Etiologically Speculative Versus Etiologically Neutral Psychoanalysis

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Abstract

This paper distinguishes between two broad approaches to psychoanalysis: etiologically speculative and etiologically neutral. Etiologically speculative approaches advance causal claims about psychopathology, often through reconstructions of early experience, whereas etiologically neutral approaches treat such formulations as provisional hypotheses whose validity depends on evidence beyond their clinical plausibility. Drawing on the distinction between narrative and historical truth, the paper argues that compelling clinical narratives do not establish etiological validity. Etiological neutrality is presented not as a prohibition against causal theorizing, but as an epistemically modest stance that distinguishes hypotheses about particular symptoms, defenses, and relational patterns from claims about the causes of psychiatric conditions as a whole. The paper situates this stance within a medical model that permits treatment in the absence of definitive causal knowledge and concludes that psychoanalysis can retain scientific credibility by clearly distinguishing meaning, mechanism, and cause.

Keywords: etiology, psychoanalysis, psychotherapy, medical model, validity

This paper describes two broad approaches to psychoanalysis, one more compatible with a scientifically grounded, medical model of psychopathology and one more willing to advance causal claims on the basis of clinical reconstruction. I refer to these as *etiologically neutral* and *etiologically speculative* psychoanalysis. These terms are intended as ideal types rather than mutually exclusive schools; individual analysts and theories may occupy positions between them. Etiological claims concern the causes and mechanisms of mental illness and therefore raise questions about the boundary between what is known, what is inferred, and what remains speculative. It is along this boundary that psychoanalysis must decide how far clinical interpretation can legitimately extend into causal explanation. Although the focus here is psychoanalysis, the same epistemic problem can arise in other psychotherapies when theory-congruent clinical observations are treated as evidence for a particular etiology.

Etiologically Speculative Psychoanalysis

Etiologically speculative psychoanalysis refers to approaches that move from clinical interpretation or developmental reconstruction to claims about the origins or causes of psychopathology without sufficient independent evidence to establish those claims. Such formulations often invoke early developmental experiences, unconscious conflicts, or relational traumas and may present reconstructed accounts of the past as explanations of how a disorder came to be. The problem is not that causal hypotheses are proposed, but that the degree of causal confidence may exceed what the available evidence can support.

Historically, etiological speculation has been central to psychoanalytic theory. Freud's early formulations of hysteria, obsessional neurosis, and other conditions were explicitly etiological, positing particular developmental events or psychosexual conflicts as necessary or important antecedents. Later schools retained this orientation in revised form. Object relations

theory, Kohutian self psychology, and relational approaches have each offered developmental accounts centered on early relationships, failures of attunement, or disruptions in self-cohesion. Some formulations have gone further still, specifying not only the type of developmental failure but its presumed timing, locating the origins of psychopathology in disruptions thought to occur at particular developmental stages or even at discrete ages (see, e.g., Masterson, 1976). Contemporary versions of these theories are often more probabilistic and nuanced than their earlier forms, but the epistemological question remains the same: What warrants moving from a clinically plausible developmental formulation to a causal claim?

There is nothing inherently unscientific about such theorizing. Theory is indispensable to clinical science: it guides observation, organizes clinical material, and generates hypotheses for empirical investigation. Psychoanalytic observations may also yield plausible causal hypotheses about particular symptoms, defenses, affects, or interpersonal patterns. The difficulty arises when the plausibility, coherence, or therapeutic usefulness of an interpretation is treated as evidence that the inferred causal account is historically or etiologically true. We must allow that some theories may be false and that a cohesive narrative may be compelling and even helpful without accurately identifying the causes of a patient's condition. A clinical observation may fit several competing causal explanations, a problem philosophers describe as *contrastive underdetermination*. A hypothesis is strengthened not merely when clinical material is compatible with it, but when the evidence helps distinguish it from plausible alternatives (Stanford, 2023). At minimum, etiological confidence should therefore depend on evidence that is independent of the clinical interpretation itself and converges across methods. As Spence (1982) famously observed, "Interpretations are persuasive not because of their evidential value but because of their rhetorical appeal; conviction emerges because the fit is good, not because we

have necessarily made contact with the past. . . . If we confuse narrative truth with historical truth, we will never make the necessary distinction, and our theory will never rise above the level of metaphor." Schafer's (1976) action language represents a related, though not identical, effort to describe psychoanalytic phenomena without treating metapsychological constructs as literal causal entities.

Spence's distinction between narrative and historical truth captures a central epistemological problem. Psychoanalytic interpretations may be coherent, emotionally resonant, and clinically useful without corresponding to the actual events or mechanisms that produced a psychiatric disorder. This does not render them meaningless or clinically unimportant. Rather, it limits what can legitimately be inferred from them. An interpretation may illuminate the meaning of a symptom or the organization of a relational pattern without establishing why the condition itself developed. When these distinct levels of explanation are collapsed, metaphor and reconstruction can be mistaken for causal mechanism. This may pose little difficulty for those who regard psychoanalysis primarily as a hermeneutic discipline, but for those of us who believe in psychoanalytic science, it presents a serious problem of validity.

Etiologically Neutral Psychoanalysis

In contrast, etiologically neutral psychoanalysis adopts a modest stance toward the causes of mental illness when those causes have not been established. It does not deny that causes exist, nor does it prohibit psychoanalysts from proposing causal hypotheses. Rather, it insists that such hypotheses be held in proportion to the evidence supporting them. It is especially important to distinguish the etiology of a psychiatric condition from the development of particular psychological features within an individual. A patient's fear of intimacy, defensive style, or recurrent interpersonal pattern may plausibly have been shaped by specific relational experiences

without those experiences having caused the psychiatric condition as a whole. Etiological neutrality therefore concerns the level of confidence attached to causal claims, not the elimination of causal thinking. This stance aligns psychoanalysis with the broader medical model, which routinely proceeds with investigation and treatment in the absence of definitive knowledge about etiology. Its relevance is especially evident in light of contemporary tendencies to attribute a wide range of psychopathology to trauma, even where the underlying mechanisms and specificity of such causal claims remain unclear (see Paris, 2022).

In a classic paper in the *American Journal of Psychiatry*, Guze and Murphy articulated such a position clearly:

> Since the causes of the major psychiatric disorders are unknown, and specific treatment is lacking, it is desirable to formulate an approach to psychotherapy that will permit treatment without commitment to any unproven theory. We endorse an empirical approach to the difficult problem of treating patients in the absence of definite knowledge about etiology and pathogenesis. It requires no acceptance of unproven theory; it is flexible and tentative (Guze & Murphy, 1963, p. 53).

Guze later expanded this position, noting that a "rehabilitative view of psychotherapy… requires no assumptions concerning etiology or pathogenesis… *Even a non-etiological approach to psychodynamic psychotherapy, in which the therapist offers no etiologically significant interpretations, can be included under the same rubric*" (Guze, 1992, p. 80, emphasis added).

While Guze was a biological psychiatrist seeking to defend psychiatry as a *bona fide* branch of medicine, this kind of etiological caution also has precedent within American psychoanalytic psychiatry. In their landmark study of the borderline syndrome, Grinker et al. (1968) noted how readily psychoanalytic writers had attributed the condition to deficiencies in

early mother-child relationships, neglectful mothering, or childhood trauma despite limited evidence establishing these factors as causal. They concluded that “no specific etiological agents or conditions are known to produce the borderline syndrome” (p. 178). Kernberg (1975) likewise emphasized the interaction of biological and environmental influences, while Gunderson (1984) stressed the complex and multicausal nature of borderline personality disorder. These formulations illustrate a psychoanalytic approach that permits developmental hypotheses while resisting the elevation of any single clinically plausible account into an established etiology.

An etiologically neutral psychoanalysis therefore views interpretations and developmental reconstructions as hypotheses: possibly true, possibly false, and varying in the degree to which they capture actual mechanisms of psychopathology. This does not mean that interpretations are without significance, nor that causal hypotheses should never be advanced. It means that their epistemic status should be made explicit. Some interpretations concern meaning; others propose mechanisms; still others imply causes. These are not interchangeable claims. From this perspective, psychoanalysis can elucidate patterns of meaning, conflict, defense, and interpersonal experience while remaining appropriately cautious about whether those patterns explain the origins of a psychiatric condition.

This position is consistent with contemporary psychodynamic psychiatry, which emphasizes integration with neuroscience, descriptive psychiatry, and empirical research. As Friedman and colleagues (2014) argue, modern psychodynamic psychiatry distinguishes clearly between psychodynamics and etiology and adopts a positive attitude toward both research and descriptive diagnosis. Psychoanalysis, in this view, contributes distinctive knowledge about psychological meaning and mental functioning without claiming privileged access to causal

mechanisms. Causal hypotheses remain legitimate, but their status depends on the quality and convergence of the evidence supporting them rather than on their clinical persuasiveness alone.

Discussion

Etiologically speculative formulations may organize experience, offer narrative coherence, generate hypotheses, and even facilitate therapeutic change. Their clinical utility, however, does not establish their etiological truth. The essential point here is that usefulness, plausibility, and causal validity are different standards. The history of psychoanalysis is replete with examples of etiological claims extending far beyond what could be established, often at the expense of the field's scientific credibility. This history supports a more explicitly modest approach to causal inference.

It also requires attention to the level at which a causal claim is being made. An interpretation may plausibly explain why a particular symptom emerged, why a defense is mobilized in a certain relationship, or how a recurring interpersonal pattern developed, while leaving unanswered the broader question of why the person developed a psychiatric condition. Clinically, this stance shifts the emphasis from reconstructing presumed causal histories to observing and working with present patterns of experience, affect, defense, and relationship while maintaining explicit awareness of the limits of inference.

If psychoanalysis is to retain its place within a scientifically grounded psychiatry, it must distinguish clearly between interpretation and explanation and, more specifically, between meaning, mechanism, and cause. In practical terms, psychoanalytic authors and clinicians should signal clearly when they are moving from description or interpretation to causal attribution, and should avoid presenting developmental reconstructions as established causes unless independent evidence warrants that claim. Etiological neutrality, understood in this sense, offers a way of

preserving scientific humility while maintaining the clinical richness and theoretical generativity of psychoanalytic work across the varied forms of psychopathology.

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