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On the scientific prospects for Freud's theory of hysteria

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ABSTRACT

Hysteria (or conversion disorder) is once again attracting concerted scientific attention. This paper looks at the extent to which recent scientific research supports Freud's theory of hysteria, which posits that repressed impulses are converted into physical or behavioral symptoms. Specifically, it looks at two prominent empirical studies, representing the most rigorous direct efforts to date to test Freud's key ideas about hysteria, in conjunction with an important new theoretical account. The empirical studies are Nicholson et al.'s (2016. Life events and escape in conversion disorder. *Psychological Medicine*, 46(12), 2617–2626.) survey-based study, which examines the impact of life events on hysteric patients, and Aybek et al.'s (2014. Neural correlates of recall of life events in conversion disorder. *JAMA Psychiatry*, 71(1), 52–60.) brain-imaging study, which looks at the neural correlates of the recall of such life events. The theoretical account is Edwards et al.'s (2012. A Bayesian account of "hysteria". *Brain*, 135(11), 3495–3512.) Bayesian account of hysteria, in which somatic symptoms are seen as the result of the entrenchment of prior expectations that appear to explain (by predicting) otherwise unexplained bodily sensations. The conclusions of the present paper are that the empirical studies offer considerable evidence in support of key aspects of Freud's theory of hysteria, that this theory is compatible with the Bayesian account of hysteria, and that reservations about Freud's theory expressed by the authors of the Bayesian account are allayed by the empirical studies.

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Introduction

Freud's theory of hysteria has been highly influential in the medical understanding of hysteria, a disorder characterized by neurological symptoms, such as paralysis and blindness, that cannot be explained by a known neurological disease. Indeed, it is due to this theory that hysteria was redesignated as *conversion disorder* (CD) by the DSM III in 1980 (henceforth, I will use "CD" and "hysteria" interchangeably).¹ However, though the influence of Freud's theory of hysteria persists, it remains controversial, like so much of psychoanalytic theory.

There have been numerous and diverse challenges to Freud's ideas about hysteria over the last few decades. For example, it has been claimed, and widely believed, that hysteria is not as prevalent as Freud saw it (Webster, 1995). It has also been claimed that the foundations of Freud's theory are suspect. Historians have cast doubt on the credibility of the case study that first inspired the theory, Breuer's famous study of Anna O., while philosophers have cast doubt on the reasoning by which Freud and Breuer inferred that unconscious thoughts were causes of symptoms in cases like Anna O.'s (Grünbaum, 2006). Others have expressed reservations about Freud's later ideas. It has been claimed

that Freud overgeneralised his findings, that his emphasis on sexuality is misleading, and that the continuing influence of his views present an obstacle to a better understanding and treatment of this mysterious illness (Edwards, Adams, Brown, Parees, & Friston, 2012).

Many of these challenges have already been met. The challenges concerning Breuer's work with Anna O., for example, have been undermined by more sophisticated historical examination (Skues, 2006),² and I have argued elsewhere (Michael, 2018) that the philosophical arguments are based on misleading logical analyses. More fundamentally, the challenges concerning the prevalence of hysteria have been authoritatively overturned. It is now accepted by leading neurologists that conversion disorder is extremely common (Stone, Hewett, Carson, Warlow, & Sharpe, 2008). For example, Feinstein (2011) reports an incidence of 20–25 percent among hospital patients of symptoms of conversion, up to a quarter of which meet the full criteria for the disorder, while Nicholson, Stone, and Kanaan (2011) remark that patients with such symptoms are "as common in neurology settings as multiple sclerosis or Parkinson's disease" (p. 1267).

But some of the challenges to Freud's ideas remain. Leading researchers have expressed doubts about the

claim that most hysterical symptoms owe to a psychological cause (Sharpe & Faye, 2006; Stone & Edwards, 2011), about the plausibility of a conversion mechanism (Brown, 2004; Stone, LaFrance, Levenson, & Sharpe, 2010), and about the role of repression (Edwards et al., 2012). Some argue that the attempt among therapists to uncover events that may have provoked a symptom can be, not only ineffective, but also dangerous to the patient (ibid.).

Against such doubts, however, recent work has emerged that appears supportive of Freud. This includes a survey-based study that suggests that psychological stressors are present for the vast majority of hysterical symptoms (Nicholson et al., 2016), and an imaging study that indicates that suppressed thoughts play a prominent role in hysteria (Aybek et al., 2014). Moreover, a new theoretical account of hysteria (Edwards et al., 2012) based on the Bayesian brain hypothesis, a potentially groundbreaking overarching theory of brain and mind that is garnering much interest among neuroscientists and philosophers, offers a possible vindication of the theoretical principles underlying Freud's conversion theory of hysteria. This is therefore an opportune time to revisit Freud's views on hysteria and to offer an initial assessment of the extent to which, if any, such new work supports them.

In what is to come, I will first provide a short review of Freud's theory of hysteria, aimed in particular at clarifying what it does and does not assert, as this is often a source of misunderstanding. I will then examine the recent attempts to directly test key Freudian claims. This will be followed by an exploration of how Freud's main ideas about hysteria – in particular the essential role played by repression – may be accommodated by the Bayesian account mentioned above. The overall message is that the recent scientific work on hysteria considered in this paper presents a favorable picture of the prospects of Freud's theory as a dominant, if incomplete, theory within the (mainstream) science of hysteria.

Freud's theory of hysteria

In reviewing Freud's theory of hysteria, it is best to present the theory through its developmental stages, since the theory developed over a period of time and a proper appreciation of it requires an understanding of that historical development. The beginning lies in the work of Freud and Breuer in the 1880s and 1890s, described in their *Studies on Hysteria*.

There are two parts to the theory of hysteria presented in *Studies*. The first, which I will call the *Freud-Breuer Pathogenicity Hypothesis (FBP)*, is as follows (Michael, 2018):

FBP: For many hysterical symptoms, (a) an unconscious memory is a cause of the symptom, and (b) this memory is unconscious due to an ongoing repression.

A few remarks about this hypothesis are in order. First, the hypothesis is modest in scope – as the authors write, “our investigations reveal, *for many, if not for most*, hysterical symptoms, precipitating causes which can only be described as psychological traumas” (1893, *S.E.*, II: 6; my emphasis).³ Second, the hypothesis is modest in its explanatory ambitions. While the authors believe that a recent unconscious memory is the chief operative cause – that is, the driving force behind the symptom – they do not preclude additional causal factors, including not only predisposing causes, such as heredity, and concurrent causes, such as illness (cf. 1895, *S.E.*, III: 123–139), but also possibly other operative causes. This is important as it allows for the aetiological elaborations that Freud was to make in future developments of the theory. Third, the key terms “unconscious” and “repression” are necessarily vague. This in itself should not be considered a shortcoming, as it is in line with what happens in science more generally – for example, the precise meaning of the concepts “species” and “gene” in biology are still being debated. Fourth, the authors specifically stipulate that it is the *memory* that is the cause and not the traumatic event that it is a memory of (1893, *S.E.*, II: 6). In other words, it is a representation of an event, hence, broadly speaking, an *idea*, that is the operative cause, rather than the experience of the event itself.⁴

The second part of Freud and Breuer's theory was an account of the process by which the unconscious memory causes the symptom. On their view, the unconscious memory is associated with strong emotion that has not been appropriately expressed, hence remains “energetic.” A symptom is brought about by a process that involves converting this emotional energy into the symptom by means of an unconscious association of ideas that establishes a connection between the memory and the symptom (1894, *S.E.*, III: 49, 60). Underlying this process is a deeper theory about how the mind works. Freud and Breuer believe there is a quantity of *something*, which they sometimes call “quota of affect,” sometimes “sum of excitations,” and sometimes “psychical energy,” that is subject to a particular general principle. This general principle is the “principle of constancy:” the mind endeavors to keep the quantity low and constant by “disposing associatively of every sensible accretion of excitation or by discharging it by an appropriate motor reaction” (1893, *S.E.*, I: 153–54). Thus, excitation can pass from idea to idea, until it facilitates some form of action, whereby it diminishes. These “metapsychological” ideas are fundamental to all of Freud's psychoanalytic theorizing.

The Freud-Breuer theory remained the core of Freud's theory of hysteria. However, one can see other elements emerging even in *Studies*. Freud was, in particular,

unsatisfied with the idea that a single recent event in the patient's history could fully account for a symptom. The main problem he saw was that such events often lacked sufficient "traumatic force" (1896, *S.E.*, III: 193) – that is, these events often appeared relatively trivial, and it was difficult to understand why they should engender a high degree of trauma. It seemed that there must be something deeper underlying the patient's excessive emotional reaction.⁵ Furthermore, Freud found that, though uncovering an unconscious memory of a recent event was sometimes sufficient to cure a symptom, oftentimes the cure was only temporary, and the symptom would return soon enough. These considerations, and others besides, led Freud to push his own adaptation of Breuer's technique, his method of analysis using free associations, further. In this way he was, on his view, able to discover deeper sources of the energy that was driving the symptom (*ibid.*, 197; 1906, *S.E.*, VII: 273).

Freud's investigations brought him to the view that there were chains of memories that led ever further back in time. He initially thought the chain began with a sexual event in puberty. However, he later concluded that this event too did not have enough traumatic force to be the ultimate source of the energy driving the symptom. Instead, digging deeper, Freud came to the view that childhood sexual experiences were the ultimate cause. These were, according to Freud, always passive sexual experiences – rape or other forms of sexual abuse – initially by an adult, but perhaps also by other children, such as older siblings or cousins. These experiences had been forgotten by the child, but then unconsciously revived by the subsequent sexual event in puberty. The energy thereby released could transfer, via an association of ideas, to new experiences, resulting in the excessive (though suppressed) emotional over-reaction that precipitated the formation of a symptom. He thought this account, rooted in the so-called "seduction theory" of childhood sexual abuse, general to all hysteria (1896, *S.E.*, III: 191–221).

There remain two further important developments to Freud's theory. One was a radical change that augured the next and most important stage in the evolution of psychoanalysis. Realizing that the psyche does not differentiate between true memory and fantasy, Freud came to the view that he had overstated the "seduction theory" (1906, *S.E.*, VII: 274). Not all, and perhaps not even most, hysteria was due to an actual experience of sexual abuse. Instead, the "memories" he was uncovering were usually childhood fantasies of a sexual experience (*ibid.*, 278; 1908, *S.E.*, IX, 159–66). This realization contributed to Freud's new ideas about infantile sexuality, which he was to develop in *Three Essays on the Theory of*

Sexuality and other works. The childhood fantasy (or fantasies) thus came to be associated with the Oedipus complex and the child's sexual wishes toward its parents (1900, *S.E.*, IV: 260–64).

The revised theory of hysteria that came with these new insights proposed that what lay behind symptoms was the intensification of a prevalent kind of intrapsychic conflict. In the hysteric, strong infantile sexual impulses run up against a powerful aversion-based need to repress such impulses. It is these repressed libidinal impulses that are the driving force behind hysterical symptoms (1905, *S.E.*, VII: 164; 1909, *S.E.*, XI: 24). External events like childhood sexual abuse would vastly increase the strength of both the libidinal impulses and the repressive force against them, hence making hysterical illness significantly more likely, but they are no longer, for Freud, necessary precursors. Indeed, Freud's new ideas blur the distinction between healthy people and hysterics, as everyone is subject to the same intrapsychic conflict, and differences in its manifestation are mainly a matter of degree – it is the strength of the libidinal impulse and the aversion to it that most predisposes one to hysterical symptoms (1906, *S.E.*, VII: 276–78).

Once again, though, Freud refuses to rule out other causal factors, such as hereditary predisposition and illness (*ibid.*, 279). Also, precipitating traumatic events, such as the kind uncovered by Breuer and Freud in *Studies on Hysteria*, remain the most important proximate causes of symptoms (1896, *S.E.*, III: 214). A traumatic event ideationally associated with the intrapsychic conflict brings up a large amount of the libidinal energy of the repressed impulses as part of the (suppressed) emotional reaction to the event. When the memory of this event is later repressed on account of its aversive associations, its compounded emotional energy becomes dissociated from it, thereby forming the ready source of excitation that finds an outlet through the symptom.

Freud's second innovation was less radical, but nevertheless of theoretical and clinical importance. This was the idea that hysterical symptoms may have both primary and secondary gain. Primary gain has to do with finding an outlet for the libidinal energy released by the trauma. Secondary gain has to do with some further practical benefit to the patient from the ensuing symptom. For example, being incapacitated by the symptom may have the benefit of freeing the patient from the obligation of work. Such secondary gain reinforces the symptom, making it harder to remove (1909, *S.E.*, IX: 231–32; 1905, *S.E.*, VII: 43–44).

In conclusion, through this historical review we see what Freud's theory is and what it is not. Freud's theory is not a comprehensive theory of the aetiology

of hysteria (cf. 1926, *S.E.*, XX: 111–12). Rather, it is a theory of the sources and byways of the psychical energy that drives the symptom. It allows, but does not focus on, additional causal factors, such as hereditary predisposition and illness. Neither does Freud's theory provide a detailed account of the mechanism of conversion, focusing instead on what is most therapeutically relevant. It is, in its mature form, a general theory of hysteria, purportedly explaining the force behind all hysterical symptoms; though, according to Freud, confirmation of this can come only through the clinical methodology of psychoanalysis.

The empirical evidence

In attempting to evaluate the scientific prospects of the above-described theory of hysteria, the first question that arises is whether there is any empirical evidence in support of this theory. It is therefore with this question that we begin our evaluation, which we will do by honing in on the two empirical studies described in the introduction.

Though a great deal of research on CD has been conducted since the millenium (see Carson et al., 2012, for a review), there have been few studies directly addressing Freud's theory (Nicholson et al., 2011, p. 1270).⁶ Nicholson et al. (2016, p. 2618) observe, in particular, that, prior to their own work, there had not been any controlled studies on the impact of life events on CD patients. Also, it is only fairly recently that the prospect of reliable extra-clinical evidence of the involvement of repression in CD symptoms has emerged, following the influential imaging work of Anderson et al. (2004). Aybek et al. (2014) provide the only imaging study to date that directly addresses the issue, as their study is designed specifically to examine the neural correlates of the recall of life events judged to be of causal significance in CD according to the study by Nicholson et al. (2016). Thus, our focus on these two studies is justified, since they constitute the most rigorous direct efforts to date to test Freud's key ideas about hysteria.

The more recent of these papers is Nicholson et al. (2016). It represents the most meticulous attempt yet to test the idea that the symptoms of CD are due to psychological causes. Though this idea is widely held among clinicians, Nicholson and colleagues argue that it has not been sufficiently scientifically validated. Their study seeks to redress this deficit through a survey that examines life events in the lead-up to symptom onset in CD patients. The prediction of the "psychological model of CD" is that CD patients compared to controls would have experienced significantly more distressing life events in the study period, and increasingly so in

the period approaching symptom onset. This prediction is borne out by the results of the study.

The study is based on the Life Events and Difficulties Survey (LEDS; Brown & Harris, 1978), a semi-structured interview that aims to identify events in a subject's life with the potential to cause stress to the subject. Each such event is evaluated as to severity by a panel blinded to the condition of the subject (e.g. whether a CD patient or control), thereby offering an "objective" rating of the traumatic potential of the event. LEDS performs well on numerous assessments of validity and reliability as a psychological instrument. Nicholson and his colleagues used this instrument on 43 motor CD patients, 28 depression patients, and 28 healthy controls, in order to investigate differences in the number and degree of stressors between these groups in the relevant time period (a year before symptom onset for CD and depression patients, two years before the interview date for the healthy controls). The events were also evaluated (by a panel, blinded as above) according to their "escape potential," that is, the degree to which the formation of a neurological symptom might enable the subject to avoid or reduce the impact of a stressor – a rating directly motivated by Freud's idea of secondary gain.

The results of the study show that CD patients have significantly more severe and escape events than controls (e.g. 58% of CD patients had high escape events, as opposed to 7% in healthy controls and 36% in depression cases),⁷ and this difference increases greatly in the time period leading up to symptom onset. It was also found that "key events," that is, events judged by a panel to have high potential causal relevance due to severity, escape potential, and closeness to symptom onset, were present in 91 percent of the CD patients. For 88.4 percent of the CD patients, these key events had not been documented by the clinicians dealing with the case prior to the study. It was further found that sexual abuse was significantly more prevalent in CD patients than controls (41.9 percent versus 14.2 percent in healthy controls), and that many of the key events were related to previous sexual abuse (over one third of female patients reported such events).

These results offer support for several aspects of Freud's theory of hysteria. Most obviously, the results offer support for the general idea that hysteria is due to psychological causes, and, more particularly, psychological causes relating to traumatic life events. The results also show that many of those events were thematically relevant to the symptoms – as the authors state, "it was possible to make a very convincing psychological formulation for the majority of patients on the basis of stressors identified in ... the few weeks and months

before the onset of the symptoms" (p. 2624). Thus, these results provide support for the idea expressed by *FBP* that traumatic events are causally relevant to hysterical symptoms.

There are at least two other aspects of Freud's theory for which the study offers favorable results. First, in line with the findings of other studies, it offers support for Freud's emphasis on sexual trauma. As the authors assert, "we found support for the importance of sexual abuse as a remote risk factor for CD through elevated rates of abuse and from the subgroup of patients for whom this abuse was clearly re-activated around symptom onset" (p. 2624). This finding is in accord with Freud's idea that the traumatic force driving (many) hysterical symptoms comes from actual sexual abuse.⁸ Second, the study offers support for Freud's idea about secondary gain. Since the results show that the CD patients had suffered significantly more severe escape events in the study period, with the degree to which this is so increasing dramatically with closeness to symptom onset, it is in accord with Freud's idea that hysterical symptoms often provide a means for the patient to escape from a stressful outcome, and that this may play a causal role in the nature, degree, and persistence of a symptom.

Some aspects of the results of this study could be interpreted as contrary to Freud's theory. For example, the authors report that no evidence was found for repression of the degree of the trauma, though they admit their methodology in this respect was coarse (p. 2624). Also, the authors found that 9 percent of CD patients did not have identifiable key events. This result, however, should not be considered particularly worrying to the Freudian theory for several reasons, though primarily two. First, according to *FBP*, it is only "at least many" symptoms that have identifiable traumatic causes. Second, and more importantly, the study focuses on events that can be consciously recalled (though it should be remembered that the vast majority of key events were not identified by the initial clinical assessment of the patient),⁹ whereas *FBP* concerns events that cannot ordinarily be recalled.

Thus, overall, the results of this study constitute valuable, if limited, empirical support for Freud's theory of hysteria. They do not establish beyond doubt that traumatic events are causally relevant to CD symptoms, but they show this to be very highly plausible. And, though they do not support a universal aetiology, they corroborate the importance of sexual trauma in CD. These results, therefore, are broadly supportive of Freud's theory, though not a full or direct confirmation of it.

This leads us to the second study, reported in a paper published in the prestigious *JAMA Psychiatry* (Aybek et al., 2014). This imaging study offers stronger and

more direct evidence in support of Freud's theory, including its key claim about the pathogenicity of repression. The study is based on a subgroup of the cohort used in Nicholson et al. (2016). Specifically, 12 patients with motor CD were recruited to undergo fMRI scans, along with 13 healthy controls. All subjects undertook recall tasks (involving giving true or false responses, via a button press, to a series of statements) in relation to three conditions: recall of a severe non-escape event (as measured by LEDS), a severe escape event, and a neutral event. The Freudian expectation would be that there would be significant differences in brain activation between the CD patients and controls. This is indeed what was found.

Specifically, it was found that, for CD patients in the escape condition, there was increased activity of the left dorsolateral prefrontal cortex (DLPFC) and decreased activity of the left hippocampus, precisely the pattern of activation found in previous research to be associated with thought suppression (Anderson et al., 2004). At the same time, it was found that "escape events elicited significantly longer [reaction times] than neutral events and were perceived as less upsetting than severe events, although both types of events were of matched objective threat" (Aybek et al., 2014, p. 56), which can be interpreted as further evidence of thought suppression in relation to such events.

These results cohere well with the hypothesis that repression plays a role in hysteria. As the authors put it, their findings are "consistent with memory suppression," and, more specifically, with the "mechanisms of 'direct suppression'," in which "the conscious recollection of an unwanted memory (mediated by the hippocampus) is disrupted by top-down regulation (mediated by the DL[P]FC)" (p. 56). As such, their study "gives greater justification to an etiological interpretation, namely, that this prefrontal dysregulation arises from, or is prompted by, a painful memory" (p. 57). The results also offer additional support for Freud's idea of secondary gain, as it was with escape events that the above-described patterns of activation were associated.

Furthermore, the study produced results that cohere well with Freud and Breuer's conversion theory. Recall that this is about the process by which affective energy generated by trauma is converted, via an association of ideas, into the innervations that drive the symptom. Aybek and colleagues found that, while areas of the brain normally involved in emotional regulation were less active for the CD patients, the "connectivity between the amygdala and motor regions" of the brain that might be associated with symptoms – and for which there was increased activity in CD patients – was higher (p. 59). This finding indicates emotional

modulation of motor activity, which corresponds nicely to the conversion theory.

One significant limitation pertains to this study as a test of Freud's theory, in that the events studied were ones that patients were able to recall during the interview and the fMRI tasks, whereas Freud focused on events the memory of which was fully unconscious. As such the inferred thought suppression in the study appears not to correspond to full repression. This should not, however, be taken as evidence that thought suppression and Freudian repression are fundamentally different processes, as some commentators would want to have them. In *Studies* it is clear that "repression" refers to a process that is, at least initially, conscious and intentional (1893, *S.E.*, II, 10), very much in line with the thought suppression described by Anderson et al. (2004) from which Aybek et al.'s study draws its neural assumptions. Freud's further developments of the notion of repression as an unconscious process are continuous with this initial understanding (Erdelyi, 2006). Instead, I suggest that the lesson of the above study is that even events the memory of which is not yet fully repressed show signs of repression-in-progress. However, due to practical limitations, it leaves untouched the possibility of the existence of fully repressed memories.

To conclude, let us return to the question of to what extent the results of the above studies support Freud's theory of hysteria. We must of course treat the results with an appropriate degree of caution, for they are, like any study, subject to methodological and interpretative flaws that may only be revealed in time. We must therefore await replication and evaluation in light of other similar studies before placing high confidence in them – though so far in this regard the prospects of the imaging study look promising, as it is consistent with the findings of numerous other imaging studies (Boeckle, Liegl, Jank, & Pieh, 2016; Hassa et al., 2017; Kanaan, 2016; Kanaan, Craig, Wessely, & David, 2007; Schmeing et al., 2013). Such caveats notwithstanding, the above results offer considerable support for key aspects of Freud's theory. Nicholson et al. (2016) provide reliable evidence of an aetiological connection between traumatic events and symptoms in the vast majority of CD patients. They also provide corroborating evidence that a substantial portion of CD patients have suffered sexual abuse, indicating that Freud must be given credit for recognizing the strong association between hysteria and sexual abuse. More crucially, Aybek et al. (2014) present evidence of an association between hysterical symptoms and attempts to suppress memories or emotions, which can be interpreted as the beginnings of repression. When one considers the

results of this study in the context of other imaging studies and in light of clinical experience, there is a compelling case that at least many CD symptoms are associated with some degree of repression.

The Bayesian account of hysteria

Though the empirical evidence may be favorable to Freud's theory, theoretical concerns remain. The historical review of Section 2 brings out the importance to Freud's theory of his notion of psychical energy. But herein also lies a worry, as it may be argued that this notion has not been articulated precisely enough. Freud and Breuer, as they themselves would well recognise, were trying to express an idea for which they did not quite have the conceptual resources, hence their resort to metaphors from physics (e.g. the electrical notion of "discharge"). To leave such a central idea theoretically underspecified in this way is, however, unsatisfactory. One problem is that, until recently, it has not been possible to convincingly relate this notion to concepts in neighboring scientific fields, such as neuroscience. This has led to considerable scepticism about its scientificity, hence also about the scientificity of the theory to which it is so fundamental. Fortunately, recent work in cognitive neuroscience offers hope of a more satisfactory rendering. This relates to the notion of "free-energy" developed by the eminent neuroscientist Karl Friston in the context of the Bayesian brain hypothesis. Friston asserts the correspondence explicitly: "the [Freudian] process of minimizing 'the sums of excitation' is exactly the same as minimizing the sum of squared prediction-error or free-energy in Helmholtzian schemes" (Carhart-Harris & Friston, 2010, p. 1270).

The Bayesian brain hypothesis posits that the brain is an inference machine that produces hierarchically organized multi-level models of the causes of sensory input (Figure 1), encoded as probability distributions by systems of neurons. The different levels of a model's hierarchy relate to different levels of causal analysis, with higher levels having to do with more abstract causes, that is, ones pertaining to a longer time frame. For example, lower to higher levels of a model relevant to reading text can represent respectively letters, words, sentences, paragraphs, and so on, all of which play a role in the interpretation of sensory information from the written page (Friston, Rosch, Parr, Price, & Bowman, 2018; Parr, Rees, & Friston, 2018).

The central idea of the Bayesian brain hypothesis is that the brain functions so as to minimize a quantity called "prediction error." At each level of the hierarchy, models make predictions that feed down to the level beneath, with the lowest level of prediction being of

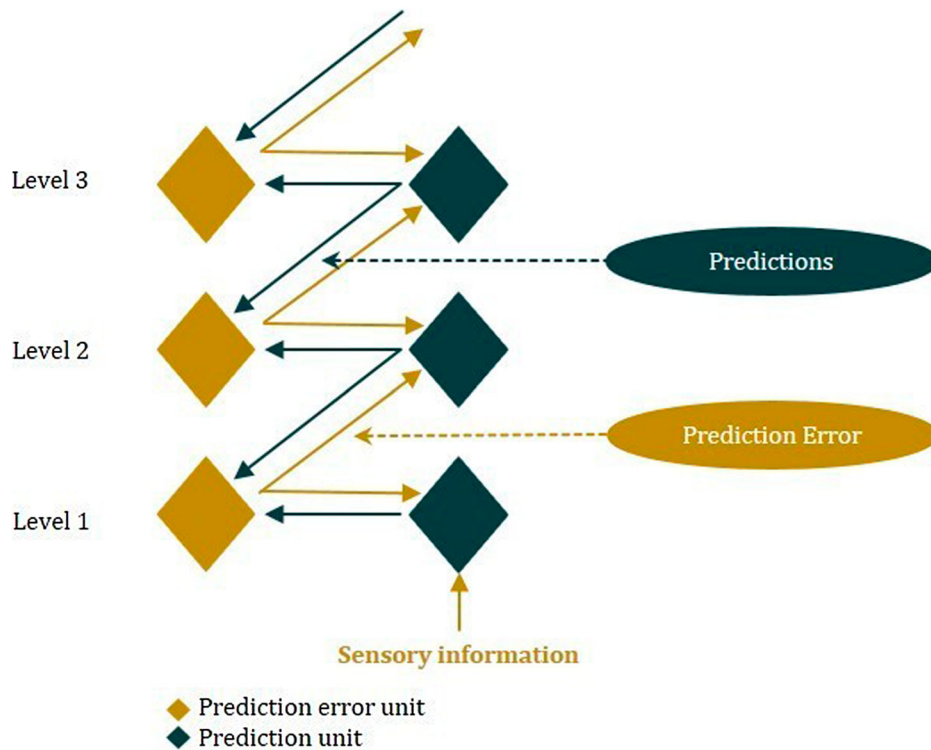


Figure 1. A schematic depiction of a hierarchical generative model. At each level of the hierarchy, prediction units (blue rhombuses) calculate predictions based on the incoming (lateral and bottom-up) prediction errors, and prediction error units (yellow rhombuses) calculate prediction errors based on incoming (top-down and lateral) predictions. The prediction calculation corresponds with that of Bayesian inference, in which a belief is updated in light of evidence according to a formula involving the prior belief and the probability of the evidence given this belief. Figure modified from Friston, Stephan, Montague, and Dolan (2014).

anticipated sensory input. In other words, at each level, circuits in the brain make predictions of what input to expect from the level below, contributing ultimately to a prediction about the incoming sensory information. Prediction error, that is, the mismatch between predictions and actual input, is communicated up the hierarchy, necessitating either a revision of the model or change in the sensory input (such as might occur through movement). This exchange of information and subsequent modifying is continued in an iterative process that results either in representations that are in line with the sensory input (the basis of perception) or movement of the body that changes the sensory input to be in line with the brain's representations (the basis of action).

Carhart-Harris and Friston (2010) argue that prediction error – or, more precisely, “free-energy,” an information-theoretic quantity representing a bound on prediction error – corresponds to Freud's notion of psychical energy, the mysterious quantity that Freud thought drove all mental activity. Recall that Freud believed this quantity to be subject to the “principle of constancy,” the general principle that the mind endeavors to keep the quantity at a constant low level. According to Carhart-Harris and Friston, what Freud was grappling

toward was the “free-energy principle,” the general principle that the brain functions so as to minimize free-energy (or prediction error). Thus, the Bayesian account of how the brain works tallies with Freud's metapsychological account of how the mind works.

The Bayesian brain hypothesis is supported both by powerful theoretical arguments and the broad range of its applications. These applications include explanations of aspects of cognition as diverse as vision, learning, and planning, and of mental disorder, including autism, schizophrenia, and hysteria (Clark, 2016; Hohwy, 2013). I suggest that it is in its application to hysteria that the Bayesian model offers the clearest prospect of a more theoretically satisfactory formulation of Freud's metapsychological ideas. Moreover, linking this particular Freudian construct to the Bayesian brain theory may shed light on other neuropsychanalytic correlations as well, to the extent that psychical energy or free-energy are central drivers of mental life.

While the possibility of a modern scientific rendering of the notion of psychical energy in Bayesian terms is quite promising, the challenge is that, unlike Friston, the (other) authors of the Bayesian account of hysteria (Edwards et al., 2012) play down any compatibility their account might have with Freudian concepts. Indeed,

they seem antagonistic to Freud's ideas about hysteria. This detracts from the potential that their account and Freud's theory have to mutually complement each other. In this section, contrary to these authors' inclinations, I hope to bring out this potential. The point is not that Freud's theory cannot be compatible with other theoretical frameworks, but that, given the widespread and increasing influence of the Bayesian approach, and the detailed correspondences between this approach and central Freudian ideas that have already been drawn by its chief architect (Carhart-Harris & Friston, 2010), this framework represents perhaps the most promising prospect for a more scientifically productive interpretation of Freud's theory of hysteria.

The picture that emerges is as follows, in Friston's terms. The brain is constantly trying to predict its sensory (including interoceptive) inputs through a set of multi-level models, or representations, of the world (both internal and external to the body). Upon receiving sensory input that does not match these predictions, prediction error (i.e. *psychical energy*) is generated based on this mismatch, and this is passed up through the hierarchy of the relevant model. In line with the principle of constancy, the brain functions so as to minimize this prediction error. One way it can do this is by "explaining away" the underlying incoming input. Such explaining away is accomplished through updating the model in light of the prediction error at each level of the hierarchy and issuing new predictions, which cascade down the hierarchy, culminating, at the lowest level, in predictions about the incoming signal. (For example, if one wakes up at 7 am expecting that it should be light outside, prediction error will be generated upon opening the drapes and looking out into the darkness; as one then remembers that one has traveled several time zones and forgot to re-set the alarm, one's model is updated, hence making sense of the darkness and enabling one to perceive the night scene more clearly.) The prediction error is thus minimized to the extent to which these cascading predictions contribute to ultimately matching the sensory input. This process is iterated continuously, thereby driving down the prediction error. On Carhart-Harris and Friston's (2010) account, the process of explaining away prediction error by a cascading set of predictions from high-level representations to low-level ones is equivalent to Freud's account of how the ego "binds" free *psychical energy*.

Most important to our present concerns is the role played in the above process by "precision-weighting." The degree to which a model is revised depends on the relative degree of precision afforded to the prediction error versus the model at different levels of the

hierarchy. In other words, at each level, the model is updated according to factors – estimates of reliability – which determine whether more consideration should be given to prediction error or to prior expectations. Degree of precision is in turn related to the placing of attention, so that, for instance, attending to certain inputs entails affording greater precision to the associated prediction error against prior expectations concerning that input. In the case of perception, if a higher degree of precision is afforded to prediction error, then such error would lead to a larger revision of the model at that level, whereas when a higher degree of precision is afforded to the model, then prediction error leads to a smaller revision of the model.

An example (given by Clark, 2016, pp. 57–58) that illustrates the above principles is that of driving in fog down a road that one knows well. In such a case, it might be better to increase the precision afforded to prior expectations and decrease that afforded to prediction error. As the road becomes less familiar and the fog clears, however, the reverse is true: it becomes important that bottom-up considerations be afforded more precision than top-down ones. The required change can be brought about by paying more attention to the road.

The Bayesian account of hysteria (Edwards et al., 2012) is that hysterical symptoms arise due to an imbalance in precision. The authors highlight the following two-part process. The first part occurs when certain sensory data, relating, in the case of somatic symptoms, to particular bodily sensations, are afforded increased precision. The second part occurs when a mid-level prior "belief"¹⁰ – that is, a middle level of the hierarchical model – that explains (by predicting) these sensations as due to a symptom is afforded increased precision. The coincidence of these two factors reinforces this mid-level symptom "belief," as increased attention on the relevant bodily sensations demands an explanation of those sensations and the mid-level "belief" provides such an explanation. This explanation is favored due to the abnormal precision afforded it, as a result of which the "belief" comes to override any contrary data and becomes entrenched. This entrenchment of the mid-level symptom "belief" determines the hysterical symptom, as the rest of the hierarchical model falls in line with this "belief."¹¹

A hysterical sensory symptom such as pain, for example, can arise via this process. It may begin when certain non-painful sensations associated with a prior illness are given increased attention. Since they have no current explanation, they constitute a source of prediction error that needs to be minimized according to the free-energy principle. The error can be minimized by changes made to the relevant hierarchical model. A

middle level of such a model may include an erroneous “belief” corresponding to the perception of pain, which predicts the sensations to some degree of accuracy and hence reduces the prediction error generated by them. For some reason, this “belief” is afforded excess precision, rendering it “resistant to extinction” (ibid., p. 3500), and the patient comes to believe that she is in pain, without realizing that her perception of pain was self-generated.

What can Freud’s theory of hysteria contribute to this account? The contribution comes by noting the role that repression can play in the process. Suppose, for example, the hysteric has emotions that engender certain bodily sensations, but these emotions have been repressed; the true source of the sensations are thus rendered inaccessible to the Bayesian process as explanations of those sensations.¹² It may be that the best strategy to deal with the resultant “dissociated” sensations is to direct attention away from them. However, if for some reason attention is directed toward these sensations – for example, because the hysteric has increased body-focused attention due to some physical precipitating factor such as illness – this creates a pressing need for some form of explanation of these sensations. A mid-level symptom “belief,” if it is of the appropriate kind, may offer such an explanation. Herein lies a second place where repression can contribute to the process: in this case, increased attention, hence precision, is afforded to the symptom “belief,” as a consequence of the repressive need to keep attention away from better explanatory “beliefs” about the true source of the sensations, due to their unconscious nature.

Thus repression, on this account, contributes to the process of entrenching the mid-level symptom “belief” in two ways. One, by creating dissociated sensations – that is, sensations with no current explanation because the correct explanation for them has been rendered inaccessible – repression creates a high demand for explanation once attention is brought upon these sensations. In other words, such dissociated sensations generate a large amount of prediction error – or “psychical energy” in Freud’s terminology. Two, by constituting a force directed toward keeping attention away from the aversive thoughts engendering these sensations, repression serves to push attention more urgently and forcefully toward the most convenient acceptable alternative explanation, which in the right circumstances would be a mid-level symptom “belief.”

This hypothesis has possible support from Edwards et al.’s (2012) own account of the causes of non-epileptic seizures. They point out that patients suffering from such “hysterical attacks” often have a history of childhood sexual abuse. They also point out that they “commonly

report somatic symptoms associated with panic attacks (palpitations, sweating, hyperventilation) at the onset of the attacks,” though do not report the associated emotions. From this they conclude that the Bayesian process “could produce physical symptoms that the patient interprets as being due to physical illness as he or she is not aware of the affective content of the panic episode” (p. 3501). What would make this hypothesis Freudian would be if the affective content is unconscious due to repression (cf. 1926, *S.E.*, XX: 111). This would illustrate my Freudian version of the Bayesian account of hysteria, where the need to explain otherwise dissociated bodily sensations gives rise to symptom “beliefs” that cause the hysterical outcome through Bayesian mechanisms.

The process might work as follows. As a result of some internal or external trigger, unconscious emotion related to a traumatic episode is stimulated. This may be due to a cue activating the (repressed) memory of the trauma, which leads to an emotional response that has physiological effects while its “affective content” remains unconscious. Especially insofar as attention is focused on these physiological effects, they constitute prediction error that needs to be minimized. The true explanation, relating to the emotion that generated the sensations, is inaccessible to the Bayesian process as an explanation of these sensations due to repression, since the emotion is intimately tied to the memories of the traumatic episode that have been repressed. Instead, increasing attention is placed on mid-level “beliefs” corresponding to the onset of a seizure. These “beliefs” may be the result of numerous factors, including prior experience,¹³ socio-cultural influences, and symbolic connections with the repressed thoughts.¹⁴ The symptom “beliefs” are afforded increased precision, to the point where they become entrenched, due to the urgency of finding a suitable alternative explanation of the sensations in order to preserve the unconsciousness of the correct explanation. These “beliefs” then generate the seizure, as movements are elicited in line with consequent motor predictions (according to the Bayesian brain hypothesis, in which proprioceptive predictions induce movement as a means of minimizing the prediction error generated by such predictions).¹⁵ These movements, along with other similarly generated effects, constitute a non-epileptic seizure.

Assuming that something like the above Freudian hypothesis is correct, the question then becomes, “To what extent is the Freudian contribution to the Bayesian account necessary for the production of hysterical symptoms?” The authors of the Bayesian account favor a diverse approach, in which repression need play no part. In taking this approach, they appear dismissive of

any significant Freudian contribution. But such an issue cannot be determined merely by theoretical considerations. While the Bayesian account theoretically allows that hysterical symptoms can arise without a Freudian contribution, in practice it may be that they cannot. So it remains possible that repression plays an essential role in the production of hysterical symptoms. In order to assess the prospects of this possibility, we need to engage with the empirical evidence.

In this regard, it would be instructive to look at some of the dismissive comments that the authors of the Bayesian account of hysteria make about Freudian ideas. In the passage where they discuss Freud (Edwards et al., 2012, p. 3501), they endorse the assertion by Sharpe and Faye (2006) that “the association with psychological issues is much less prominent than expected.” The results of the Nicholson et al. (2016) study provide a riposte to this assertion, as a potential association with psychological issues was found in 91 percent of CD patients. After a brief description of the Freudian theory, the authors go on to state:

Although many of the constructs and the symbolism proposed by Freud have been discarded, the idea that FMSS [Functional Motor and Sensory Symptoms] are an unconsciously generated expression of (otherwise uncommunicated) psychological conflict retains considerable popularity. We suggest, however, that this provides a rather one-dimensional approach to causation that may not be appropriate for many patients with FMSS. (p. 3501)

They do not explain who has discarded the “constructs and symbolism” and on what grounds, but simply leave the impression that it is a closed issue. This seems to me more rhetorical flourish than objective observation. The key point, however, is the one I made earlier, that no conclusion can be reached about causation on the basis of purely theoretical considerations, and thus must await empirical evidence to adjudicate. The evidence of the study by Aybek et al. (2014) is supportive of the “popular” view that unconscious psychological conflict is relevant to hysterical symptoms. As such, rather than being dismissive, the authors would do better to explore ways in which Freudian theory may be compatible with, and indeed enhance, their own highly promising ideas about hysteria.

Conclusion

Overall, the important recent scientific work on hysteria that we have considered, both empirical and theoretical, is supportive of Freud’s theory of hysteria. There is empirical support for the link between hysterical symptoms and psychological causes, the relevance of

secondary gain, the strong association between sexual abuse and hysteria, the aetiological significance of repression, and the process of conversion. This evidence suggests that Freud was on the right path in his theorizing. Furthermore, one of the most promising recent theories of hysteria, the Bayesian account, is compatible with Freud’s ideas. Indeed, when the evidence described above is brought to bear, Freud’s ideas suggest an improvement of the theory toward a more complete explanation of the emergence of hysterical symptoms.

To be sure, there are many elements of Freud’s theory that have not yet obtained extra-clinical empirical support. These include the proposal that repressed infantile sexual wishes provide the ultimate driving force for hysterical symptoms. It is, however, understandable that there has not yet been such support, as the proposal is based on evidence obtained using the distinctive clinical methodology that Freud developed, and such evidence for the moment lies beyond the reach of extra-clinical empirical methods. But given that Freud seems to have been on the right track about much else concerning hysteria, these further claims should not be dismissed too lightly.

Notes

1. *DSM-I* (1952) had used “conversion reaction”, and *DSM-II* (1968) “hysterical neurosis (conversion type)”.
2. Elsewhere Skues (2017) also offers a penetrating critique of the questionable historiography employed by some anti-Freud polemicists, in which the “picture of Freud as a liar” plays a guiding role in the interpretation of the evidence. The points he makes apply also to the recent revival of claims by Crews (2017) that Freud fabricated his early case reports on hysteria. One should not confuse such polemically motivated “exposés” with genuine historical scholarship—as the historian Forrester (2012) puts it, such accounts “may sound right to a contemporary for whom psychoanalysis carries no theoretical or therapeutic weight,” but they “won’t do as history.”
3. In citing from Freud’s work, I refer to the Standard Edition (Freud, 2001), giving year of original publication, volume of Standard Edition, and page numbers.
4. The above formulation of *FBP* takes poetic liberties, as it does not accurately reflect Breuer’s views. It is an account of what Freud called “defence hysteria,” whereas Breuer emphasized “hypnoid hysteria,” in which condition (b) is replaced by one in which the cause of the unconsciousness of the memory is that the event it is a memory of occurred when the sufferer was in a hypnosis-like state. Altogether, the authors describe three kinds of hysteria, the third being “retention hysteria,” in which the memory of the precipitating event may be conscious, but the emotion associated with it has not been appropriately expressed. Even in *Studies*, however, Freud expressed scepticism about the other kinds of hysteria, and soon after committed to the view that all hysteria

was (ultimately) defence hysteria, hence our focus on just this kind (1895, *S.E.*, II: 286).

5. Freud observed that the emotional reaction to the trauma was almost always suppressed at the time of the traumatic experience, but usually expressed once the memory of the event was recovered in therapy. The degree of emotional reaction was thus inferred by the degree of emotion expressed when the memory was recovered, but also by the need to explain both the severity and persistence of the symptom.
6. Brown, Nicholson, Aybek, Kanaan, and David (2014) note that, in relation to CD, “psychodynamic theories have been largely untested” (p. 171). Their own study, whose results may be interpreted as negative for Freud’s theory, illustrate just how indirect such tests have been. The study focuses on the degree to which CD patients are more liable than controls to suppress random words when directed to do so, yet there is nothing in Freud’s theory that implies that hysterics should be more liable to repress than non-hysterics in such cases. As the authors concede: “it is possible that results ... do not support the Freudian theory of repression because the negative valenced words are not specifically salient for each individual, or related to their proposed causal life event(s)” (p. 183).
7. Escape was judged on a scale of 0 to 3, with 0 representing “no escape” and 2 or 3 representing “high escape”; severity was judged on a scale of 1 to 4, with ratings of 1 or 2 representing a “severe event”.
8. That over half the CD patients did not report sexual abuse does not necessarily falsify Freud’s early theory of hysteria, which claimed sexual abuse general to all hysteria, since, for Freud, most of this abuse was repressed. However, the above findings on sexual abuse are more consistent with Freud’s later theory, in which sexual abuse, although prevalent, is not general to all hysteria.
9. In light of the results of Aybek et al. (2014), it is plausible that this failure to identify the key events was due to the beginnings of repression, though other possible explanations, such as “a conspiracy of silence between the patient and physician” are also possible (thanks to the editor for this suggestion).
10. “Belief” here is not intended to be understood with its usual commonsense psychological meaning, as a propositional attitude. It is rather a representation of the causes of sensory data in the form of a probability distribution encoded by the activity of a collection of neurons.
11. There is an additional important part to the theory, though one less relevant to our present concerns, in that, in order to explain the emergence of the symptom belief, the Bayesian process leads to “a misattribution of agency in the sense that top-down attentional processes induced the belief but did not predict its content” (Edwards et al., 2012, p. 3499).
12. On Freud’s theory, such emotions would be intimately associated with a whole series of unconscious thoughts, including memories, fantasies, and wishes, the unacceptable (to the ego) nature of which is what necessitates the repression.
13. It is notable that there is a high prevalence of non-epileptic seizures among those who have experienced epileptic seizures (Sharpe & Faye, 2006).
14. Representations that correspond closely with repressed material could arise because they provide suitable explanations of the sensations generated by this material. For example, a representation “underlying a fixed dystonic hand” may have arisen because it predicts sensations generated by the repressed memory of “a clenched fist associated with angry feelings experienced at the time of trauma” (Brown, 2004, p. 806).
15. See e.g. Edwards et al., 2012, p. 3498; and Clark, 2016, Ch. 4, for fuller descriptions of how this process works.

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