The science of psychoanalysis

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Can psychoanalysis take its place in the science that is psychology? I want, for now, to put aside the therapy, and ask about the theory, its evidence and generation. For at the heart of psychoanalysis as theory and therapy is a theory about the nature, development and functioning of the human mind, especially in relation to motives. There are a number of features of this theory, in particular the role and nature of unconscious mental states and processes, that makes it recognizably distinct and a competitor with other psychological theories deriving, for instance, from cognitive psychology or neuroscience.[1] I take the question of whether this theory is scientific to be the question of *how* we can establish whether its claims are true or not. It is a question about the nature of the evidence and the methods that are used to gather that evidence. It is also a question of the way the evidence and methods are marshaled in support of theoretical claims – for if a discipline were consistently to extend its claims beyond what the evidence could support, we would wish to rein in, or at least contextualize, such speculation before recognizing the enterprise as a solid part of science. Furthermore, if psychoanalysis can rightly be thought of as scientific, given what this entails regarding the nature of its evidence base and methods, there should be good empirical reason – though of course, not proof – to think many of its central claims are true.

Psychoanalysis has a distinct method that has generated a distinct body of evidence, the clinical data. *Clinical data* are the data produced in the clinical setting, comprising the behavior, including verbal behavior, of the patient. Clinical data therefore include manifestations of occurrent thoughts, feelings, and free associations; reports of dreams, memories, fantasies, and physical symptoms; responses to questions and interpretations. In addition to the words spoken, the manner and tone of speech, pauses, corrections, moments of forgetting or going blank, facial expression, body language, and so on, are all part of the data. Many analysts also include, as a further part of the clinical data, the emotional responses felt by the analyst in response to the verbal and non-verbal behavior of the patient (the counter-transference).

This methodology and subject matter clearly places psychoanalysis outside the natural sciences. It may, however, fall within the social sciences, as do certain other branches of psychology, including personality psychology and social psychology. These and other social sciences concern motives, deploy interview methods (among others), and involve the interpretation of human behaviour and communication. There is a worthy philosophical debate, starting with Dilthey, over whether any discipline that interprets human behaviour and motives can be deemed a 'science'. My concern here is not to engage with this larger debate. I shall assume an ecumenical understanding of science, grounded in such practicalities as methodological sophistication, the growth of agreed results, and integration with cognate branches of the wider discipline. We

[1] While psychoanalysis, cognitive psychology and neuroscience all posit unconscious mental states and processes, the nature and role of such states and processes differ on *typical* accounts of each theory. However, no clear relation between the psychoanalytic unconscious and the cognitive unconscious can be made out at present, as there are a variety of interpretations within psychoanalysis and there is extensive debate over the nature and relation of 'personal' and 'sub-personal' states in cognitive psychology and neuroscience. See Gardner (2000) for an overview of some of the issues.

should, for example, accept personality and social psychology as scientific disciplines. I shall assume, then, that a 'hermeneutic science' is possible, not a contradiction in terms, and that if psychoanalysis qualifies as a science, it is a social and 'hermeneutic' science, rather than a natural science.

It is important to recognize at the outset that it is possible that many of the claims of psychoanalytic theory are true without their being established or even evidentially supported by the clinical data. If they are true, we would expect to find *extra*-clinical data that supported the claims, deriving, for instance, from neuroscience or social psychology. But if the clinical data play little role in establishing their truth, psychoanalysis would not be established as part of the science of psychology, since it would not be contributing materially to the project of evidentially establishing its own theory of mind.

Thus, for psychoanalysis to qualify as scientific psychology, we want it to generate data that can evidentially support theoretical claims. Its methods, therefore, must at least be capable of correcting for biases produced in the data during the process of generating it; and we must be able to use the data in sound forms of inference and reasoning. Of course, I mention all this precisely because critics of psychoanalysis have claimed that it fails on both counts, and thus whatever warrant its claims have derive from other sources. In what follows, I discuss three key objections, and then consider their implications together with recent developments in the generation and testing of psychoanalytic theory. The first and most famous is that of 'suggestion'; if it sticks, clinical data may be biased in a way that renders all inferences from them unreliable. The second, sometimes confused with the first, questions whether the data are or can be used to provide genuine tests of theoretical hypotheses. The third will require us to consider the question of how psychology can reliably infer motives from behavior. Before embarking on discussion of the three objections, I shall put my cards on the table.

1. The psychodynamic model

I shall argue that parts of psychoanalytic theory can be defended against these objections, but that other parts cannot. In other words, the clinical data contribute materially to establishing the truth of certain psychoanalytic claims, but much less to others, which will therefore require significant extraclinical support to be warranted. However, there is good reason for considering those aspects of psychoanalytic theory that are defensible as central to the theory as a whole and its historical development. I refer to *the psychodynamic model of mind*. This is not an original conclusion, but one that has been reached by many 'scientifically-minded' psychoanalysts over the last 40 years.

The psychodynamic model posits the existence of unconscious motivational forces and of psychic defenses, unconscious mental processes that change what we think and experience so as to reduce or eliminate anxiety and other painful feelings (such as guilt, shame, and envy), especially when connected to self-esteem. Examples include projection, denial, passive aggression, intellectualization, and repression. It claims that these motivations and defenses significantly influence our behavior and conscious mental functioning, and that understanding such influences is central to understanding the development of our selves, our relationships with each other, and mental health. The model is elaborated in the *clinical theory* of psychoanalysis, which postulates typical structures of motivation, their typical effects, and their manifestations in the consulting room, e.g. the existence and nature of defense mechanisms, such as repression, projection, and so on, and their clinical manifestations in conflict, compromise, resistance and transference. The clinical theory additionally provides an account of the causal role of these mental structures in the manifestations of mental illness and character traits, and a theory of how therapy works.

The clinical theory may be contrasted with the *metapsychological* and *etiological* theories of psychoanalysis. Metapsychological theories – and there are rival schools here – provide accounts of the fundamental structure or organization of the mind, e.g. the id, ego and superego in Freud, the paranoid-schizoid and depressive positions in Klein, how such structures function, and their relation to mental illness, character and mental health. Etiological theories describe the causal origins and typical development of the structures falling under clinical and metapsychological theory, esp. in relation to in childhood experience.

I believe that for many metapsychological and etiological claims, critics of psychoanalysis have been right to argue that psychoanalysts cannot reliably infer them from clinical data alone. Such theories are much harder to tie closely to the clinical data, and so depend much more heavily on extraclinical research. However, I shall argue below that psychoanalysis can and does develop the clinical theory in a scientifically rigorous and respectable way. This defence presupposes that the clinical theory can be satisfactorily separated from metapsychological and etiological theories. There are aspects of clinical theory, esp. relating to theories of clinical *technique*, which I grant cannot; and it may be that central concepts and claims require more precise formulation, unburdened of metapsychological or etiological baggage, for the separation to be fully successful.[2] However, in support of the separation, it is worth noting that while schools of psychoanalysis disagree over matters of metapsychology, etiology and technique, they discern and conceptualise clinical phenomena in shared *psychodynamic* terms – resistance, defence, conflict, compromise, transference (see Wallerstein 1988, 1990, 2005; Rosenblatt 1989; Fonagy and Target 2003, Ch. 1.2; Miller 1988; Westen 1998, 334-5). That these terms are so often *further* theorised in relation to each particular schools' metapsychology should not be allowed to obscure this common ground.

Before embarking on discussion of the objections, it is worth noting that the assumption that psychoanalysis infers its theoretical claims using only the traditional methodology of case studies taken from clinical data – which I shall call the 'clinical methodology' – is *narrow and out-of-date*. This is a matter to which we will return in §6.

2. Suggestion

> there is a risk that the influencing of our patient may make the objective certainty of our findings doubtful… This is the objection that is most often raised against psychoanalysis… If it were justified… we should have to attach little weight to all that it tells us about what influences our lives, the dynamics of the mind or the unconscious. (Freud 1917, 452)

> There is great confusion regarding the terms 'suggestion' and 'suggestibility'. Both concepts are mingled with related notions like obedience, persuasion, imitation, social influence, or hypnosis, or they are subsumed under the heading of 'influence' without further distinctions. (Gheorghiu et al. 1989, ix)

2.1 The problem

What is 'suggestion' and what is the challenge it poses to the credibility of psychoanalysis as a science? There is no exact definition, but the main features, agreed upon by most theorists today,

[2] There has been little effort by the psychoanalytic community as a whole to undertake the task, which, I believe, is a result of the commitment of psychoanalysts to their particular theoretical schools.

are these: Suggestion comprises communications and features of the structure and setting of communication that, while bypassing the subject's critical and/or conscious reflection, lead to a change in their mental states (beliefs, memories, desires, etc.), mental state reports, and/or behavior. Empirical and psychoanalytic understandings alike describe the effects of suggestion as non-rational or irrational, bypassing critical reflection and conscious control (e.g. Eysenck, Arnold & Meili 1975, 1077; Levy & Inderbitzin, 2000, 743). On this understanding, suggestion frequently occurs in everyday life, e.g. in advertising, and perhaps even forms a part of all communications intended to influence what people think (Schwanenberg 1989, 263-7). Suggestive influences will be heightened in certain social settings (Gheorghiu 1989), such as education, therapy, or psychological experiments. Quite rightly, then, the idea that suggestion occurs in the clinical setting of psychoanalysis is widely accepted by psychoanalysts themselves.

This is a problem because the responses of the patient in analysis form the clinical data from which psychoanalysis draws its theoretical inferences. If suggestion occurs, then there is the possibility that the data have been *biased* in favor of psychoanalysts' prior theories. As a result, the data falsely appear to provide evidential support for such theories. The best way to understand this, methodologically, is in terms of 'experimenter expectancy effects'. Expectancy effects occur when our expectations work as self-fulfilling prophecies, helping to bring about just what we expected (Rosenthal 2000). Experimenter expectancy effects occur when the experimenter's expectations – created by his or her favored theory – affect subjects' behavior in such a way as to confirm those expectations. Such effects are widespread in psychological experiments (Rosenthal & Rosnow 2009, 173-4), and occur in everyday life (Rosenthal 2000). They occur as a result of the experimenter unintentionally and unconsciously providing suggestive cues that affect subjects' behavior, though precisely how this occurs is still unclear. The best hypothesis is that highly complex auditory and visual cues are involved. In psychoanalysis, the analyst's interpretations and unconscious reinforcing of certain types of patient communication (through vocalizations, verbal and non-verbal displays of interest etc.) are thought to be central. The suggestive effects of such communications are reinforced by the analyst's authority and the dynamics of the transference (Fisher & Greenberg 1977, 363; Erwin 1996, 96; Gellner 1985, 56-66).

So we can put the charge of suggestion more precisely in these terms: it is possible that the theoretically-based expectations of the analyst influence the behavior and free associations of the analysand so as to produce erroneous data that support those very expectations. Unless this possible bias can be corrected for, clinical data do not supply independent support for psychoanalytic theoretical claims.

2.2 The solution

The nature of experimenter expectancy effects is such that we cannot look for a satisfactory solution at the level of individual analysts and their understanding of individual patients, e.g. by trying to find some form of training analysts that would prevent such effects from occurring. Such a solution would also stand in stark contrast to the rest of science, in which the functioning of the community in testing the claims of individuals and producing corroborative results takes centre stage. This applies in the case of experimenter expectancy effects as elsewhere. If independent enquirers produce similar data and come to the same conclusions, then we can have confidence that experimenter expectancy effects have not substantially influenced the data (Rosenthal & Rosnow 2009, Ch. 18). The solution, then, doesn't lie *in* the clinical setting, but in stepping back from it.

It may be thought, however, that this solution is unavailable to psychoanalysis. There is a striking lack of theoretical agreement between different psychoanalytic schools, and the findings of one

school are not taken to corroborate the findings of another. Indeed, just this thought has been used to support the charge of suggestion (Grünbaum 1984, 211; Gellner 1985, 92; Erwin 1996, 99; Jopling 2008, 154). But this apparent lack of agreement is not what it seems, as it almost exclusively concerns the *metapsychological and etiological theories* (and implications drawn from these for the theory of therapy). Over the psychodynamic model, there is considerable agreement. As noted in §1, despite their other differences, there is good reason to think that psychoanalysts from all schools discern common clinical phenomena, and arrive at common conceptualizations of these phenomena in psychodynamic terms. It is these common clinical phenomena and their common psychodynamic conceptualization that count as 'corroborative findings' of the form that may dispel the charge of suggestion, given the fact that they are found across schools of psychoanalysis that differ in other areas of theory.

This response to the charge of suggestion is worth restating precisely. It is possible, using the usual appeal to corroborative findings by different researchers, to judge reliably where suggestion, understood here in terms of experimenter expectancy effects, has not biased the data. Data relevant to the psychodynamic model of the mind is corroborated, and we may thus have confidence in it. However, metapsychological or etiological claims are not corroborated. So, when looking for data that support such claims, we cannot use corroboration to rule out biases in the data that result from suggestion.

Of course, specific interpretations of individual cases cannot be defended against the possibility of suggestion using this defense by appeal to corroboration (unless independent raters have access to the data). This is not uncommon; scientific psychology establishes claims of statistical generality rather than accounts of individual behavior. But psychoanalysts can use well-established general claims to inform their interpretations about individuals, which need to established in a different way (see §4), and individual judgments may work well enough for the practical purposes of understanding clinical data and contributing to therapeutic improvement. Our concern here, in any case, is the theory, not its clinical deployment.

This is not the last word on the problem of suggestion. For example, critics may argue that psychoanalysts do not count as sufficiently 'independent' researchers, and so corroboration across different psychoanalytic schools counts for little.[3] Independently of this objection, we are right to think that psychoanalysis should seek consilience with non-psychoanalytic theories. But this applies to schools of scientific thought generally – no theory stands in isolation against its rivals. Theories that are supported by a variety of evidence, produced in different contexts and with different methods, are more strongly supported thereby. I shall briefly rehearse a small sample of such evidence in §5, and discuss its implications in §6.

[3] I consider this at length in Lacewing (2013). In brief, I argue that psychoanalysts have clearly been willing to challenge each other and their teachers, as the existence of differing schools demonstrates; that if suggestion were the explanation for agreement over the psychodynamic model, we would also expect the clinical data of each school to strongly support not only the psychodynamic model, but also its own distinctive metapsychological and etiological theories, and there is scant evidence of this; and that nothing in the empirical literature on the operation of suggestion indicates that an effect could be produced that is sufficiently strong and widespread to account for the agreement on the psychodynamic model of mind and its support by the clinical evidence.

3. Popper and confirmation bias

3.1 Popper

Popper famously argued that scientific claims must be tested – or at least be able to be tested – against experience in a 'critical' manner, one that seeks possible refutation and enables undermining evidence to come to light. Science proceeds by attempting to refute hypotheses; we may provisionally take as confirmed those hypotheses we have tried and failed to refute. Psychoanalysis, by contrast, Popper states, proceeds by seeking out verifications of its claims. Clinical data are 'interpreted in accordance with established psychoanalytic theory', but genuine evidential support only comes from testing claims in ways that may demonstrate the claims are false. Adopting this approach 'immunizes' its claims against refutation. Psychoanalysts do not countenance refuting evidence: not only do psychoanalytic concepts such as 'ambivalence' make it 'difficult, if not impossible' to agree on when a psychoanalytic interpretation has been falsified, but the theory attempts to explain 'practically everything' about human behavior, allowing no conceivable event to refute it: 'we can say, prior to any observation, that every conceivable observation will be interpretable in the light of psychoanalytic theory' (1995, 87). Therefore, psychoanalysis cannot qualify as a science (1963, 34-38; 1983, 162-174; 1995, 87-89).[4]

Popper provides very little argument or evidence for the claim I have quoted, and it is unpersuasive. Freud claimed that 'the theory of psychoanalysis is an attempt to account for two striking and unexpected facts of observation… the facts of transference and of resistance' (1914, 16). These are key indicators of the operation of dynamic unconscious processes, and if there were no evidence of them in the clinical setting, psychoanalysis would probably be placed beyond rescue. It is not *inconceivable* that there should be no such evidence (though we may have to suppose much about human behavior to be different). For example, as evidence against resistance, we could observe that people are equally disposed to consciously recall and recognize motives that caused them psychic pain as those that did not, that situations that arouse feelings of helplessness or anxiety do not tend to increase rates of forgetting or other symptoms (see §5.1). Or again, against transference, we might observe that in their relation to their analyst, subjects remain calm, objective, and friendly over a span of months or years. Grünbaum (1984, 104-26) argues that there is significant evidence showing not only that central psychoanalytic claims are falsifiable, but that Freud (at least) gave up a good number as false on good grounds.

Popper sums up his 1983 discussion of Freud's theory of dreaming by complaining that Freud 'nowhere compares his theory with a promising competitor to it, weighing the one against the other, in the light of the evidence; and he never criticizes it: he has got his theory and tries to verify it; and he makes it fit, as long as possible' (1983, 173). In effect, Popper's objection diagnoses a type of confirmation bias, which involves the selective gathering, weighing or interpretation of evidence that supports one's existing beliefs or favored hypothesis while neglecting or discounting evidence that tells against one's view.[5] Judged by standard psychoanalytic case study reports and key theoretical texts, many psychoanalysts have undoubtedly manifested confirmation bias in their handling and reporting of data. Highlighting

[4] Popper's view of science has fallen into disfavour. It is generally agreed that scientific theories (as opposed to specific hypotheses) are not refuted by single observations or sets of observations, but rather, as Lakatos (1978) argued, through a widening gap between the facts that need to be explained and the explanations offered by the theory, a gap that brings the research programme of the theory to a halt. Making adjustments to a theory and to the 'auxiliary hypotheses' that link the theory to specific empirical predictions, in order to preserve the truth of the theory, is part of normal scientific procedure. However, Popper's objection to psychoanalysis, as I interpret it, can stand on its own.

[5] See Nickerson (1998) for a review.

this fact, or even the dogmatism displayed by psychoanalysts since Freud, is a criticism of the *practice* of psychoanalysts. But if this practice is easily corrected, if nothing in the logical structure and evidence base of psychoanalytic theorizing from clinical data prevents better practice, then we cannot conclude that psychoanalysis cannot be a science, though we may have to admit that changes to psychoanalytic training and critical standards of psychoanalytic work are needed to realize this more widely (§6).

3.2 Confirmation bias

Popper's comment on Freud points us in the right direction for correcting confirmation bias, viz. considering alternative explanations of the evidence to the one we favor (Lord et al 1984; Hirt & Markman 1995). Indeed, this is more effective than attempting to be 'fair and unbiased'. On this approach, we need to discover evidence that distinguishes between alternative explanations, and this provides the grounds for a more objective assessment of the evidence available. It is not enough, as Popper argues, to argue that one's hypothesis is verified; this establishes only that it is *an* explanation of the evidence. What is needed is to show that it is the *best* explanation of the evidence. Inference to the best explanation has good claim to be a key component of scientific method generally (Lipton 2004), and there is no reason why psychoanalysts should not or could not deploy it, even if, historically, they have not done so rigorously.

In arguing for some theoretical claim maintained by one psychoanalytic school, inference to the best explanation would require a demonstration that the claim explains the evidence better than claims from rival schools. Perhaps more importantly for establishing the science of psychoanalysis, psychoanalytic explanations need comparison with non-psychoanalytic theories, e.g. alternative theories of dreaming, neurosis, child development and so on. Now, it may be that many of the phenomena that psychoanalysis seeks to explain simply do not have satisfactory alternative explanations – the data has simply not been adequately theorized outside psychoanalysis – and that the unifying power of psychoanalytic explanation continues to provide reason to accept it. But this can only be established by knowing what alternative explanations there currently are, and the evidence on which they rest.

As already noted, this has implications for psychoanalytic method and training, to which we'll return at the end. For now, we may conclude that working explicitly with inference to the best explanation as described can meet the objection from confirmation bias well enough to confirm the credentials of psychoanalysis as a science.[6]

4. Causal inference

Our final objection is somewhat more complex and involves both theoretical and empirical arguments. It also takes in not just psychoanalysis, but everyday psychological thinking. It will, therefore, take longer to present and resolve.

4.1 Inferring motives

Psychoanalysis, we have said, is a theory of the mind concerned especially with motives. The branch of empirical psychology perhaps most concerned with motives and our attribution of them to ourselves and others is social psychology. But the methods of psychoanalysis and social

[6] Confirmation bias still occurs in scientific enquiry. For example, if a scientist hypothesizes that two events are causally related, this increases the chance that they will find supportive evidence and decreases the chance that they will find disconfirming evidence (Nisbett and Ross 1980, 97). Nickerson (1998, 194) suggests that it is the operation of science as an institution or community that neutralizes confirmation bias as far as it does.

psychology stand in stark contrast, and what social psychology investigates looks to be what psychoanalysis deploys: our commonsense psychological framework for interpreting why people do what they do. If psychoanalysis is to be a science, then we need to consider the place of commonsense psychology of motives in scientific psychology.

I shall assume – as I cannot here defend the claim at any length – that inferences from someone's behavior to their motives is a species of causal inference, because explanations of why someone behaved as they did in terms of motives is a form of causal explanation. They acted as they did in virtue of having the motives they had.[7] We arrive at our attributions of why someone acted as they did by *interpreting* their behavior: we place it in relation to a motive or set of motives which would explain the behavior in virtue of its intentional content and phenomenology (Hopkins 1988). So, if someone gets a drink of water, we infer that they are thirsty. In doing this, we would usually draw on background information, both general and specific. We may bear in mind that people drink even when they are not thirsty, and are sensitive to specific facts in this circumstance that may accord with that, e.g. that the drinking followed a challenge to see how much the person could drink. To take another example, we cannot know whether someone is motivated by *revenge* without knowing more about their beliefs about the previous actions of those they seek to harm. That information may be gleaned from other behavior, including verbal behavior. The interpretation that someone is seeking revenge therefore rests on a range of behavior, and, importantly, integrates it into a pattern. Thus, we attribute many mental states at once, linked by connections of intentional content.

We arrive at our attribution of a motive by means of inference to the best explanation. We form and revise our judgments in light of the evidence available to us, the scope of the explanation (how much behavior it can explain), whether they cohere with other motives which we attribute, the simplicity of the explanation, and how plausible it is, given our background knowledge. That this is the way we proceed has received considerable support from empirical studies in social psychology, envisaged in Kelley's (1972, 1973) ground-breaking work in attribution theory and rendered more complex in studies since (see Gawronski 2004).

Arguably, psychoanalysis works in the same way.[8] Certain familiar patterns, e.g. imagining something to be the case because one wishes it were (Hopkins 1988, §2) or explaining irrational action in terms of unacknowledged desire (Miller 1988, 671), are extended and applied to rather less familiar data. The behavior to be explained comprises the clinical data. Central to producing this data is free association. The connection to any motives, unifying the freely associated material or making sense of its starting point – a thought, a dream, whatever it is – may at first be obscure, but the free association provides further material rich with intentional content, from

[7] Many philosophers and psychologists refer to motives as causes. I believe this raises problems in the theory of agency, and prefer to say that agents are the causes of their actions. However, as with all causes, it is in virtue of some, and not other, of their properties – the motives cited in the explanation – that they cause what they do. (For two recent views of this kind, see Lowe 2007 and Mayr 2011.)

[8] Psychoanalysts may be drawing on a wider range of background knowledge (or theory), and thus guided in making interpretations in ways that differ from commonsense psychology (see Kitcher 1992, Manson 2003). The relative contribution of these background ideas to interpretations in the particular case – rather than the formulation of more general theoretical claims – may be debated, and may differ both historically and from analyst to analyst. Freud's practice differs considerably from current psychoanalysis (Brearley 2008). My interest here is primarily the form that inference takes, so I shall set this issue of background knowledge to one side, though the importance of interdisciplinary work arises again in §§5 and 6.

which, over time, the motives and other mental states of the patient can be inferred. By interpreting free associations, psychoanalysts infer the unconscious causes of the clinical data, led by the connections in intentional content as they appear. As in everyday attributions, such inferences become more justified as more evidence, fitting or correcting the best explanation, is accrued, more of the clinical data explained (Freud 1937, Hopkins 1988). On this reading, given the centrality of interpreting the meaning of behaviour in relation to the subject's motivations, psychoanalysis is a hermeneutic discipline – and so, if it is a science, it is a 'hermeneutic science'.

4.2 The objection

Is this form of inference appropriate and reliable for a science of motives? There are both theoretical and empirical reasons for concern. The theoretical argument comes from Adolf Grünbaum (1984, Ch. 3; 2004). To infer that a factor, X (e.g. a motive), is causally relevant to the occurrence of some Y (e.g. some behavior), we need to show that the occurrence of X makes a difference to the occurrence of Y, and the *only* way to do this is to compare classes of cases. We should compare cases in which X occurs with ones in which it doesn't, and examine whether there is a difference in the incidence of Ys. For example, we compare smokers and non-smokers, and see if there is difference in the incidence of lung cancer. Or again, we should compare cases in which Y occurs with ones in which it doesn't, and see if there was a preceding difference in whether X occurred. So we compare cases of lung cancer with cases of its absence, and see whether there is a difference in whether the patient is a smoker. Without making such comparisons, we can't infer whether or not X is causally relevant to Y, whether smoking is causally relevant to lung cancer.

The same, argues Grünbaum, goes for understanding the causes of people's behavior, including motives. Neither commonsense psychology nor psychoanalysis appear to deploy this form of comparative investigation. By contrast, it is notable that many experiments in social psychology do. Two quick examples (from Nisbett and Wilson 1977):

1) Two groups of insomniacs were given a placebo pill. One group was told that it would produce symptoms of arousal, the other that it caused relaxation. As predicted, the 'arousal' group attributed their initial insomnia to the effects of the pill, and fell asleep sooner than usual (by 28%); the 'relaxed' group attributed their continued insomnia to being especially worried, and took longer to fall asleep (by 42%).
2) Four pairs of identical stockings were placed on a table in a shopping center and passers-by asked to identify which they preferred. Most indicated a preference, citing qualities such as smoothness, hue, and so on. But there was a very strong positional effect, toward the right, with nearly ¾ of subjects preferring the stockings on the far right or position immediately adjacent.

There is, it seems, a difference in the methods used in commonsense psychology and psychoanalysis on the one hand, and scientific psychology on the other, and a question mark over whether the former is appropriate for establishing motives as the causes of behaviors.

The empirical objection comes from social psychology, which diagnoses a number of errors and biases in our attributions of motives to others. Perhaps the most famous is the 'fundamental attribution error': 'the assumption that behavior is caused primarily by the enduring and consistent dispositions of the actor, as opposed to the particular characteristics of the situation to which the actor responds' (Nisbett and Ross 1980, 31). The existence and falsehood of the fundamental attribution error is supported by evidence for a 'correspondence bias' (Gilbert and Jones 1986), our tendency to infer from someone's behavior that it was motivated by a corresponding disposition. In the original experiment establishing this (Jones and Harris 1967), subjects are asked to read an essay defending a controversial moral position, and then asked

whether the author holds the position argued for. In one condition, they are informed that the author had a free choice in which position to adopt. In the other condition, they are informed that the author was *instructed* to write an essay defending the position. They nevertheless tend to infer that the author holds the position defended. Psychoanalysis can be charged with the fundamental attribution error (Nisbett and Ross 1980, 24), because, in its explanations of behavior, it has concentrated almost exclusively on the role of motives.

More generally, experiments show that we can fail to recognize factors, especially situational factors, that are causally relevant to our behavior, and we identify irrelevant factors as influencing what we do or say. When the insomniacs discussed above were asked why they took more or less time to fall asleep, both groups produced rationalizations and rejected the experimenters' explanation in terms of the psychological effects of the placebo pill. And those expressing a preference for one set of stockings over another were clearly identifying features that were not relevant because non-existent, as the stockings were identical, and they almost universally rejected the idea that the position of the stockings influenced their choice. Nisbett and Wilson's (1977, 248) general conclusion is that our explanations of our own and others' behavior, including the attribution of motives, are guided by 'judgments about the extent to which a particular stimulus is a plausible cause of a given response'. The sense of plausibility is often culturally, or sometimes idiosyncratically, informed. Where we have no clear sense of a plausible explanation, we come up with an explanation based on the 'connotative relations' (or meanings) of the stimulus and response. Where the causally relevant factors are 'implausible', as we see this, we fail to identify them, identifying more 'plausible' but in fact irrelevant factors in their stead. Such a method may well seem insufficiently reliable to play a role in a science of motives.

4.3 The reply

Let us start from the empirical case, and work back toward the theoretical one. The empirical case, as presented by Nisbett, Wilson and Ross, is overstated. Three factors, at least, significantly correct the correspondence bias:

1. subjects engage in careful, in-depth processing of information
2. subject are highly 'socially intelligent', i.e. they use a complex schema in attributing motives and are motivated to explain behavior accurately;
3. subjects have high 'negative capability', i.e. they aren't motivated, by the desire to explain, to reach an obvious explanation too quickly or easily (Fletcher 1995, 73-9; Gawronski 2004)

So when we make judgments regarding people's motives casually or quickly, we are liable to the bias. But this result doesn't generalize, and thus we don't hold the general *assumption* claimed by the fundamental attribution error. Furthermore, there is some evidence that what is true of the correspondence bias is true more generally: the typical mistakes relating to plausibility are increased in situations in which we make judgments casually or automatically and decreased when we meet the three conditions specified (Fletcher 1995, 77). We may take it, given their training and the clinical setting, that psychoanalysts fulfill the three correcting conditions. Thus the empirical case against psychoanalytic inference of motives fails.[9]

[9] Many of the causally relevant factors overlooked or rejected in Nisbett and Wilson's experiments are situational, rather than dispositional. However, the situation under which clinical data are produced remains stable and invariant as far as possible, so there are no varying situational factors to appeal to, to explain varying free associations and other behaviors. Psychoanalytic methodology thus removes the potential for many of the errors Nisbett and Wilson identify.

The empirical case doesn't, in fact, undermine commonsense psychology either – or show that it has no place in scientific psychology – for two reasons. First, Nisbett and Ross (1980, 211) are clear that our inferences also very often accurate, and when we make mistakes, we most commonly fail to identify *all* the causally relevant factors, but remain accurate about some. The case of insomnia doesn't show that anxiety doesn't cause insomnia. Thus, the method is not inappropriate, but needs correction in the ways already identified.

Second, social psychology experiments depend upon commonsense psychological inference at a more fundamental level. For instance, they assume that verbal reports (of preference, of causes of insomnia) reflect conscious beliefs with the same content and a desire to answer the question posed. This observation marks the beginning of our response to Grünbaum's theoretical objection.

Grünbaum's model of causal inference cannot apply to inferences about motives. Suppose our question is 'was Fred's getting a drink caused by his desire to get a drink?'. On Grünbaum's model, we need to identify cases in which people have a desire to drink and those in which they don't, and see whether in the first class of cases, people are more likely to get a drink. Or again, we should identify cases in which people drink and ones in which they don't, and establish whether people in the first class are more likely to have the desire to drink. But this supposes that we can meaningfully identify and talk about the desire to get a drink without already supposing that it typically causes people to get a drink. On every widely accepted philosophical account of desire, what makes a mental state a *desire*, rather than some other type of state, is precisely the pattern of its relations (causal and/or normative) to behavior and other mental states. What gives a desire the *content* it has is also determined (in part) by such relations. There is nothing to the desire to get a drink being the mental state it is, viz. a *desire* and a desire *to get a drink*, in the absence of any such relations. So to understand what a desire is, and what it is a desire for, is already to understand it in a (causal) explanatory relation to certain behavior. We cannot suppose that it might turn out – in general – to be causally impotent or to cause people to want to go to sleep.

This conceptual connection between mental state and behaviour explains why social psychology must itself rest upon commonsense psychological inference, since the concepts which it deploys are defined in this context (Rudder-Baker 1999; see also 1987, Ch. 7). And given this conceptual connection between motives and behavior, we have a defeasible, prima facie reason for inferring, from someone's getting a glass of water that she wants a glass of water. Of course, we can be wrong about this, and both background and situational factors may defeat the inference. But our method of inferring motives takes account of this. Given that there are ways in which we may correct the biases to which we are susceptible, the theoretical objection to commonsense psychological inference as a method of inferring motives, and to its deployment within psychoanalysis, fails.

4.4 A significant restriction

The argument that we have rehearsed defends the possibility of inferences in psychoanalysis that move, as commonsense psychological inferences do, from current patterns of behavior to current motives. These are inferences within the clinical theory of psychoanalysis, as identified in §1. Etiological and metapsychological inferences, however, do not receive support from the arguments we have rehearsed. Inference to the best explanation in commonsense psychology concerns itself with the relation between behavior and motives, but it is unclear that it can be reliably extended to accounts of the origins of motives or overarching structures of the mind. There are potentially many causally relevant factors in the formation of long-term dispositions

that may not be picked up by our commonsense psychological approach, because they are 'implausible' (from the commonsense point of view). If we are to justify such inferences, we will need to appeal to more than inferences drawn by interpretation of the clinical data. The arguments regarding causal inference, then, support the conclusion independently reached in the case of suggestion.[10]

5. Scientific evidence for psychoanalysis

We noted at the end of the discussion on suggestion that the solution proposed is not definitive. Results generated independently of the clinical method would provide additional confirmation that the corroborative clinical results are sound. This finding would also help allay fears of confirmation bias, since different disciplines start from alternative hypotheses and ways of conceptualizing the field. The chances of a highly flawed method producing results that are consilient with results from other fields are slim. What follows are three very brief accounts of a very few of the results supporting such a finding for the psychodynamic model of mind. (More complete accounts and additional references can be gleaned from Masling and Bornstein 1983-1998; Westen 1998; Valone 2005; Wallerstein 2006; and Eagle 2011.)

5.1. The clinical data

We have discussed the three methodological objections to psychoanalysis being a science assuming the traditional clinical methodology of defending claims on the basis of case studies. But the data may be subjected to various forms of empirical and statistical analysis,[11] and over the last forty years, a number of researchers have done just this (early examples include Dahl 1972; Luborsky 1977; Gill 1982; and Gill & Hoffman 1982). In this kind of work, the researcher is not, or not only, the therapist, and uses the standard method of comparing classes of cases when drawing causal inferences. The results confirm many of the essential claims of the psychodynamic model (e.g. Dahl, Kächele, & Thomä 1988; Luborsky & Luborsky 2006). As an example: Luborsky (2001) sought to identify the preconditions for recurrent symptoms, such as momentary forgetting, in psychotherapy. He examined the recurrence of seven symptoms manifested across seven audio-recorded analyses. The occurrence of symptoms was sampled in the context of the preceding 30-50 words and the preceding 300-400 words. These contexts were examined for psychological conditions they have in common. 12 differentiating qualities were identified across the cases, and these were then rated for significance and intercorrelation, and five identified as most significant (viz. hopelessness, lack of control, anxiety, feeling blocked, and helplessness). A control group of contexts in which the symptoms were not manifest, chosen by arbitrary principles, was compared. Independent judges and a variety of scoring systems were deployed in generating the results. The five psychological conditions for symptom onset were then compared with those predicted by a range of classical psychoanalytic theories. The result

[10] The disagreements between psychoanalytic schools regarding etiology and metapsychology is evidence either that everyone outside one's favoured viewpoint is incompetent in drawing conclusions from clinical data or that clinical data doesn't determine the correct metapsychological theory. I prefer the second, more humble conclusion. It is noteworthy that as psychoanalysis has developed, so greater emphasis has come to be placed on working in the transference in the present, and less emphasis has been given to a reconstruction of the origin of neurosis. The usual reason offered for this shift is that the clinical data cannot reliably support historical reconstructions, but can give insight into present conflict.

[11] There is an ongoing debate over whether recording clinical sessions may influence the data; and whether more data is available to the analyst 'live' than any recording can capture. It may be true that a fine level of detail and accuracy regarding the individual patient is lost, but there is good reason to think, from the work that has already been done, that enough remains – and remains the same – for general hypotheses to be tested.

was a high match (2001, 1148). The psychodynamic model predicts the clinical data.

A number of databases of clinical material now exist, open to scrutiny and collectively running to tens of thousands of hours. It should be possible, therefore, to discover the extent to which the clinical data systematically differ in ways that would lend support to the school of the analyst, and thus have some hard evidence regarding the operation of suggestion regarding metapsychological and etiological claims. I am aware of only one study of this kind (Fischer & Kächele 2009), which finds suggestion effects at the *beginning* of therapy, but they *diminish* over time. The authors suggest that this is a result of the patient's becoming more independent of their analyst in the course of therapy.

5.2 Consilience

Many of the central theoretical commitments of the psychodynamic model receive support from other quarters. That there is *unconscious mental processing* is a commonplace now. The occurrence of *unconscious emotion* is likewise becoming more widely accepted (see Feldman-Barrett, Niedenthal, & Winkielman 2005; for a summary of the neuroscientific evidence, see Winkielman, Berridge & Sher 2011; for philosophical discussion of the very idea, see Lacewing 2007). For example, it has been shown that positive and negative affective reactions can be elicited subliminally, while a person is completely unaware of any affective reaction at all, and that such unconscious reactions can go on to influence behavior (Winkielman, Berridge & Wilbarger 2005). Other work demonstrates the existence of *unconscious motivation* more generally (e.g. Shevrin et al 1996, Westen 1999, Bargh 2005). The work of Elizabeth Andersen and colleagues (Andersen & Glassman 1996; Andersen & Chen 2002; Andersen & Thorpe 2009) has also shown that past emotions and patterns of relating ('significant other' representations) can unconsciously influence present emotions and behavior in relationships ('*transference*'). Westen & Gabbard (2002a, 2002b) provide a useful review of developments in cognitive neuroscience, especially the role of implicit memory, that support psychodynamic claims about *conflict* and *transference* via the idea of 'associational networks', operating outside and independently of consciousness to influence judgments, decisions, emotions, and behavioral reactions. Such networks may involve connections between representations and affects, e.g. that authority figures will always be angry; or they may involve wishes that others behave in certain ways; or they may involve beliefs, fantasies, and expectations about interpersonal relations, e.g. that if one expresses anger, others will withdraw their love.

Cramer (2006) provides an excellent review of the empirical evidence for *psychic defenses*. They have been established by a variety of empirically validated measures, which may be observational, involve coding narrative material (e.g. from Thematic Apperception Tests or clinical interviews) or self-report questionnaires (Cramer 2006, 15-18; Conte & Plutchik 1995). The use of defenses – which type and how much – changes with age (Cramer 2006, 38-9), increases under conditions of stress and anxiety (whether internal or external) and decreases the subjective experience of anxiety (142-4, 159-60). Excessive use of neurotic defenses, and the use of age-inappropriate immature defenses, is associated with psychopathology (235-6, 253-4), while the use of mature defenses soon after adolescence predicts positive adjustments to life's challenges later on (210-11). However, almost all of us use distorting (and not only mature) defenses to a greater or lesser degree. Vaillant (1993, 132: Table 4) provides evidence that of those in the top 20% on a scale of psychosocial adjustment at 65 years old, 50% still use less than mature defenses, and the percentage for those lower on the scale is considerably higher. Cramer remarks (2006, 224) that 'within any normal, nonclinical group we might expect to find individuals who possess more or fewer of the characteristics that define a psychological disorder'. These may be individual symptoms, e.g. depressive tendencies, phobias, or pathological aggression, or personality

disorders, e.g. antisocial traits or narcissism. The evidence is that these are all associated with the use of defenses (235). This is all just as the psychodynamic model would lead us to expect.

5.3 Outcome studies

It has been a typical finding of outcome studies that psychodynamic therapy is no more (but no less) effective than other forms (e.g. Wampold 2001). However, the vast majority of such studies have considered only short-term psychodynamic therapy (up to six months). Evidence regarding the effectiveness of *long-term* psychodynamic psychotherapy (over 50 weeks) has only begun to emerge in the last ten years. For example, Leichsenring and Rabung (2008) performed a meta-analysis of 23 studies with a total of 1053 patients with complex mental disorders, including personality disorders, chronic or multiple mental disorders, and depressive and anxiety disorders associated with these. The 23 studies included 11 random controlled trials and 12 observational studies, all selected for their prospective design and reliable outcome measures. Overall, patients undergoing long-term psychodynamic psychotherapy were better off than 96% of patients undergoing shorter therapies. Or again, Knekt et al (2007) randomly assigned 326 out-patients with mood or anxiety disorders to long-term psychodynamic psychotherapy, short-term psychodynamic psychotherapy, or solution-focused therapy. Standard measures for depression and anxiety were deployed at the outset, and after each year for three years. Long-term psychodynamic psychotherapy, while no better than other forms at the outset, produced outcome measures of depression and anxiety 14-37% lower than the two other therapies at the three year follow-up. Given that other therapies tend to perform only as well as short-term psychodynamic psychotherapy, we may infer that long-term psychodynamic psychotherapy outperforms other therapies as well. But it is notable that its benefits are not apparent in the short-term. Similar and updated results are reported in Leichsenring (2011), Knekt et al. (2011), and de Maat, de Jonghe, Schroevers, & Dekker (2009). From these and other studies (see Shedler 2010 for a review, and Levy, Ablon & Kächele 2011 for an excellent collection), we may tentatively claim that, for complex mental disorders and mood and anxiety disorders at least, long-term psychodynamic therapy outperforms other therapies over time.

In summary, the psychodynamic model of the mind is supported by the clinical data of psychoanalysis, by recent empirical work on unconscious emotion and motivation, implicit memory and models of relationship, psychic defense and therapy. This confirmation of results by multiple methods confirms the thought that there is a science of psychoanalysis.

6. The science of psychoanalysis

Our discussion of the three methodological objections to considering psychoanalysis as a science led us to the following conclusions:

1) that corroborative results from across psychoanalytic schools, and preferably beyond, are necessary to meet the charge of suggestion, and that the greater variety of evidence that supports psychoanalytic claims, the better justified they are;
2) that psychoanalytic researchers need to consider alternative explanations, from both other psychoanalytic schools and non-psychoanalytic theories, in order to deal with the possibility of confirmation bias; and
3) that to establish causal inferences that extend beyond an account of current motives for current behavior, e.g. inferences regarding the etiology of motivational structures, requires us to use other methods in addition to interpreting clinical data.

All three point to the need for psychoanalysts – when developing psychoanalytic theory, for we have not here been concerned with clinical practice – to be aware of and engage in debate with non-psychoanalytic theories. For example, to assess an inference as the best explanation, we

need knowledge of the alternatives, and many of these may be generated outside the consulting room. What the alternatives are constantly changes – new evidence may overturn a previous explanation or a theoretical advance may generate a more powerful one. We also need knowledge of relevant evidence generated outside the consulting room. Such evidence may undermine, corroborate or improve upon a hypothesis that we have developed on the basis of the clinical data. A wider evidential base is especially crucial for metapsychological and etiological claims, which, I have argued, cannot be established by inference from clinical data alone.[12]

Such engagement by psychoanalytic researchers with empirical methods or non-psychoanalytic theories should not be seen as 'not doing psychoanalysis'. Such a conclusion would require an illegitimate separation of clinical psychoanalysis from the development of the psychoanalytic theory of the mind. The idea that psychoanalysts don't do empirical research is, in any case, out of date, as the discussion of §5 shows. (A letter by Mary Target to the *New Scientist*, 27 October 2010, responding to an ill-informed article on the scientific status of psychoanalysis by Mario Bunge, states 'The 54 signatories to this letter include distinguished researchers in psychoanalysis in the science faculties of leading world universities, who have acquired major public grants and have published papers in high-impact, peer-reviewed scientific journals.') Psychoanalysis has always been interdisciplinary, supplementing its clinical findings with ideas from cognate disciplines (Kitcher 1992). This makes the need to engage with contemporary developments all the more pressing, as the remarks on inference to the best explanation above indicate. Evidence and explanations change, but can become frozen within psychoanalytic theory if it is not updated over time.

None of this makes the clinical method and the clinical data irrelevant to the justification of psychoanalytic claims. As noted in the introduction, such a result would support the view that psychoanalysis is not a science, as it cannot contribute to the validation of its own theory. But we have established that the clinical method is legitimate, defensible against the objections of suggestion, confirmation bias, and inappropriate causal inference, at least in relation to the psychodynamic model of mind. Together with the data on which it draws, it makes an independent contribution to theoretical developments and their justification. As a result, the relation between psychoanalytic and non-psychoanalytic evidence and explanations is reciprocal: each is beholden to take account of the other in presenting arguments for being the best explanation for the phenomena explained.

Nevertheless, the claim of psychoanalysis to be a science would be strengthened if awareness of a) the methodological pitfalls and means to avoid them, and b) alternative theories and their evidence bases, were more widespread. This can be directly addressed by the education and training of psychoanalysts, which needs to encourage psychoanalysts to test and compare explanations and theories, which in turn requires both that an atmosphere of intellectual enquiry and rigorous questioning is engendered and that trainees are provided with an education that covers a range of theories. That psychoanalysts are painfully aware of these requirements is exemplified in Kernberg's series of articles (2000, 2006, 2007, 2010) that provide seminal contributions to, and helpful overviews of, the debate so far. He argues that many psychoanalytic training institutes fail to teach research skills and actively oppose original thought, and they ignore both the contributions of other schools of psychoanalysis and relevant information in non-psychoanalytic disciplines. He advocates a series of significant reforms to psychoanalytic training and education, including the abolition of the training analyst. Some of these are already

[12] A nice example here is provided by the results of attachment theory, as relevant to the developmental theories of psychoanalysis (see e.g. Eagle 1997; Holmes 2000; Fonagy 2001; Fonagy & Target 2003, Ch. 10).

being put in practice, in Atlanta, Philadelphia, Buenos Aires, Berlin and Paris. Such developments are assurance that the science of psychoanalysis has a bright future.

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