy, but rather to a theory of mind that attends to an individual’s unique developmental trajectory within a familial and cultural context, with attention to the important impact of unconscious factors on human thought and behavior. Given these 2 definitions, we can think of psychodynamic psychiatry as the area of intersection between the domain of psychoanalysis as a theory of mind and the domain of general psychiatry. Psychodynamic psychiatry offers a perspective that allows us to engage, understand, and be useful to difficult-to-treat patients.¹

All of us have experienced work with patients we come to view as difficult to treat or, as they are sometimes called, “treatment resistant.”² There are patient-specific and disorder-specific characteristics that make patients difficult to treat, but that which is difficult often resides not in them, but in us, and in the limitations of our treatments.

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KEYWORDS
- Difficult patient
- Biopsychosocial model
- Enactment
- Treatment-resistant disorders
- Psychodynamic psychiatry

KEY POINTS
- Psychodynamic psychiatry is the intersection between general psychiatry and psychoanalysis as a theory of mind, and is built on a biopsychosocial model for understanding and treating mental disorders.
- The biomedical model has not lived up to its promise and is not supported by emerging science as robustly as is the biopsychosocial model.
- The “difficult patient” emerges in part from the limits of our treatment models and treatment methods.
LIMITATIONS OF THE BIOMEDICAL MODEL

Mathematician George Box noted that, “All models are wrong, but some are useful.”3 It was George Engel4 who proposed the biopsychosocial model, a model suggesting that understanding and treating people with mental disorders requires attention to the contributions of their biology, individual psychology, and social context. The biopsychosocial model is entirely congruent with psychodynamic psychiatry. However, over the last several decades, a narrower biomedical model has been in ascendancy and the biopsychosocial model has been in decline. Popular psychiatrist authors like Nasir Ghaemi, for example, have criticized the biopsychosocial model as lacking rigor.5

There was hope in the 1990s that the eventual decoding of the human genome and findings from brain research would confirm the value of a biomedical model. Current director of the National Institutes of Health, Francis Collins, who was then director of the National Human Genome Research Institute, suggested in 1999 that a genetic revolution throughout medicine would emerge from the Human Genome Project. At that time, Collins6 described 6 major outcomes expected to follow from decoding the human genome:

1. Common diseases will be explained largely by a few DNA variants with strong associations to disease;
2. This knowledge will lead to improved diagnosis;
3. Such knowledge will also drive preventive medicine;
4. Pharmacogenomics will improve therapeutic decision making;
5. Gene therapy will treat multiple diseases; and
6. A substantial increase in novel targets for drug development and therapy will ensue.

Although there are some small advances toward achieving these outcomes in the rest of medicine, in psychiatry the promise has fallen short. Associated with these hopes for the future are 3 implicit assumptions related to the biomedical model:

1. Genes equal disease,
2. Patients present with single disorders that respond to specific evidence based treatments, and
3. The best treatments are pills.
As it turns out, however, these assumptions are not supported by the emerging data. As Box suggests, both models are inevitably wrong, but emerging research more strongly supports the biopsychosocial model than the biomedical model.

In neuroscience, research has tended to associate multiple disorders with the same brain regions—usually the prefrontal cortex, amygdala, anterior cingulate cortex, and some others. In the absence of clear evidence that psychopathology is localized to specific brain regions, interest has shifted toward understanding how the “connectome” functions in states of mental health and disease.

**Genes Equal Disease**

Despite Collins’ predictions, we have not found genes that cause most common mental disorders. In genome-wide association study studies of depression, using samples large enough to discover relevant single nucleotide polymorphisms involved in some medical disorders, 17 single nucleotide polymorphisms account for only a small amount of the variance in heritability of depression, whereas in schizophrenia more than 125 single nucleotide polymorphisms have been detected, and some of these are found across disorders. The most significant schizophrenia single nucleotide polymorphism is a C4 gene variant related to synaptic pruning, which is associated with an increase in the base rate of schizophrenia from 1% to 1.25%. Copy number variation studies show that spontaneous mutations account for roughly 2% of the variance in causation of schizophrenia.

Although mental disorders are clearly heritable, other research reveals the importance of psychosocial factors. The presence of major depressive disorder in mothers during childrearing increases the risk of depression in their adolescent biological and adopted children. Similarly, in twin mothers with anxiety disorders, the transmission of anxiety to their offspring was better accounted for by environmental than genetic factors.

Although no genetic or other biomarkers have been found for mental disorders, early adverse experiences have emerged as an “enviromarker” associated with an increased risk of 1 or more mental or substance use disorders, and with more medical disorders.

There is widespread acknowledgment that the genetics of mental disorders are complex and polygenic, and that there is not a simple equation of genes equal disease. Some have begun to question the underlying biomedical brain and genomics focused “big idea” that drives theory and research funding. The field has shifted toward study of “gene-by-environment interactions,” which is just another way of saying “biopsychosocial.”

**Patients Present with Single Disorders That Respond to Specific Evidence-Based Treatment**

Although practice guidelines from the American Psychiatric Association are written for 1 disorder at a time, we have learned that the presence of multiple comorbid disorders is the rule rather than the exception in clinical populations. For example, Wisniewski and colleagues found that 78% of patients from the large, multisite Sequenced Treatment Alternatives to Relieve Depression (STAR*D) study of depression had comorbidity or suicidal ideation that would exclude them from randomized controlled trials (RCTs) of treatment for depression. Although we test treatments for depression on specially selected groups of patients, such patients are not representative of the majority who present in clinical settings. In the STAR*D sample, the larger group of depressed patients generally excluded from RCTs was less tolerant of medication, and had lower rates of treatment response (39% vs 52%) and remission (25% vs
Comorbid depressed patients represent 1 segment of the group of difficult patients—difficult because they do not respond as well to evidence-based treatments as we might expect from RCTs testing treatments on noncomorbid patients.

Another group of difficult patients is those with borderline personality disorder (BPD) and other personality disorders. Although part of what makes patients with BPD difficult to treat is the immature defenses that engage us in enactments, as described elsewhere in this article, our relative blindness to the presence or impact of BPD also plays a role, because we cannot treat what we will not see.

In the Collaborative Longitudinal Personality Disorder study, the presence of a personality disorder adversely affected depression outcome, caused persistent functional impairment, and extensive treatment use, and was associated with significant suicide risk. The presence of personality disorders, especially BPD, “robustly predicted persistence” of major depression. Skodol and colleagues thus call for careful assessment of the presence of personality disorders in depressed patients. However, the most frequent personality disorder diagnosis offered in the Diagnostic and Statistical Manual of Mental Disorders (DSM)-IV was “deferred,” a common practice of clinicians that leaves them blind to what may be making a patient difficult to treat—and leaves such depressed patients with undiagnosed comorbid BPD without effective treatment.

- “Difficult” patients may have undiagnosed comorbid BPD.
- We cannot treat what we will not see.

The reality that patients tend to have multiple comorbid disorders and that comorbidity predicts a greater likelihood of “treatment resistance” suggests that there are limits to our current diagnostic system as codified, even in the most recent versions of the DSM and International Classification of Disease. This recognition led Tom Insel, former director of the National Institutes of Mental Health, to launch the research domain criteria (RDoC) matrix in an effort to think beyond traditional diagnostic categories that overlap and lack specificity. The RDoC matrix includes behavior, emotion, cognition, motivation, social behavior, genes, molecules, and neural circuits. A move beyond the DSM categories makes sense, given the absence of clear links between brain mechanisms or genetic findings and clinically familiar diagnostic categories; but RDoC searches for biomarkers—a search some have likened to that for the Holy Grail—with little opportunity provided in the RDoC matrix for the role of environmental factors like relationships and attachment within gene-by-environment interactions.

Recognizing that most patients have multiple comorbid disorders, that the same brain circuitry is implicated across disorders, and that we use the same drugs for multiple disorders, Caspi and colleagues tested their hypothesis that 3 underlying factors (externalizing, internalizing, and thought disorders) account for all mental disorders in a sample of more than 1000 patients whose symptoms were followed for 20 years. Although there was some statistical support for the 3-factor model of mental disorders, a better fit was found for a 1-factor model. Calling this factor “p” for psychopathology, Caspi and colleagues note that those with a high p are at significantly greater risk for 1 or more mental or substance use disorders over time, with the opposite true for those with low p. Further, p seemed to be a function of compromised early brain development, early and recent adversity, and a family history of a mental disorder.
The findings from Caspi and associates suggest that diagnostic categories as we know them are a surface representation. An analogy to mountain ranges helps to clarify this. Those who know the Alps know Mont Blanc or the Matterhorn by their distinctive silhouettes, just as we know major depression or schizophrenia by their distinctive surface representation. However, in the case of both mountains and diagnostic categories, they are really the result of unseen forces, whether these forces are collisions between tectonic plates and erosion owing to wind and water or, in the case of diagnoses, p. Diagnostic categories as we know them seem to be surface illusions built by unseen forces.

The Best Treatments Are Pills

Consistent with the biomedical model, much of psychiatric practice has shifted toward diagnosis and prescribing in 15-minute medication checks—with some of that time spent interfacing with an electronic health record rather than the patient. Prescribing medications is seen by some as the best we have to offer, but data from RCTs reveals their significant limitations. Caspi and colleagues accurately observed that we tend to use the same medications for all disorders. And, as noted, in the STAR*D study, RCT exclusion criteria mean that antidepressant drugs work for a minority of those seeking treatment. Meanwhile, the efficacy of antidepressants has been overestimated by about one-third when unpublished studies are included in analyses, and the placebo effect accounts for as much as 75% of their benefit. In the case of psychotic disorders, the Clinical Antipsychotic Trials of Intervention Effectiveness (CATIE) study shows us that patients do not find the benefit of our medications worth the risks. In bipolar disorder, the Systematic Treatment Enhancement Program for Bipolar Disorder (STEP-BD) study suggests more is going on in this disorder than can be corrected by prescription of mood-stabilizing and neuroleptic medications. Although the biomedical model teaches us to regard medications as psychiatry’s primary treatment modality, such tunnel vision contributes to biologically focused treatment strategies that are inadequate and thus contribute to patients becoming difficult or treatment resistant. Simply put, often the source of a patient’s treatment resistance lies in us.

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The use of a biopsychosocial rather than a biomedical model invites us to include other ways of understanding and treating psychopathology beyond biology. More than 1000 studies demonstrate the efficacy of cognitive behavioral therapy (CBT) and 200 the efficacy of psychodynamic therapy (PDT) for a range of individual and complex comorbid disorders. CBT researchers have often taken the lead in providing research evidence, but a recent high-quality metaanalysis shows the equivalence of
PDT to CBT for a range of disorders—using therapists with allegiance to the therapies tested and without sham therapy comparisons that are intended to fail, as when Gilboa-Schechtman and coworkers compared prolonged exposure for posttraumatic stress disorder with sham PDT that required PDT therapists to change the subject if patients discussed their trauma.

Consistent with the limitations of diagnosis and the reality of comorbidity, Barlow and colleagues recently published a study of so-called unified protocol CBT, which targets not the symptoms of specific disorders, but underlying emotional dysregulation and neuroticism, demonstrating this form of CBT was as effective as disorder specific CBT for anxiety disorders. Psychoanalysis was the first unified protocol treatment to target underlying psychopathology rather than the surface disorder, but, as is so often the case, it is CBT therapists who took the lead in designing and conducting studies.

In summary, the biomedical model limits us as clinicians because genes do not equal disease, most patients have multiple comorbid disorders, there are high failure rates even for our evidence-based treatments, and BPD is underdiagnosed and thus undertreated. The biomedical model leads us to pursue biology when emerging science suggests we should be mindful of complexity, gene-by-environment interactions and a biopsychosocial perspective. These shortcomings of the biomedical model contribute to the proportion of patients perceived as difficult.

ENACTMENT: A CO-CREATED PSYCHOSOCIAL MECHANISM THAT MAKES PATIENTS DIFFICULT

One reason patients are experienced as difficult is their engagement of us in ways that leave us feeling confused, hurt, angry, guilty, lost, or all of these. We might wonder, “What’s going on here? Why does this patient have to be so ‘difficult’?” It is the propensity to evoke such feelings in treaters that makes patients with BPD, for example, so stigmatized—disliked, avoided, and seen as difficult—with the term borderline sometimes hurled as an epithet. Patients with BPD and other personality disorders have a unique ability to get under our skin in unpleasant ways that contribute to our experience of them as difficult. When this happens, we and they are often caught up in a process called an enactment by psychodynamic psychiatrists. Enactment is formally defined as “a pattern of non-verbal interactional behavior between 2 parties in a therapeutic situation.” Understanding enactments offers an opportunity to untangle difficult interactions and be of more use to patients, with more equanimity in ourselves.

The origin of such experiences with patients is in their immature defenses—and the way they engage our own. Humans, including those with BPD, and those who treat patients, deploy a range of defenses from the more mature (eg, intellectualization, sublimation, humor) to the more immature (eg, splitting, projective identification, acting out). Those with BPD differ from most of us who do treatment by virtue of the greater frequency with which they deploy immature defenses like projective identification, which is the relevant defense to understanding enactment.
From Projective Identification to Enactment

The simplest definition of projective identification involves patients putting into us (i.e., projecting) a disavowed affect that represents elements of their life history. In this definition, our role is passive—like an antenna picking up a transmitted signal. The following case example illustrates this definition.

**Case Vignette for Projective Identification**

A 30-year-old man in weekly PDT for feelings of self-doubt receives a reduced fee from his woman therapist. In the course of therapy, he meets and decides to marry a wealthy woman, but struggles in therapy with worry that he is marrying her for money rather than love. When the woman therapist raises the reduced fee after the marriage, as she said she would, the patient is enraged at the therapist's greediness.

In this example of projective identification, the patient struggling with self-doubt disavows fear of his own potential greediness and finds it in the therapist.

Of course, simple definitions are just that, and in reality projective identification is more complex. That is, the other onto whom the projection is placed has some kind of "hook" on which the projection can be hung. We unwittingly accept the projection because of this hook that comes from our own character and life history. In the case vignette, this hook might be the therapist's discomfort with being seen as greedy, especially when she raises the fee.

However, things get even more complicated when we remember that projective identification is an immature defense used by all humans to differing degrees. And therapists are decidedly human. When both parties in a therapeutic situation are involved in mutual and complementary projective identification—a patient disavowing an unacceptable affect and putting it on a hook in the therapist, and a therapist reciprocally disavowing an unacceptable affect and putting it on a hook in the patient—then they are in an enactment.29

An "enactment" is a set of mutual and complementary projective identifications involving both participants in a therapeutic situation, and informed by the life histories of both participants.

We can see the transition from projective identification to enactment by returning to the case vignette and adding a bit more detail.

**Case Vignette for Enactment**

A 30-year-old man in weekly PDT for feelings of self-doubt receives a reduced fee from his woman therapist. Both are therapists, but the patient works in a low-fee community mental health setting for a low salary, whereas the therapist is in a lucrative private practice—and struggles throughout the treatment with countertransference guilt about the difference between their levels of compensation for similar work. In the course of therapy, the patient meets and decides to marry a wealthy woman, but struggles in therapy with worry that he is marrying her for money rather than love. When the woman therapist raises the reduced fee after the marriage, as she said she would, the patient is enraged at the therapist's greediness. The therapist feels she is doing the sensible thing, but also feels guilty, and repeatedly makes accounting mistakes in preparing bills for the patient—sometimes overcharging and sometimes undercharging.
We can see how each party in the enactment is caught projecting something disavowed, while finding a hook in the other to hang it on. The result is a tangled engagement that is difficult for both. Sometimes enactments like these lead to fights or impasses in therapy, but they also offer an opportunity to grasp something important in the entanglement and deepen understanding and engagement between the parties.

Enactments are ubiquitous and inevitable therapeutic phenomena. They are not limited to dynamic psychotherapy, but occur in prescribing relationships, on treatment teams, and in hospital or clinic systems. In individual work with patients, whether therapy or other clinical work, enactments are most common when immature defenses are most common, as in work with patients with BPD or those with early adverse experiences involving previous caretakers that affects new caretaker relationships. Enactments may be isolated events, but in work with enactment-prone patients, enactments are often the terrain over which treatment progresses. Our task is not simply to avoid them, but to learn to use them.

Skiing offers a useful analogy for enactments in therapeutic situations. In both skiing and therapy, one is on a slippery slope. As in skiing, sliding down hill on the slippery slope is expected—even inevitable. What separates experienced from inexperienced skiers, and therapists, is how well they find their edges as they slide down hill, so that they can control the speed of their slide to stay in control and avoid crashing. Finding ones edges, that is, learning to use enactments, is addressed elsewhere in this article.

Patients with Abuse Histories as Difficult Patients

Psychodynamic therapists and psychodynamic psychiatrists learn to operate from a stance of nonjudgmental, warm, and empathic technical neutrality. Technical neutrality does not imply that the therapist or other clinician is distant, uncaring, silent, or uninvolved. Psychodynamic clinicians learn to “take” the transference that is offered, that is, they tolerate the transference the patient brings into the work and the associated countertransference. However, some transferences are hard to take, and therapists and other clinicians often enter enactments by unwittingly refusing or actualizing the transference. This is a special problem with the group of difficult patients with histories of abuse.

In work with patients with abuse histories there are 3 readily available transference roles for the therapist or other clinician: perpetrator of abuse, victim of abuse, and silent witness who tolerates abuse. Refusal or actualization of these transferences leads to predictable patterns of enactment illustrated in Table 1.

When the transference is to the clinician as perpetrator of abuse, actualizing the transference may lead the clinician unwittingly to become abusive, sadistic, and unempathic. If the perpetrator transference is refused, that is, if the clinician has trouble tolerating being seen as a perpetrator of abuse, he or she may become excessively kind and solicitous, demonstrating how much he or she cares for the patient, sometimes in extreme cases leading to “loving” boundary violations, including extending sessions and even sexual boundary violations.

When the transference is to the clinician as victim of abuse, actualizing the transference may lead the clinician unwittingly to become abusive, sadistic, and unempathic. If the perpetrator transference is refused, that is, if the clinician has trouble tolerating being seen as a perpetrator of abuse, he or she may become excessively kind and solicitous, demonstrating how much he or she cares for the patient, sometimes in extreme cases leading to “loving” boundary violations, including extending sessions and even sexual boundary violations.

When the transference is to the clinician as victim of abuse, actualizing the transference may lead the clinician becoming
distant, unempathic, uninvolved, and dissociated in the work. If the silent witness transference is refused, we may see the clinician become an overzealous rescuer, and intolerance of not knowing may lead to implanting of false memories of abuse that create pseudocertainty about that which cannot really be known.

For psychiatrists caught in refusing or actualizing these transferences with difficult patients, overmedication of the patient is a frequent result, as if to medicate away the transference such difficult patients bring into the encounter.

**Using Enactments**

The capacity to use enactments involves 3 steps: detect, analyze, and use the enactment. We detect enactments by attending to our free-floating responsiveness in sessions. Analysis of enactments requires unpacking their meaning. What are we caught in? What are the bits of projective identification unfolding between therapist and patient? Analyzing an enactment requires that one know one’s blind spots and hooks, and often is facilitated by consultation with a colleague or supervision. Using an enactment may involve engaging a patient in serious discussion of the details of what you are both caught in, with due caution about undue disclosure of the therapist’s life history. Often the unpacking of an enactment, with each party owning their role in the tangled situation that has emerged, leads to a deeper and more intimate engagement. In other situations, therapists may simply realize they are caught in repeating behavior that they need to understand, contain, and stop repeating, without necessarily discussing the issue with the patient.

**Table 1**

Refusing and actualizing transferences with patients who have experienced abuse

<table>
<thead>
<tr>
<th>Transference to Therapist in Role of →</th>
<th>Perpetrator</th>
<th>Victim</th>
<th>Silent Witness</th>
</tr>
</thead>
<tbody>
<tr>
<td>Result if transference is actualized</td>
<td>Therapist becomes abusive</td>
<td>Therapist in masochistic surrender</td>
<td>Therapist distant</td>
</tr>
<tr>
<td></td>
<td>In extreme cases, “sadistic” boundary violations</td>
<td>Guilty</td>
<td>Unempathic</td>
</tr>
<tr>
<td></td>
<td>Overmedicates</td>
<td>Burnout from “vicarious traumatization”</td>
<td>Uninvolved</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Dissociated</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Overmedicates</td>
</tr>
<tr>
<td>Result if transference is refused</td>
<td>Therapist becomes excessively kind, solicitous</td>
<td>Counterattacks</td>
<td>Overzealous rescuer, may overmedicate</td>
</tr>
<tr>
<td></td>
<td>In extreme cases, “loving” boundary violations</td>
<td>Termination</td>
<td>Intolerance of not knowing, with implanting of false memories</td>
</tr>
<tr>
<td></td>
<td>Overmedicates</td>
<td>Overmedicates</td>
<td></td>
</tr>
</tbody>
</table>

The capacity to use enactments involves 3 components:

- Detect the presence of enactment,
- Analyze its meaning, and
- Use what is learned.
Ultimately, the best protection against destructive enactments, in therapy, but also in other kinds of clinical work with difficult patients, is (1) to have the experience of a personal analysis or therapy to learn one’s hooks and blind spots, (2) supervision or consultation, (3) careful negotiation of a therapeutic alliance that includes exploration of what unfolds in the treatment relationship (whether or not it involves therapy), and (4) developing the capacity to “take” the transference that is offered from a stance of warm, empathic, nonjudgmental technical neutrality.

The best protection against destructive enactments:
- Personal analysis or therapy,
- Supervision or consultation,
- Negotiate a therapeutic alliance that includes exploration of the treatment relationship, and
- Develop the capacity to “take” the transference from a stance of warm, empathic, nonjudgmental technical neutrality.

SUMMARY

There will always be difficult patients, but we are wise to recognize the role we play as individuals and as a profession in contributing to their creation. The biomedical model has not lived up to its promise, but it is currently entrenched in our scientific culture as a relevant “big idea” for the field. The biopsychosocial model is more fully supported by emerging science than is the biomedical model, with “gene-by-environment interaction” just another way of saying “biopsychosocial.”

At the level of individual work with patients, we are wise to learn to use psychodynamic concepts like projective identification and enactment to help us understand how we unwittingly co-create the so-called difficult patient.

REFERENCES


