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# Between Mind and Body? Psychoneuroimmunology, Psychology, and Cognitive Science

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*Over the past half century, our best scientific understanding of the immune system has been transformed. The immune system has turned out to be extremely sophisticated, densely connected to the central nervous system and cognitive capacities, deeply involved in the production of behavior, and responsive to different kinds of psychosocial event. Such results have rendered the immune system part of the subject-matter of psychology and cognitive science. I argue that such results, alongside the history of psychoneuroimmunology, give us good reason to be skeptical about the characterization of cognitive science and psychology as studying the mind and the mental.*

## 1. Introduction

Over the past half century, our best scientific understanding of the immune system has been transformed in ways of great, and often underappreciated, philosophical significance. My aim in the following is to start to make the nature of this impact precise, taking my cues primarily from recent empirical literature within immunopsychiatry and psychoneuroimmunology, before explaining why I hold that the relevant findings are philosophically significant. The immune system has turned out to be extremely sophisticated, densely connected to the central nervous system—including to those parts that appear to serve as the material basis of the highest reaches of cognition—deeply involved in the production of behavior, and responsive to different kinds of psychosocial event

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(section 2). I argue that such results render the immune system both a cognitive and psychological system, in the specific sense of being part of the subject-matter of cognitive science and psychology (section 3). In section 4, I consider what this means for the construal of cognitive science and psychology as studying the mind and the mental, arguing that the history of psychoneuroimmunology gives us good reason to be skeptical about the utility of characterizing cognitive science and psychology in this way.

## 2. Why the Immune System?

In the 1970s, the psychologist Robert Ader stumbled across a groundbreaking result for immunology while using a Pavlovian conditioning paradigm to study learning in rats (Ader 1976; Ader and Cohen 1975). He administered a sweet substance, saccharin, with no harmful effects (the conditioned stimulus), paired with a sickness-inducing chemical, cyclophosphamide (the unconditioned stimulus). As expected, the rats learned to avoid saccharin. However, that was not the exciting part.

The groundbreaking finding was that if continually forced to take saccharin, the rats began to die of a range of infections; furthermore, the rate of the rats' deaths increased with the amount of saccharin administered. Saccharin is totally harmless, so there is no obvious reason that this should be the case—but even so, the rats died (Pincock 2012). The key to solving this mystery was that coincidentally, as well as upsetting stomachs, cyclophosphamide is an immunosuppressant. It seemed to Ader that by using cyclophosphamine to condition the rats to avoid saccharin, they had accidentally also conditioned the rats' immune systems to “switch off” in response to the taste of saccharin—and that this left the rats vulnerable to the normally-harmless environmental pathogens that killed them (Ader and Cohen 1975).

Ader, working with others (most notably the immunologist Nicholas Cohen), went on to prove his hypothesis (see Ader 1980, 2000; Ader and Cohen 1982, 1985, 1993; Ader, Felten, and Cohen 1990; Bovbjerg, Ader, and Cohen 1982). He did so in the face of a great deal of skepticism. Amusingly, although Ader dubbed the area of study he had begun (or at least reinvigorated), “psychoneuroimmunology,” many immunologists insisted on avoiding the “psycho,” dubbing the topic “immune-neuroendocrine interactions” (see Daruna 2012).

The discipline won over sceptics as the details of the circuitry were unveiled. In particular, Felten and Felten (1988) showed that the sympathetic nervous system (SNS) innervates immune tissues, and that synaptic terminals are to be found extremely close to immune cells, and Besedovsky et al. (1991) showed that interleukin-1 could activate the hypothalamic-pituitary-adrenal axis. These, and other similar results, gave a basic

mechanism by which chemical signals in peripheral tissues might be translated into nervous signals and propagated all the way up to higher brain areas (most notably, the hippocampus, amygdala, and areas of the neocortex such as the insular cortex, anterior cingulate cortex, and ventromedial prefrontal cortex), and—importantly—vice versa. Dantzer found an array of parallel signals that mediate bidirectionally between the highest levels of cognition and the lowest levels of the immune system, apparently responsible for both chemical changes at the level of cells and behaviors at the level of the whole organism (Dantzer 1994; Dantzer et al. 1998; Dantzer et al. 2000). Other key results include the fact that the blood-brain “barrier” often acts as a selective interface, selectively facilitating non-nervous (including immune) activity within the brain (Banks 2016; Erickson and Banks 2018).

Nowadays, psychoneuroimmunology is a largely respectable, growing area of study (Pariante 2019), with several journals devoted to the field, most notably *Brain, Behavior and Immunity* founded in 1987. Unfortunately, the field still has a somewhat dubious reputation in some areas, although I believe that this is largely unwarranted, and more to do with the extra-scientific marketing and reception of its claims than to do with the science itself—an issue I return to in section 4.

Recently, a closely related area of study dubbed “immunopsychiatry” has begun to receive significant attention. The precise relationship between psychoneuroimmunology and immunopsychiatry is a little contentious, since both acknowledge bidirectional interaction between the immune system and (other) psychological systems. Pariante suggests that immunopsychiatry places greater emphasis on one direction of interaction, focusing on cases where “our behaviors and emotions are governed by peripheral immune mechanisms” (Pariante 2015, p. 1), primarily in the context of pathology.

There are other aspects to the adoption of the term “immunopsychiatry” by many researchers. The shift in emphasis was partly initiated by the discovery, still ongoing, of potentially causal correlations between several important psychiatric disorders and inflammation of various sorts (e.g., Khandaker et al. 2015), particularly by the finding that a depression-like syndrome could be induced in rats through peripheral inflammation (Yirmiya 1996), and the later finding that symptoms of depression including anhedonia and low social motivation can be induced in humans through similar means (Eisenberger, Berkman, et al. 2010; Eisenberger, Inagaki, et al. 2010; Moieni et al. 2015). A related factor is the ongoing search for a “respectable” basis for psychiatry, in which the immune system has recently become one of the most promising prospects (see Morgan 2017).

Whichever name one prefers, the field is rife with exciting results exploring the links between the immune system and phenomena traditionally viewed as within psychology and psychiatry. Two sorts of results are, I think, particularly exciting: correlations between personality and immune system variables (see Segerstrom 2000) and correlations between various kinds of psychosocial stressors, most notably various kinds of trauma and immune system variables (Agorastos et al. 2019; Neigh and Ali 2016; Ulmer-Yaniv et al. 2018; Wang et al. 2017). These results suggest that the immune system plays important roles in shaping one's personality and behavior, including an important role in perpetuating the effects of trauma.

Immunoglobulin A (IgA) is an immune system molecule which plays an important role in early responses to pathogens; it is also extremely abundant and secreted in saliva. Its secreted form, (S-IgA), serves as a reliable biomarker for the level of functioning of the immune system (Bosch et al. 2002; Macpherson et al. 2008). Secretion of S-IgA can be affected by physiological stressors, but also by psychological stressors such as loneliness (Engeland et al. 2016), and the magnitude of this effect depends in part of how much anxiety is caused by the stressors (Ulmer-Yaniv et al. 2018). Yirmiya et al. (2018) examined the development of anxiety symptoms following chronic trauma, by following a cohort of adolescents and their mothers in Sderot, which is a small town in Israel near the border with Gaza that frequently experiences rocket attacks. They found evidence of three pathways by which trauma might result in anxiety: one mediated by parenting behavior; one mediated by dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, as suggested by increased cortisol levels, and one mediated by the immune system, indicated by increased levels of S-IgA.

Results such as these suggest an important role for the immune system in perpetuating, and shaping, the effects of trauma and stress. Related work, some studying the same cohort, makes use of the notion of biobehavioral synchrony (Feldman 2012), synchrony between the online physiological and behavioral processes of attachment partners (e.g., parents and children). This work suggests that the immune system interacts with many other systems, both bodily and mental, in the formation of parent-child bonds, the development of empathy, and measures of resilience (Levy et al. 2019; Motsan et al. 2021; Ulmer-Yaniv et al. 2016).

To understand how the immune system plays its part in trauma and stress, one must recognize the significance of gene regulation and the SNS. Gene regulation plays an important role in orchestrating immune system responses—the deployment of populations of cells, cell differentiation, and more (see Amit et al. 2011; Rothenberg 2014; Roy 2019; Smale

and Fisher 2002; Smale et al. 2014). The SNS innervates the bone marrow, where blood is made (hematopoiesis), and monocytes (the largest white blood cells) differentiate; SNS activity can affect at least the mobilization of the involved (hematopoietic) stem cells (Beiermeister et al. 2010; Katayama et al. 2006; Méndez-Ferrer, Lucas, Battista, and Frenette 2008). The sympathetic nervous system (SNS) can also respond differentially to social stressors, bringing on specific states of the immune system in response to social conditions through gene regulation (Powell et al. 2013).

This process of gene regulation is capable of responding to quite fine-grained sorts of psychosocial event. Murray et al. (2019) find that falling in love results in immune system gene regulation. With a particularly important signaling role played by circulating oxytocin, falling in love results in “up-regulation of Type I interferon response genes” and “down-regulation of  $\alpha$ -defensin-related transcripts,” possibly reflecting “selective up-regulation of innate immune responses to viral infections” and “dendritic cell facilitation of sexual reproduction.” It seems, in this case, that the immune system has a specific, tailored response to a complex psychosocial event.

Immune system responses to psychosocial stressors are a very general feature of the immune system and the stress response (see McEwen 1998; McEwen and Wingfield 2003); in many cases, psychosocial stressors appear to compete for resources with those stressors the immune system is geared towards dealing with, for example, wound-healing is disrupted by psychosocial stress (Gouin and Kiecolt-Glaser 2011), leading Broadbent and Koschwanez (2012) to propose a “psychology of wound healing.” Furthermore, as suggested by the immune system response to love, the immune system responds highly differentially to different kinds of stressors. For example, the immune system appears to respond differentially to chronic interpersonal and non-interpersonal stress, with recent results suggesting only the former is implicated in the development of depression (Slavich et al. 2020).

Other fine-grained biomarkers for immune system functioning have been associated with other sorts of psychosocial event. Proinflammatory cytokines, for example, have been associated with increased anxiety surrounding public speaking (Auer et al. 2018), and more generally with social stress (Slavich et al. 2015; Slavich and Szabo 2019); findings interpreted by the authors as evidence that immune system function can modulate vulnerability to such stressors. Entringer (2021) reports that childhood abuse accelerates inflammation-associated aging. Penz et al. (2018) show that stressful life events predict changes in the composition of white blood cells in peripheral blood. Immune system markers have also

been studied in relation to combat stress and posttraumatic stress disorder (see Agorastos et al. 2019; Neigh and Ali 2016; Wang et al. 2017).

Such results lead Miller et al. (2009) to propose that the immune system, more specifically chronic inflammation, is an important way that the social environment becomes embodied (Krieger 2005)—the way it “gets under the skin.” As Priest (2021) highlights, responding to a study by Maurel et al. (2020) correlating five inflammatory markers with measures of educational status across multiple cohorts, evidence for this proposal is continually accruing. As Priest puts it, following Krieger, such results show that “bodies tell stories about, and cannot be studied separate from, the conditions of their existence” (Priest 2021, p. 23).

As well as bearing the marks of past experience, immune system variables are also correlated with personality traits (such as neuroticism and conscientiousness). This has long been recognized (see Segerstrom 2000), and is much-studied. A caveat is in order here: inferring causation from correlations is difficult, and the direction of causation is often not clear (as discussed in relation to inflammation and psychiatric illnesses by, e.g., Khandaker et al. 2017). Where correlations are found it is not entirely clear which way causation runs. It is worth noting that much of the research on personality and the immune system focuses on explaining the relevant correlations as resulting from the effect of personality traits on health-related behaviors (such as smoking and drinking alcohol), which in turn impact the immune system (see M. S. Allen et al. 2019; Bogg and Roberts 2004).

However, as mentioned above there is strong evidence that signals run in both directions. There is strong evidence for both psychological effects on the immune system (most notably Ader’s work), and the immune system’s effects on the psychological (most notably the work on inflammation-induced quasi-depression in rats). As such, it seems safe to assume that presently-unexplained correlations are often at least partially the result of the immune system and psychological functioning interacting reciprocally—although in any particular instance, it is not clear. Given that there is evidence of such direct links, it seems that the psychological can affect the immune system without the need of mediation by health-related behaviors, and changes in the immune system can change personality-related psychological states. It is of course plausible that personality affects health-related behaviors, and this in turn affects the immune system. It is deeply implausible, however, that this behaviorally mediated, one-directional interaction is the *only* sort of interaction between the immune system and personality traits.

In sum, supported by myriad nervous and non-nervous signals and pathways, the immune system plays at least two important roles. First,

it appears to be an important mechanism by which past experiences, including trauma and psychosocial stress, have their long-term causal impact. Secondly, it appears to be involved in shaping people's behavioral dispositions and personality traits. Taking these roles together, the immune system appears as a potentially important mechanism by which past experiences shape the personality and behavior of an individual in the long-term.

There is one final feature of the immune system that I would like to mention. Shields et al. (2020) recently released the first systematic review and meta-analysis of randomized clinical trials examining the effectiveness of psychosocial interventions on immune function. They found that all the psychosocial interventions studied improved immune function, most reliably cognitive behavioral therapy, and combined or multiple psychotherapies. Furthermore, they found that CBT can achieve approximately the same level of improvement in inflammation levels in rheumatoid arthritis as the maximum dose of infliximab (among the most common medications for autoimmune conditions, including rheumatoid arthritis), for less than 10% of the cost, and with longer-term effects. In short, the immune system, as well as being wired into the brain areas associated with higher cognition, shaping personality and mental health, and retaining traces of past experience, is amenable to talk therapy.

### 3. The Status of the Immune System

#### 3.1. Some Previous Proposals

These results, gathered over the past 45 or so years, constitute a proper object for philosophical discussion. As the immune system's sophisticated interactions with the central nervous system, including those parts of the brain associated with the highest reaches of cognition, as well as its own internal complexity and plasticity, have come more clearly into view, theorists have tried to keep up, offering novel accounts of the immune system that can account for these features.

An important historical point here is that the internal complexity and plasticity of the immune system began to come into view even before the foundation of psychoneuroimmunology, in the work of N.K. Jerne (see especially 1955, 1974, 1985). Jerne's theory was built on still-acknowledged points about the organization and operation of the immune system (Tauber 2000, 2017; see also Pradeu 2011, 2020). Even without the cross-talk between the central nervous system and the immune system, and even without the immune system's effects on mood, thought, and behavior, these points constitute serious pressure to count the immune system as a cognitive system (see especially Tauber 2013).

In 1979, Francisco Varela's *Principles of Biological Autonomy* was published, a seminal work in the autopoietic approach to biology and cognitive science, which argues that the immune system is a cognitive capacity, since it bears the key features of cognition (by his lights)—a domain of sensitivity, adaptive flexibility, and a certain sort of organization which he calls “closure.” There have since been other proposals to view the immune system as a cognitive system, based on similarly structured arguments—for example, Hershsberg and Efroni (2001) argue that the immune system is cognitive because its “perceptual sensitivities” are determined partly by interaction with its environment. Others, in particular Blalock (2005), proposed that the immune system should be viewed as a sense, concerned with the detection of pathogens. This idea is similar to that recently offered by Bhat et al. (2021) working within the free energy framework (e.g., Friston 2012; Friston et al. 2006), who argue that the immune system is an inferential system in the business of looking for non-self antigens (however, see Pradeu 2011, 2020 for dissent). D'Acquisto and collaborators (e.g., Brod et al. 2014; D'Acquisto 2017) propose that emotions and immune system responses exist on a continuum, seemingly suggesting that they form a natural kind, in light of recent results that suggest that there is a great deal of overlap between the mechanisms that underlie emotion and those that underlie the immune system (see also Chiurchiù and Maccarrone 2016).<sup>1</sup>

Any of these proposals (if correct) would provide some pressure to count the immune system as part of the proper subject-matters of psychology and cognitive science. I wish to argue for this claim as directly as possible, without a detour through a purely theoretical definition of “cognitive” or “psychological.” There are three main reasons that I argue in this way. First, because there are already many arguments that run via theoretical definitions. Secondly, because any argument that runs via a theoretical definition will be entirely unpersuasive to those who do not believe in that theoretical definition, or who do not believe in the relevance of such definitions in contexts such as this. Finally, because I harbor significant skepticism about the utility and legitimacy of using theoretical definitions of “cognitive” and “psychological” in arguments about whether a particular capacity is cognitive or psychological. In my view, these too often proceed

1. Tauber's view that the immune system is cognitive appears to rely on the intuitive view that a system is cognitive if its operation is aptly described in ordinary “cognitive” terms, that is, in terms of “such functions as ‘perception’, ‘recognition’, learning’, and ‘memory’” (Tauber 2013, p. 239). It is important to note that even this is contentious, and if called on in this context, would beg the question against various opposed accounts of how to define cognition or demarcate the subject-matter of cognitive science (e.g., Corcoran et al. 2020; Rupert 2013).



by definitional stipulation, and even where they do not, I struggle to see the justification for choosing a theoretical definition (see especially Akagi 2018; C. Allen 2017; Gough 2022).

There are many ways to understand both terms. To focus just on “cognition,” there is a sense of “cognition” that can be contrasted with perception (see Block 2016), meaning something like Fodor’s *central cognition* (Fodor 1983). There is another sense of “cognition” defined to exclude emotion, that predates cognitive science (see Boden 2006). There is yet another sense of “cognition,” perhaps the oldest and going back to its etymological origins, where it is defined as to do with knowledge. However, the senses of “cognition” and “psychological” that I am interested in are those which are defined primarily in relation to cognitive science and psychology.

Because of this, I prefer a more practice-oriented than theoretical approach to defining “psychological” and “cognitive.” There are two senses in which my preferred approach is more practice-oriented. First, I am adopting a broadly pragmatist view of what “psychological” and “cognitive” mean (e.g., Peirce 1878), according to which the psychological is whatever our best possible psychology would study, and the cognitive is whatever our best possible cognitive science would study. This involves a mix of prescription and prediction. The reason for this is that sciences tend to be riddled with errors, including errors about the nature and extent of their domains. When talking of the physical, for example, we cannot simply defer to current physics, but must defer to an idealized future physics; this involves guessing at how a discipline would progress, if it continued as it ought.

Secondly, my approach is practice-oriented in that I think that it is best to defer to the practicalities of a science when characterizing that science’s subject matter—that is, to defer to issues such as the aims, methods, and models housed within a science, and the interests of the scientists. The reason for this is that I think that it is hard, perhaps impossible, to separate the question of how a discipline would end up if it progressed as it ought from actual trends and practical considerations within the discipline as it currently exists.

It is possible that the theoretical definitions of cognition on offer are intended to be practice-oriented in at least the first sense of being directly tied to the subject-matter of cognitive science. One option is that they are intended as wholly top-down prescriptions for what cognitive scientists ought to study. This seems an uncharitable interpretation, since it is not clear what would justify such a prescription. The other option is that they are intended as guesses about the subject-matter of our best possible cognitive science: non-circular characterizations of what the best possible cognitive science would study. This latter option strikes me as equally

uncharitable as an interpretation of what, for example, Varela is up to (Varela 1979; Varela et al. 2016). A guess of this kind about the ultimate limits of cognitive science or psychology is surely unjustifiable in light of the sheer level of uncertainty in those disciplines: it involves making a series of very difficult guesses about the future of the disciplines, what models and issues they might turn up, and what phenomena will turn out to be relevant to each other and amenable to similar models (see also C. Allen 2017).

My argument is also distinct from many previous arguments for the cognitive status of the immune system in another way. Many of these arguments take for granted an understanding of which capacities are cognitive, and argue that the immune system has one of two special relationships to such capacities—either it is part of the system that implements them (a version of the “embodied mind” proposal, discussed further below, especially in section 4), or because it itself instantiates them. I have no objection to such arguments, and indeed similar points form part of my overall argument in section 3.2, since I view each kind of argument as providing good, if partial, reasons for counting the immune system as cognitive.

There are two key reasons that I do not offer an argument along these lines. The first is that there is good reason to count the immune system as cognitive even setting aside the fact that the immune system implements and instantiates conventionally cognitive capacities. As I highlight in section 2 and below, the immune system is instrumental in shaping personality, in the effects of trauma, and in the propagation of past experience; it is also important to psychiatry, and a flexible and adaptive system with a reasonable claim to being an intentional system, as well as causally, functionally, and structurally similar to (other) cognitive systems. All these constitute strong reasons to count the immune system as cognitive, irrespective of whether or not it implements or instantiates conventionally cognitive capacities (see also section 4).

The second, related, reason is that there is an important area of inquiry within the “embodied mind” tradition—reconsidering which capacities are cognitive in the first place (see also section 4). Arguments based exclusively on the instantiation or implementation of conventionally cognitive capacities are ill-suited to this kind of question, since they generally take for granted which capacities are cognitive at the outset. Conversely, arguments based on similarity to other cognitive capacities and systems, or the explanatory and practical goals of cognitive science, are well-suited to furnishing reasons for counting capacities as cognitive which are not conventionally counted as such. Overall, therefore, an exclusive focus on implementation and instantiation risks underselling the strength of the case for counting the immune system as a cognitive system, and risks

missing cognitive aspects of the immune system, in particular, cognitive capacities which are not currently conventionally considered cognitive.

### 3.2. The Immune System as Cognitive and Psychological

I therefore believe that the best way to argue that a phenomenon is psychological or cognitive is to argue that there is currently a strong reason for psychology or cognitive science to count that phenomenon as part of its subject matter on the basis of its current models, methods, and aims, and that it would be practically feasible for the discipline to do so. This is a much more conservative approach, aiming to predict and prescribe progress only by a single small step, rather than aiming to jump all the way to the end, on the basis of current trends, aims, and methods, rather than on the basis of theoretically elegant definitions.

Since my understanding of the terms “psychological” and “cognitive” defers to psychology and cognitive science, it is worth saying more about these disciplines and the relationship between them. It is common to see cognitive science construed as the latest and greatest version of psychology (see Mandler 2007). If this is correct, then the psychological and the cognitive are one and the same. It is also common to see psychology conveyed as a subdiscipline of cognitive science; if this is the case, then the psychological is a proper subset of the cognitive. Neither is my preferred construal of the disciplines. I see cognitive science and psychology as distinct but overlapping disciplines: psychology is characteristically concerned with complex behavior, personality, and (in many of its iterations) consciousness; cognitive science grew partly out of psychology, especially the Gestalt school (Mandler 2007), but also grew partly out of computer science, cybernetics, and information theory (among others; Boden 2006), giving it not only slightly different models and methods, but also slightly different explanatory interests and aims. It is therefore not safe to assume that the psychological and the cognitive are one and the same. Furthermore, since there are parts of psychology (e.g., psychometrics) which are not obviously a part of cognitive science, it is not safe to assume that the psychological is a proper subset of the cognitive.

My claim is that the immune system is both psychological and cognitive. By this I mean that the immune system forms a part of both disciplines’ ultimate subject matters, and I intend to support this claim by arguing that there are good reasons for psychologists and cognitive scientists to study it. This invites a question: what sort of thing is a good reason for a discipline to count a phenomenon as part of its subject matter?

Importantly, for psychology and cognitive science to count the immune system as part of their subject matter is not merely for the disciplines to discuss the immune system. The mere fact that a science has to talk about a

certain phenomenon in order to understand its domain does not automatically make that phenomenon part of its domain. In this instance, one might think that the immune system is merely coupled to (densely, reciprocally, causally linked to) the psychological/cognitive, and must be discussed by psychologists/cognitive scientists on this basis, even though it is not itself psychological/cognitive. Analogously, social psychologists often have to consider cultural phenomena, but this does not always mean that those cultural phenomena are themselves psychological, only that they greatly influence the psychological.

This is an important worry to address, since it helps to clarify how the past half century's findings in psychoneuroimmunology and immunopsychiatry suggest that the immune system is itself psychological/cognitive, rather than merely being coupled to the psychological/cognitive. The idea is that psychology and cognitive science will be more successful sciences if practitioners take an interest in at least some aspects and capacities of the immune system in their own right, not just an interest in how they causally impact (other) psychological and cognitive capacities.

I offer a number of reasons for cognitive science and psychology to count the immune system as part of their ultimate subject-matters in this sense. I offer these reasons only as jointly sufficient—taken together, they constitute a compelling case. I do not hold any of these reasons to be necessary, because I think it is often plausible that a good reason for a discipline to count a phenomenon as part of its subject matter can be as broad as that “it’s interesting and we can.” For example, key models and skills in the discipline might end up being appropriate for a broader range of interesting phenomena than anticipated. However, I think it is possible to offer stronger, and more specific, reasons in this case.

The first reason for counting the immune system as psychological and cognitive is that there is currently a significant number of cognitive scientists and psychologists who do, in fact, study the immune system (e.g., DeAngelis 2002)—several of whom are cited in this article. The proposal that the immune system is therefore psychological or cognitive is in this sense a conservative, non-revisionary proposal. The results adduced so far give us good reason to believe that this will (and should) remain the case.

One key feature of these results is that the immune system appears to densely reciprocally interact with, and overlap with, the nervous system in its (other) cognitive and psychological roles (see also Pradeu 2020, ch. 5). These results support a version of the “embodied mind” view, discussed further below, according to which the immune system is part of the system that implements psychological and cognitive capacities.

Another key aspect of the results above is that they suggest that the immune system is in many important ways similar to (other) psychological

and cognitive systems (again, see Pradeu 2020, ch. 5). The immune system has many salient dimensions of similarity to (other) psychological and cognitive systems that make it amenable to similar models and methods, and therefore amenable to the expertise of psychologists and cognitive scientists. As Ader's work shows, the immune system is amenable to traditional psychological models such as Pavlovian models of learning. It is also amenable to cognitive models, in particular computational models<sup>2</sup> (see especially Cohen and Efroni 2019; Forrest and Hofmeyr 2001) and the free energy framework (Bhat et al. 2021). This amenability to similar models, methods, and expertise is accompanied by other key kinds of similarity between the immune system and the (rest of the) psychological and cognitive—similarity in their mechanisms and material basis (particularly of note, the nervous system; see section 2), relevant kinds of causal influence (e.g., psychosocial stressors), and relevant kinds of causal consequence (e.g., complex organism-level behaviors).

Perhaps most importantly, the immune system is relevant to the core goals of cognitive science and psychiatry. Cognitive science, since its inception, has been interested in principles of self-organization, the relationship between such principles and intelligence, and in the range of possible kinds of intelligent, adaptive behavior (see Boden 2006)—all considerations to which the immune system is relevant. Psychology has, at least since the twentieth century, been interested in understanding personality, complex human behavior (especially social behavior), motivation, and responses to trauma—interests shared across schools of psychology as diverse as psychoanalysis<sup>3</sup> and behaviorism. Again, in order to achieve these aims, it is helpful to study the immune system. As stressed in section 2, the immune system appears to play key roles in shaping personality and behavior, as well as propagating the effects of trauma and other experiences. A shared aim of both psychology and cognitive science is to facilitate and support psychiatry. As also stressed in section 2, the immune system appears to be a key factor in various psychiatric conditions. Additionally, the rapid growth and burgeoning significance of immunopsychiatry also strongly suggests that studying the immune system forms a part of supporting psychiatry.

The point, therefore, is not just that the (rest of the) psychological/cognitive can affect the immune system, and vice versa, but that the immune system shares many other characteristic features of the psychological/cognitive—at the least, behavioral outputs, external (including social)

2. In the sense of modelling a system as, in some sense, “a computer,” not just modelling done on a computer.

3. On similar grounds, D'Acquisto goes so far as to argue that “Carl Jung and Sigmund Freud were immunologists, but they did not know it” (2017, p. 10).

inputs among which it makes fine-grained discriminations, similar principles of operation, amenability to talk therapy, and relevance to psychiatric illness. The issue is not just to do with coupling, but also to do with salient similarity between the psychological/cognitive and the immune system (see, for example, Jerne 1985). Such similarity is significant because it, along with the fact that the immune system is coupled to the (rest of the) psychological/cognitive, underwrites significant practical benefits to studying the immune system as a psychological/cognitive system.

An important residual worry relates to whether there are representations in the immune system. One might believe that processing representations is among the most important features of those systems in the domain of cognitive science and psychology. This point is highly important, especially if one believes in prescriptive definitions of “psychological” and “cognitive.” My response is twofold.

First, the cognitive models that I mentioned above as applying to the immune system are extremely hard to interpret without ascribing representations, or at least something along those lines, to the immune system (Swiatczak and Tauber 2020). Furthermore, in as much as these models can be interpreted in non-representational terms when applied to the immune system, they can be so-interpreted when applied to (other) psychological and cognitive systems—so the proper applicability of these models gives us as significant a reason to believe in immune system representations as the applicability of such models to, e.g., the brain, gives us to believe in neural representations. This comes out particularly clearly in Tauber’s (2013) discussion, which carefully traces out many of the ways in which the debate over the mode of operation of the immune system mirrors the debate over the mode of operation of cognition more generally. More generally, “intentional language” involving the assignation of content is systematically used in the discussion of the immune system (see especially Cohen and Efroni 2019; Howes 2000; Matthen and Levy 1984; Swiatczak and Tauber 2020)—for example, discussion of recognition, perception, memory, and learning is widespread and ostensibly unavoidable in characterizing the operation of the immune system (again, see Tauber 2013).<sup>4</sup>

4. Some have argued that such “intentional” language in immunology is merely metaphorical (e.g., Melander 1993; Rosenberg 1989). However, see, Hesse (1965); Keller (2002); Reynolds (2018); Swiatczak and Tauber (2020) for the argument that, even if so, this does not make such language dispensable, unscientific, or easily dismissed. See also Figdor (2017, 2018) for an argument that I think counts against the interpretation of such language as metaphorical in the first place. For an interesting argument advocating caution about the kind and specificity of content that can be assigned to at least some immuno-endocrine signals, see Churchland and Winkielman (2012).

Secondly, and more importantly in my view, if there are good reasons for psychologists and cognitive scientists to study the immune system in its own right, it does not really matter whether the immune system involves representations by my lights. Instead, it would show that it is wrong to place such emphasis on representations in characterizing the psychological and the cognitive in the first place (see also Ramsey 2007, 2017)—especially given that psychologists and cognitive scientists do in fact study the immune system.

#### 4. The Immune System and the Mind

In this final section, I wish to consider the significance of these results for our understanding of psychology and cognitive science: in particular, whether the above argument undermines the construal of psychology and cognitive science as studying the mind and the mental. The most obvious extant philosophical view of the mind to which these results are relevant is the view of the mind as *embodied* (Varela et al. 2016). According to a dominant construal of this view, at least some mental capacities and processes are implemented by “bodily” systems outside the brain (Wilson and Foglia 2017). Taking one’s cues from this position, one might claim that much of the psychological and cognitive activity of the immune system is the implementation of mental capacities. One might claim, for example, that the immune system is involved in the implementation of perception, specifically the perception of pathogens, that the immune system is involved in the implementation of emotions, such as anxiety and stress, and/or that the immune system is involved in the implementation of unconscious beliefs and fears as part of its role in the processing of trauma.

“Mind” can be understood as gesturing towards a set of capacities (see Kenny 1989; Ryle 1949), and as referring to the system that underlies those capacities—which is not to claim that these are the only meanings of “mind.”<sup>5</sup>

5. It has been suggested to me by an anonymous reviewer on a previous version of this paper that this section requires a discussion of the mind-body problem, and I agree that the lack of such a discussion may read as strange to many readers. Unfortunately, I think that a full discussion of the mind-body problem would take me far beyond the scope of this piece. I think this because I am skeptical that there is any such thing as the “mind-body problem.” I believe that instead, there are many problems that go under this name (the problem of content, the problem of representation, of normativity, of free will, of consciousness, agency, nonspatiality, causal exclusion, reason, and so on; see especially Rorty 1970, 1979, 1982). I hope that any such problems have been adequately addressed in as much as they are relevant as I go along.

This provides me with an excuse to mention a positive view, which I can only indicate briefly here, since I lack the space to argue for it. I believe that the immune system, as well as the immune-CNS axis and the brain, are among many, many overlapping, control systems that make up a human being (Bechtel and Bich 2021), none of which is appropriately called “the mind,” and all of which do some of the work attributed to the mind in the “folk” understanding of human beings as composed of a mind and a body.

Applying the above understanding of the embodied mind thesis to the immune system, the immune system is part of the mind-as-system because and in as much as it implements some of the mind-as-capacities. None of this puts any pressure on the construal of psychology and cognitive science as the study of the mind and the mental.

However, there is another interesting question that can be raised. Some of the immune system's capacities might be psychological or cognitive because of the kinds of considerations above, but are not generally counted as part of the mind(-as-capacities). Suppose, for example, that orchestrating cell differentiation in response to viral infection turns out to be cognitive. There are good reasons to suspect that this is the case: it is amenable to cognitive models, relevant to understanding self-organization and flexible adaptivity, and underwritten by saliently similar mechanisms (notably, the sympathetic nervous system) as (other) cognitive capacities.

Importantly, none of this turns on whether or not orchestrating cell-differentiation in response to viral infection is ever involved in the implementation of conventionally mental capacities. More generally, being involved in the implementation of such capacities is far from the only reason that a capacity not conventionally counted as mental might be cognitive. The point generalizes to psychology: there are good reasons to believe that whatever processes lower social motivation following peripheral inflammation are psychological—after all, these processes generate complex, social behavior on a short time-scale and may over time alter personality traits. However, there is no good reason to believe that these processes can be construed as implementing a traditionally mental capacity.<sup>6</sup>

This ties into a different strain of inquiry within the embodied mind tradition, especially evident in the work of Varela: questioning our categorization of capacities as mental or not. Cognitive science and psychology are both widely construed as the study of the mind (see Boden 2006; Mandler 2007). If it turns out that there are capacities which are cognitive or psychological but not mental, we have two options: deny that cognitive science/psychology is the study of the mind, since there are non-mental capacities in the domain of cognitive science/psychology; or retain this construal of cognitive science/psychology by changing which capacities we count as mental—for example, counting orchestrating cell differentiation in response to viral infection as a mental capacity.

There are several reasons, I believe, to prefer the former option. For one, I believe that although psychology and cognitive science are both

6. This is not a problem specific to the immune system and its capacities. For example, the brain's role in autonomic regulation might be both psychological and cognitive, even though *autonomic regulation* is not a traditionally "mental" capacity.



construed as the study of the mind and the mental, the psychological and the cognitive can come apart because of divergence in their explanatory interests. For example, while orchestrating cell differentiation in response to viral infection may well be cognitive, I do not believe that there is any reason to suspect that it is psychological. Psychology does not appear to share cognitive science's interest in the breadth of adaptive behavior and self-organization—it instead appears to remain more firmly focused on understanding complex behavior, personality traits, and the like. There is no reason to think that psychologists should take an interest in this particular immune capacity, because there is no reason to think that this capacity is particularly relevant to those aims. As such, if we revise our understanding of the mental to make the mental and the cognitive align, then we would either create (or at least increase) a misalignment between the mental and the psychological, or introduce further ambiguity to the term “mental.”

Construing psychology and cognitive science as studying the mind and the mental is therefore doomed to be difficult—it will be revisionary, and probably involve the introduction of further ambiguity to the terms. Worse still, construing the disciplines this way does not even seem particularly desirable. The terms “mind” and “mental” appear to have a range of uses much broader than the terms “psychological” and “cognitive”: “mind” and “mental” have a wide-range of uses, metaphysical, ethical, and idiomatic. They are, as such, not particularly informative terms to use in characterizing psychology and cognitive science. Personally, I prefer the terms “psychological” and “cognitive” because they are at least more obviously associated with the kind of pragmatic concerns that I think are relevant to determining the subject-matter of psychology and cognitive science.

As well as being uninformative, “mind” and “mental” come with connotations which at least sometimes make them actively misleading, and actively harmful, as characterizations of psychology and cognitive science. Essentially, I believe that the terms “mind” and “mental” are so ambiguous, in such a disorderly way, that they introduce an entirely unnecessary risk of conflation, miscommunication, and misinterpretation (consider, e.g., Taylor and Vickers 2017). While this is a general problem of using these terms in this way, the danger is most pertinently embodied in the history of psychoneuroimmunology.

For too much of its early life, psychoneuroimmunology was marred by skepticism and neglect—although as discussed in section 2, this skepticism and neglect have now largely been overcome. It was often viewed as pseudoscientific and not worth engaging with. This was a barrier to greater progress in the area, and to more clinical uptake of its findings (e.g., Brod et al. 2014; D'Acquisto 2017; Daruna 2012). Interestingly,

there is not much evidence that the skepticism directed towards psychoneuroimmunology is driven by some sort of Cartesian strawman view according to which mind and body are separated by an unbridgeable divide, nor any skepticism of top-down causation (see also Harrington 2008).

In fact, the problem is quite the opposite. Certain supposed supporters of psychoneuroimmunology accidentally create a façade of snake oil salesmanship and illegitimacy over what is fundamentally a cautious and well-evidenced branch of medical science. For example, many textbooks and articles on the topic (e.g., the otherwise excellent Daruna 2012) draw uncritically on an origin story that makes reference to practices of alternative medicine, in particular, a set of practices thought of as traditional Chinese medical practices. These practices are understood through a highly orientalist lens. It is ignored, for example, that this set of practices was deliberately constructed by Mao in an attempt to create a new Chinese cultural identity and lessen dependence on Russia (again, see Harrington 2008). Additionally, these practices are presented as stemming from a holistic Chinese worldview—but as Slingerland (2019) carefully argues, the idea of Chinese holism is an orientalist myth.

This entirely unforced own goal is particularly associated with the popular construal of psychoneuroimmunology as (a branch of) mind-body medicine (e.g., Harrington 2008; Yan 2016). Mind-body medicine includes many approaches to medicine that look at interactions between the traditionally mental and the traditionally bodily. Much mind-body medicine is entirely legitimate (which I hope goes without saying), studying the interaction between the complex and overlapping systems that a human being comprises, and multisystem phenomena such as stress. Unfortunately, the label “mind-body medicine” is also applied to large swathes of alternative medicine and many exaggerated claims—work that is at best legitimate but nonmedical self-help, and at worst con artistry.

This is no coincidence—there is an element of symbiosis according to Harrington (2008), who herself is an influential researcher in the area. Medical practitioners and researchers benefit in the short term from the label “mind-body medicine” by being perceived to be pushing at the boundaries of the Western worldview, rediscovering ancient wisdom, and most importantly, working on topics that excite the public. Conversely, those selling snake oil benefit because they can inherit some of the medical and scientific legitimacy and prestige associated with legitimate areas of research like psychoneuroimmunology.

I suspect that the terms “mind” and “mental” play no small part in enabling this deeply undesirable situation. The seamless slides between serious science and unserious semi-spiritual self-help are enabled by the

multifaceted and multitudinous ways in which “mind” and “mental” can be used. There are uses of the terms associated with psychology and cognitive science, uses associated with discussions of the mind-body problem, uses associated with religion and spirituality, and uses associated with self-help and pop psychology. This creates a superficial appearance that, in some sense, all these areas are talking about the same thing, priming people to see findings in one area as relevant to the others.

Robert Ader saw much of this coming. In 1999, in an interview with Beth Azar, he worried that the discipline would

be undermined by “so-called-friends”—clinicians and researchers who embrace the idea of holistic or alternative medicine too zealously and use [psychoneuroimmunology] data to legitimize claims for all types of alternative therapies. (Robert Ader, in Azar 1999)

As he put it,

The basic research isn’t a fad ... but the way some people are using the term [“psychoneuroimmunology”] could turn it into a fad. Some of our biggest followers and fans are trashing it ... If you’re an immunologist and you read a lay magazine about how psychoneuroimmunology means you can boost your immune system and make you healthy, wealthy and wise, you’re not going to want any part of it. (Robert Ader, in Azar 1999)

In short, Ader worried that people would be understandably put off the discipline because of the way its results were being used to support “holistic and alternative medicine” and to promise a new kind of self-help that will also make you healthy—thereby turning a legitimate discipline into a mere fad. Inasmuch as these worries have been borne out, it has been supported and facilitated by the construal of psychoneuroimmunology as to do with the mind and the mental. Overall, then, it may be better just to abandon these terms, in order to avoid the risks of misunderstanding and miscommunication that result from their misleading connotations and unwanted associations.

It is important to clarify the nature of my objection to the characterization of psychology and cognitive science as studying the mental. The problem is not just that the terms “mind” and “mental” are ambiguous or vague. Plenty of scientific terms are ambiguous and vague—indeed, this is often an advantage (e.g., Neto 2020). However, it is an advantage when, and because, it facilitates desirable sorts of communication and integration. My issue here is that the supposed communication and integration facilitated by the terms “mind” and “mental” is of an undesirable sort, building bridges between psychology and cognitive science (in particular,

psychoneuroimmunology), self-help, and sloppy orientalist holism—bridges that it is in the best interest of all but a few con artists to burn.

## 5. Conclusion

I began by surveying some important results in psychoneuroimmunology and immunopsychiatry in section 2. In section 3, I argued that these results show that the immune system and some of its capacities are psychological and cognitive, in the sense of being part of the proper subject-matter of psychology and cognitive science. In section 4, I considered what this means for the construal of cognitive science and psychology as the study of the mind and the mental. There, I argued that there is a dilemma to be faced: abandon this construal of psychology and cognitive science, or count the immune system and many of its capacities as mental. Finally, on the basis of psychoneuroimmunology's short history, I argued that the former option is to be preferred, because the terms "mind" and "mental" are simply too ambiguous and disorderly, with too many unhelpful connotations, to be fit for characterizing scientific disciplines.

Going forward, another particularly interesting question is: to which other systems arguments like this one might be applied? There is a great deal of relevant empirical work going on, often associated with psychiatry, on the interactions between mind, body, and brain—work looking at interoception and the importance of the viscera and the monitoring thereof for cognition and mental health (e.g., Quaidt et al. 2018), work looking at the interactions between gut, brain, and microbiota (e.g., Cryan et al. 2020), and work looking at the interaction between the brain and the endocrine system (e.g., Leng 2018; Sapolsky 2017). Multi-system phenomena like stress are particularly important in understanding how these systems interact and communicate, and how they relate to person-level behavior and states (see McEwen 1998; McEwen and Wingfield 2003). Formal theoretical frameworks are in the works which provide a common language for understanding the workings of these many systems, and their interaction (Friston 2010, 2012, 2013; Friston et al. 2006; Friston, Thornton, and Clark 2012; Pezzulo et al. 2015). It is surely among the most interesting questions for the philosophy of mind how to account for these systems that seem to be at once paradigmatically bodily, shockingly sophisticated, and in many ways overlapping with traditionally mental systems.

## References

- Ader, R. 1976. "Conditioned Adrenocortical Steroid Elevations in the Rat." *Journal of Comparative and Physiological Psychology* 90(12): 1156–1163. <https://doi.org/10.1037/h0077290>, PubMed: 993394

- Ader, R. 1980. "Psychosomatic and Psychoimmunologic Research." *Psychosomatic Medicine* 42(3): 307–321. <https://doi.org/10.1097/00006842-198005000-00001>, PubMed: 7192419
- Ader, R. 2000. "On the Development of Psychoneuroimmunology." *European Journal of Pharmacology* 405(1–3): 167–176. [https://doi.org/10.1016/S0014-2999\(00\)00550-1](https://doi.org/10.1016/S0014-2999(00)00550-1), PubMed: 11033324
- Ader, R., and N. Cohen. 1975. "Behaviorally Conditioned Immunosuppression." *Psychosomatic Medicine* 37(4): 333–340. <https://doi.org/10.1097/00006842-197507000-00007>, PubMed: 1162023
- Ader, R., and N. Cohen. 1982. "Behaviorally Conditioned Immunosuppression and Murine Systemic Lupus Erythematosus." *Science* 215(4539): 1534–1536. <https://doi.org/10.1126/science.7063864>, PubMed: 7063864
- Ader, R., and N. Cohen. 1985. "CNS-Immune System Interactions: Conditioning Phenomena." *Behavioral and Brain Sciences* 8(3): 379–395. <https://doi.org/10.1017/S0140525X00000765>
- Ader, R., and N. Cohen. 1993. "Psychoneuroimmunology: Conditioning and Stress." *Annual Review of Psychology* 44: 53–85. <https://doi.org/10.1146/annurev.ps.44.020193.000413>, PubMed: 8434895
- Ader, R., D. Felten, and N. Cohen. 1990. "Interactions Between the Brain and the Immune System." *Annual Review of Pharmacology and Toxicology* 30: 561–602. <https://doi.org/10.1146/annurev.pa.30.040190.003021>, PubMed: 2188579
- Agorastos, A., R. L. Hauger, D. A. Barkauskas, I. R. Lerman, T. Moeller-Bertram, C. Snijders, ... D. G. Baker. 2019. "Relations of Combat Stress and Posttraumatic Stress Disorder to 24-h Plasma and Cerebrospinal Fluid Interleukin-6 levels and Circadian Rhythmicity." *Psychoneuroendocrinology* 100: 237–245. <https://doi.org/10.1016/j.psyneuen.2018.09.009>, PubMed: 30390522
- Akagi, M. 2018. "Rethinking the Problem of Cognition." *Synthese* 195(8): 3547–3570. <https://doi.org/10.1007/s11229-017-1383-2>
- Allen, C. 2017. "On (Not) Defining Cognition." *Synthese* 194(11): 4233–4249. <https://doi.org/10.1007/s11229-017-1454-4>
- Allen, M. S., S. Laborde, and E. E. Walter. 2019. "Health-related Behavior Mediates the Association Between Personality and Memory Performance in Older Adults." *Journal of Applied Gerontology* 38(2): 232–252. <https://doi.org/10.1177/0733464817698816>, PubMed: 28380727
- Amit, I., A. Regev, and N. Hacohen. 2011. Strategies to Discover Regulatory Circuits of the Mammalian Immune System. *Nature Reviews Immunology* 11(12): 873–880. <https://doi.org/10.1038/nri3109>, PubMed: 22094988

- Auer, B. J., J. L. Calvi, N. M. Jordan, D. Schrader, and J. Byrd-Craven. 2018. Communication and Social Interaction Anxiety Enhance Interleukin-1 Beta and Cortisol Reactivity During High-stakes Public Speaking. *Psychoneuroendocrinology* 94: 83–90. <https://doi.org/10.1016/j.psyneuen.2018.05.011>, PubMed: 29775877
- Azar, B. 1999. “Father of PNI Reflects on the Field’s Growth.” *Monitor on Psychology* 30(6). Retrieved from <https://www.apa.org/monitor/jun02/brightfuture>.
- Banks, W. A. 2016. “From Blood–Brain Barrier to Blood–Brain Interface: New Opportunities for CNS Drug Delivery.” *Nature Reviews Drug Discovery* 15(4): 275–292. <https://doi.org/10.1038/nrd.2015.21>, PubMed: 26794270
- Bechtel, W., and L. Bich. 2021. “Grounding Cognition: Heterarchical Control Mechanisms in Biology.” *Philosophical Transactions of the Royal Society B: Biological Sciences* 376(1820): 20190751. <https://doi.org/10.1098/rstb.2019.0751>, PubMed: 33487110
- Beiermeister, K. A., B. M. Keck, Z. C. Sifri, I. O. ElHassan, E. J. Hannoush, W. D. Alzate, ... A. M. Mohr. 2010. “Hematopoietic Progenitor Cell Mobilization is Mediated Through Beta-2 and Beta-3 Receptors After Injury.” *Journal of Trauma* 69(2): 338–343. <https://doi.org/10.1097/TA.0b013e3181e5d35e>, PubMed: 20699742
- Besedovsky, H. O., A. del Rey, I. Klusman, H. Furukawa, G. Monge Arditi, and A. Kabiersch. 1991. “Cytokines as Modulators of the Hypothalamus–Pituitary–Adrenal Axis.” *The Journal of Steroid Biochemistry and Molecular Biology* 40(4–6): 613–618. [https://doi.org/10.1016/0960-0760\(91\)90284-C](https://doi.org/10.1016/0960-0760(91)90284-C), PubMed: 1659887
- Bhat, A., T. Parr, M. Ramstead, and K. Friston. 2021. “Immunceptive Inference: Why Are Psychiatric Disorders and Immune Responses Intertwined?” *Biology & Philosophy* 36(3): 1–24. <https://doi.org/10.1007/s10539-021-09801-6>, PubMed: 33948044
- Blalock, J. E. 2005. “The Immune System as the Sixth Sense.” *The Journal of Internal Medicine* 257(2): 126–138. <https://doi.org/10.1111/j.1365-2796.2004.01441.x>, PubMed: 15656872
- Block, N. 2016. “Tweaking the Concepts of Perception and Cognition.” *Behavioral and Brain Sciences* 39: e232. <https://doi.org/10.1017/S0140525X15002733>, PubMed: 28355865
- Boden, M. A. 2006. *Mind as Machine: A History of Cognitive Science*. Oxford; New York: Clarendon Press; Oxford University Press.
- Bogg, T., and B. W. Roberts. 2004. “Conscientiousness and Health-related Behaviors: A Meta-analysis of the Leading Behavioral Contributors to Mortality.” *Psychological Bulletin* 130(6): 887–919. <https://doi.org/10.1037/0033-2909.130.6.887>, PubMed: 15535742

- Bosch, J. A., C. Ring, E. J. de Geus, E. C. Veerman, and A. V. Amerongen. 2002. Stress and Secretory Immunity. *International Review of Neurobiology* 52: 213–253. [https://doi.org/10.1016/S0074-7742\(02\)52011-0](https://doi.org/10.1016/S0074-7742(02)52011-0), PubMed: 12498106
- Bovbjerg, D., R. Ader, and N. Cohen. 1982. “Behaviorally Conditioned Suppression of a Graft-versus-host Response.” *Proceedings of the National Academy of Sciences* 79(2): 583–585. <https://doi.org/10.1073/pnas.79.2.583>, PubMed: 6952209
- Broadbent, E., and H. E. Koschwanez. 2012. “The Psychology of Wound Healing.” *Current Opinion in Psychiatry* 25(2): 135–140. <https://doi.org/10.1097/YCO.0b013e32834e1424>, PubMed: 22156976
- Brod, S., L. Rattazzi, G. Piras, and F. D’Acquisto. 2014. “‘As Above, so Below’ Examining the Interplay Between Emotion and the Immune System.” *Immunology* 143(3): 311–318. <https://doi.org/10.1111/imm.12341>, PubMed: 24943894
- Chiurchiù, V., and M. Maccarrone. 2016. “Bioactive Lipids as Modulators of Immunity, Inflammation and Emotions.” *Current Opinion in Pharmacology* 29: 54–62. <https://doi.org/10.1016/j.coph.2016.06.005>, PubMed: 27372887
- Churchland, P. S., and P. Winkelman. 2012. “Modulating Social Behavior with Oxytocin: How Does It Work? What Does It Mean?” *Hormones and Behavior* 61(3): 392–399. <https://doi.org/10.1016/j.yhbeh.2011.12.003>, PubMed: 22197271
- Cohen, I. R., and S. Efroni. 2019. “The Immune System Computes the State of the Body: Crowd Wisdom, Machine Learning, and Immune Cell Reference Repertoires Help Manage Inflammation.” *Frontiers in Immunology* 10: 10. <https://doi.org/10.3389/fimmu.2019.00010>, PubMed: 30723470
- Corcoran, A. W., G. Pezzulo, and J. Hohwy. 2020. “From Allostatic Agents to Counterfactual Cognisers: Active Inference, Biological Regulation, and the Origins of Cognition.” *Biology & Philosophy* 35(3): 32. <https://doi.org/10.1007/s10539-020-09746-2>
- Cryan, J. F., K. J. O’Riordan, K. Sandhu, V. Peterson, and T. G. Dinan. 2020. “The Gut Microbiome in Neurological Disorders.” *The Lancet Neurology* 19(2): 179–194. [https://doi.org/10.1016/S1474-4422\(19\)30356-4](https://doi.org/10.1016/S1474-4422(19)30356-4), PubMed: 31753762
- D’Acquisto, F. 2017. “Affective Immunology: Where Emotions and the Immune Response Converge.” *Dialogues in Clinical Neuroscience* 19(1): 9–19. <https://doi.org/10.31887/DCNS.2017.19.1/fdacquisto>, PubMed: 28566943
- Dantzer, R. 1994. “How do Cytokines Say Hello to the Brain? Neural Versus Humoral Mediation.” *European Cytokine Network* 5(3): 271–273. PubMed: 7948764

- Dantzer, R., R.-M. Bluthé, S. Layé, J. L. Bret-Dibat, P. Parnet, and K. W. Kelley. 1998. "Cytokines and Sickness Behavior." *Annals of the New York Academy of Sciences* 840(1): 586–590. <https://doi.org/10.1111/j.1749-6632.1998.tb09597.x>, PubMed: 9629285
- Dantzer, R., J.-P. Konsman, R.-M. Bluthé, and K. W. Kelley. 2000. "Neural and Humoral Pathways of Communication From the Immune System to the Brain: Parallel or Convergent?" *Autonomic Neuroscience* 85(1–3): 60–65. [https://doi.org/10.1016/S1566-0702\(00\)00220-4](https://doi.org/10.1016/S1566-0702(00)00220-4), PubMed: 11189027
- Daruna, J. H. 2012. *Introduction to Psychoneuroimmunology*. Cambridge: Academic Press. <https://doi.org/10.1016/C2009-0-01965-5>
- DeAngelis, T. 2002. "A Bright Future for PNI." *Monitor on Psychology* 33(6). Retrieved from <https://www.apa.org/monitor/jun02/brightfuture>.
- Eisenberger, N. I., E. T. Berkman, T. K. Inagaki, L. T. Rameson, N. M. Mashal, and M. R. Irwin. 2010. "Inflammation-Induced Anhedonia: Endotoxin Reduces Ventral Striatum Responses to Reward." *Biological Psychiatry* 68(8): 748–754. <https://doi.org/10.1016/j.biopsych.2010.06.010>, PubMed: 20719303
- Eisenberger, N. I., T. K. Inagaki, N. M. Mashal, and M. R. Irwin. 2010. "Inflammation and Social Experience: An Inflammatory Challenge Induces Feelings of Social Disconnection in Addition to Depressed Mood." *Brain, Behavior, and Immunity* 24(4): 558–563. <https://doi.org/10.1016/j.bbi.2009.12.009>, PubMed: 20043983
- Engeland, C. G., F. N. Hugo, J. B. Hilgert, G. G. Nascimento, R. Junges, H. J. Lim, ... J. A. Bosch. 2016. "Psychological Distress and Salivary Secretory Immunity." *Brain, Behavior, and Immunity* 52: 11–17. <https://doi.org/10.1016/j.bbi.2015.08.017>, PubMed: 26318411
- Entringer, S. 2021. "Childhood Abuse Accelerates Inflammaging." *Brain, Behavior, and Immunity* 94: 25–26. <https://doi.org/10.1016/j.bbi.2021.01.038>, PubMed: 33581248
- Erickson, M. A., and W. A. Banks. 2018. "Neuroimmune Axes of the Blood–Brain Barriers and Blood–Brain Interfaces: Bases for Physiological Regulation, Disease States, and Pharmacological Interventions." *Pharmacological Reviews* 70(2): 278–314. <https://doi.org/10.1124/pr.117.014647>, PubMed: 29496890
- Feldman, R. 2012. "Bio-behavioral Synchrony: A Model for Integrating Biological and Microsocial Behavioral Processes in the Study of Parenting." *Parenting* 12(2–3): 154–164. <https://doi.org/10.1080/15295192.2012.683342>
- Felten, D. L., and S. Y. Felten. 1988. "Sympathetic Noradrenergic Innervation of Immune Organs." *Brain, Behavior, and Immunity* 2(4):



- 293–300. [https://doi.org/10.1016/0889-1591\(88\)90031-1](https://doi.org/10.1016/0889-1591(88)90031-1), PubMed: 3076478
- Figdor, C. 2017. “On the Proper Domain of Psychological Predicates.” *Synthese* 194(11): 4289–4310. <https://doi.org/10.1007/s11229-014-0603-2>
- Figdor, C. 2018. *Pieces of Mind: The Proper Domain of Psychological Predicates*. Oxford: Oxford University Press. <https://doi.org/10.1093/oso/9780198809524.001.0001>
- Fodor, J. A. 1983. *The Modularity of Mind*. Cambridge, MA: MIT Press. <https://doi.org/10.7551/mitpress/4737.001.0001>
- Forrest, S., and S. Hofmeyr. 2001. “Immunology as Information Processing.” Pp. 361–387 in *Design Principles for Immune System & Other Distributed Autonomous Systems*. Edited by L. A. Segel and I. R. Cohen. Oxford: Oxford University Press.
- Friston, K. 2010. “Is the Free-Energy Principle Neurocentric?” *Nature Reviews Neuroscience* 11(8): 605. <https://doi.org/10.1038/nrn2787-c2>, PubMed: 20631713
- Friston, K. 2012. “A Free Energy Principle for Biological Systems.” *Entropy* 14(11): 2100–2121. <https://doi.org/10.3390/e14112100>, PubMed: 23204829
- Friston, K. 2013. “Life as We Know It.” *Journal of the Royal Society Interface* 10(86). <https://doi.org/10.1098/rsif.2013.0475>, PubMed: 23825119
- Friston, K., J. Kilner, and L. Harrison. 2006. “A Free Energy Principle for the Brain.” *Journal of Physiology-Paris* 100(1–3): 70–87. <https://doi.org/10.1016/j.jphysparis.2006.10.001>, PubMed: 17097864
- Friston, K., C. Thornton, and A. Clark. 2012. “Free-Energy Minimization and the Dark-Room Problem.” *Frontiers in Psychology* 3. <https://doi.org/10.3389/fpsyg.2012.00130>, PubMed: 22586414
- Gough, J. 2022. “Cognitive Science Meets the Mark of the Cognitive: Putting the Horse Before the Cart.” *Biology & Philosophy* 38(1): 1. <https://doi.org/10.1007/s10539-022-09889-4>
- Gouin, J.-P., and J. K. Kiecolt-Glaser. 2011. “The Impact of Psychological Stress on Wound Healing: Methods and Mechanisms.” *Immunology and Allergy Clinics of North America* 31(1): 81–93. <https://doi.org/10.1016/j.iac.2010.09.010>, PubMed: 21094925
- Harrington, A. 2008. *The Cure Within: A History of Mind-Body Medicine*. New York: WW Norton & Company.
- Hershberg, U., and S. Efroni. 2001. “The Immune System and Other Cognitive Systems.” *Complexity* 6(5): 14–21. <https://doi.org/10.1002/cplx.1046>
- Hesse, M. 1965. “Models and Analogies in Science.” *British Journal for the Philosophy of Science* 16(62): 161–163. <https://doi.org/10.1093/bjps/XVI.62.161>

- Howes, M. 2000. "Self, Intentionality, and Immunological Explanation." *Seminars in Immunology* 12(3): 249–256; discussion 257–344. <https://doi.org/10.1006/smim.2000.0238>, PubMed: 10910747
- Jerne, N. K. 1955. "The Natural-Selection Theory of Antibody Formation." *Proceedings of the National Academy of Sciences of the United States of America* 41(11), 849–857. <https://doi.org/10.1073/pnas.41.11.849>, PubMed: 16589759
- Jerne, N. K. 1974. "Towards a Network Theory of the Immune System." *Annales d'immunologie (Paris)* 125C(1–2): 373–389. PubMed: 4142565
- Jerne, N. K. 1985. "The Generative Grammar of the Immune System." *The EMBO Journal* 4(4): 847–852. <https://doi.org/10.1002/j.1460-2075.1985.tb03709.x>, PubMed: 2410261
- Katayama, Y., M. Battista, W.-M. Kao, A. Hidalgo, A. J. Peired, S. A. Thomas, and P. S. Frenette. 2006. "Signals from the Sympathetic Nervous System Regulate Hematopoietic Stem Cell Egress from Bone Marrow." *Cell* 124(2): 407–421. <https://doi.org/10.1016/j.cell.2005.10.041>, PubMed: 16439213
- Keller, E. F. 2002. *Making Sense of Life: Explaining Biological Development with Models, Metaphors, and Machines* (Vol. 36). Cambridge: Harvard University Press. <https://doi.org/10.4159/9780674039445>
- Kenny, A. 1989. *The Metaphysics of Mind*. Oxford: Oxford University Press.
- Khandaker, G. M., L. Cousins, J. Deakin, B. R. Lennox, R. Yolken, and P. B. Jones. 2015. "Inflammation and Immunity in Schizophrenia: Implications for Pathophysiology and Treatment." *The Lancet Psychiatry* 2(3): 258–270. [https://doi.org/10.1016/S2215-0366\(14\)00122-9](https://doi.org/10.1016/S2215-0366(14)00122-9), PubMed: 26359903
- Khandaker, G. M., R. Dantzer, and P. B. Jones. 2017. "Immunopsychiatry: Important Facts." *Psychological Medicine* 47(13): 2229–2237. <https://doi.org/10.1017/S0033291717000745>, PubMed: 28418288
- Krieger, N. 2005. "Embodiment: A Conceptual Glossary for Epidemiology." *Journal of Epidemiology & Community Health* 59(5): 350–355. <https://doi.org/10.1136/jech.2004.024562>, PubMed: 15831681
- Leng, G. 2018. "The Endocrinology of the Brain." *Endocrine Connections* 7(12): R275–R285. <https://doi.org/10.1530/EC-18-0367>, PubMed: 30352398
- Levy, J., K. Yirmiya, A. Goldstein, and R. Feldman. 2019. "Chronic Trauma Impairs the Neural Basis of Empathy in Mothers: Relations to Parenting and Children's Empathic Abilities." *Developmental Cognitive Neuroscience* 38: 100658. <https://doi.org/10.1016/j.dcn.2019.100658>, PubMed: 31121480
- Macpherson, A. J., K. D. McCoy, F. E. Johansen, and P. Brandtzaeg. 2008. "The Immune Geography of IgA Induction and Function." *Mucosal*

- Immunology* 1(1): 11–22. <https://doi.org/10.1038/mi.2007.6>, PubMed: 19079156
- Mandler, G. 2007. *A History of Modern Experimental Psychology: From James and Wundt to Cognitive Science*. London; Cambridge, MA: MIT Press. <https://doi.org/10.7551/mitpress/3542.001.0001>
- Matthen, M., and E. Levy. 1984. “Teleology, Error, and the Human Immune System.” *Journal of Philosophy* 81(7): 351–372. <https://doi.org/10.2307/2026291>
- Maurel, M., R. Castagné, E. Berger, M. Bochud, M. Chadeau-Hyam, S. Fraga, ... M. Kelly-Irving. 2020. “Patterning of Educational Attainment across Inflammatory Markers: Findings from a Multi-Cohort Study.” *Brain, Behavior, and Immunity* 90: 303–310. <https://doi.org/10.1016/j.bbi.2020.09.002>, PubMed: 32919037
- McEwen, B. S. 1998. “Stress, Adaptation, and Disease. Allostasis and Allostatic Load.” *Annals of the New York Academy of Sciences* 840: 33–44. <https://doi.org/10.1111/j.1749-6632.1998.tb09546.x>, PubMed: 9629234
- McEwen, B. S., and J. C. Wingfield. 2003. “The Concept of Allostasis in Biology and Biomedicine.” *Hormones and Behavior* 43(1): 2–15. [https://doi.org/10.1016/S0018-506X\(02\)00024-7](https://doi.org/10.1016/S0018-506X(02)00024-7), PubMed: 12614627
- Melander, P. 1993. “How Not to Explain the Errors of the Immune System.” *Philosophy of Science* 60(2): 223–241. <https://doi.org/10.1086/289730>
- Méndez-Ferrer, S., D. Lucas, M. Battista, and P. S. Frenette. 2008. “Haematopoietic Stem Cell Release is Regulated by Circadian Oscillations.” *Nature* 452(7186): 442–447. <https://doi.org/10.1038/nature06685>, PubMed: 18256599
- Miller, G. E., E. Chen, A. K. Fok, H. Walker, A. Lim, E. F. Nicholls, ... M. S. Kobor. 2009. “Low Early-life Social Class Leaves a Biological Residue Manifested by Decreased Glucocorticoid and Increased Proinflammatory Signaling.” *Proceedings of the National Academy of Sciences* 106(34): 14716–14721. <https://doi.org/10.1073/pnas.0902971106>, PubMed: 19617551
- Moieni, M., M. R. Irwin, I. Jevtic, E. C. Breen, and N. I. Eisenberger. 2015. “Inflammation Impairs Social Cognitive Processing: A Randomized Controlled Trial of Endotoxin.” *Brain, Behavior, and Immunity* 48: 132–138. <https://doi.org/10.1016/j.bbi.2015.03.002>, PubMed: 25770082
- Morgan, J. 2017. “If Other Ways Don’t Work, Try the Immune System?” *The Lancet Neurology* 16(2): 109. [https://doi.org/10.1016/S1474-4422\(16\)30307-6](https://doi.org/10.1016/S1474-4422(16)30307-6)
- Motsan, S., E. Bar-Kalifa, K. Yirmiya, and R. Feldman. 2021. “Physiological and Social Synchrony as Markers of PTSD and Resilience Following

- Chronic Early Trauma." *Depression and Anxiety* 38(1): 89–99. <https://doi.org/10.1002/da.23106>, PubMed: 33107687
- Murray, D. R., M. G. Haselton, M. Fales, and S. W. Cole. 2019. "Falling in Love is Associated with Immune System Gene Regulation." *Psychoneuroendocrinology* 100: 120–126. <https://doi.org/10.1016/j.psyneuen.2018.09.043>, PubMed: 30299259
- Neigh, G. N., and F. F. Ali. 2016. "Co-morbidity of PTSD and Immune System Dysfunction: Opportunities for Treatment." *Current Opinion in Pharmacology* 29: 104–110. <https://doi.org/10.1016/j.coph.2016.07.011>, PubMed: 27479489
- Neto, C. 2020. "When Imprecision is a Good Thing, or How Imprecise Concepts Facilitate Integration in Biology" *Biology and Philosophy* 35(6): 1–21. <https://doi.org/10.1007/s10539-020-09774-y>
- Pariente, C. M. 2015. "Psychoneuroimmunology or Immunopsychiatry?" *The Lancet Psychiatry* 2(3): 197–199. [https://doi.org/10.1016/S2215-0366\(15\)00042-5](https://doi.org/10.1016/S2215-0366(15)00042-5), PubMed: 26359887
- Pariente, C. M. 2019. "The Year of Immunopsychiatry: A Special Issue that Foresaw the Future." *Psychoneuroendocrinology* 103: 49–51. <https://doi.org/10.1016/j.psyneuen.2019.01.002>, PubMed: 30640037
- Peirce, C. S. 1878. "How To Make Our Ideas Clear." *Popular Science Monthly* 12(Jan.): 286–302.
- Penz, M., C. Kirschbaum, A. Buske-Kirschbaum, M. K. Wekenborg, and R. Miller. 2018. "Stressful Life Events Predict One-year Change of Leukocyte Composition in Peripheral Blood." *Psychoneuroendocrinology* 94: 17–24. <https://doi.org/10.1016/j.psyneuen.2018.05.006>, PubMed: 29751249
- Pezzulo, G., F. Rigoli, and K. Friston. 2015. "Active Inference, Homeostatic Regulation and Adaptive Behavioral Control." *Progress in Neurobiology* 134: 17–35. <https://doi.org/10.1016/j.pneurobio.2015.09.001>, PubMed: 26365173
- Pincock, S. 2012. "Robert Ader." *The Lancet* 379(9813): 308. [https://doi.org/10.1016/S0140-6736\(12\)60134-2](https://doi.org/10.1016/S0140-6736(12)60134-2)
- Powell, N. D., E. K. Sloan, M. T. Bailey, J. M. G. Arevalo, G. E. Miller, E. Chen, ... S. W. Cole. 2013. "Social Stress Up-Regulates Inflammatory Gene Expression in the Leukocyte Transcriptome via  $\beta$ -adrenergic Induction of Myelopoiesis." *Proceedings of the National Academy of Sciences* 110(41): 16574–16579. <https://doi.org/10.1073/pnas.1310655110>, PubMed: 24062448
- Pradeu, T. 2011. *The Limits of the Self: Immunology and Biological Identity*. Oxford University Press. <https://doi.org/10.1093/acprof:oso/9780199775286.001.0001>
- Pradeu, T. 2020. *Philosophy of Immunology*. Cambridge: Cambridge University Press. <https://doi.org/10.1017/9781108616706>

- Priest, N. 2021. "How Does the Social Environment Become Embodied to Influence Health and Health Inequalities?" *Brain, Behavior, and Immunity* 94: 23–24. <https://doi.org/10.1016/j.bbi.2021.02.006>, PubMed: 33607234
- Quaidt, L., H. D. Critchley, and S. N. Garfinkel. 2018. "The Neurobiology of Interception in Health and Disease." *Annals of the New York Academy of Sciences* 1428: 112–128. <https://doi.org/10.1111/nyas.13915>, PubMed: 29974959
- Ramsey, W. M. 2007. *Representation Reconsidered*. Cambridge: Cambridge University Press. <https://doi.org/10.1017/CBO9780511597954>
- Ramsey, W. M. 2017. "Must Cognition be Representational?" *Synthese* 194(11): 4197–4214. <https://doi.org/10.1007/s11229-014-0644-6>
- Reynolds, A. S. 2018. *The Third Lens: Metaphor and the Creation of Modern Cell Biology*. Chicago: University of Chicago Press. <https://doi.org/10.7208/chicago/9780226563435.001.0001>
- Rorty, R. 1970. "Incorrigibility as the Mark of the Mental." *The Journal of Philosophy* 67(12): 399–424. <https://doi.org/10.2307/2024002>
- Rorty, R. 1979. *Philosophy and the Mirror of Nature*. Oxford: Blackwell.
- Rorty, R. 1982. "Contemporary Philosophy of Mind." *Synthese* 53(2): 323–348. <https://doi.org/10.1007/BF00484908>
- Rosenberg, A. 1989. "Perceptual Presentations and Biological Function: A Comment on Matthen." *The Journal of Philosophy* 86(1): 38–44. <https://doi.org/10.2307/2027175>
- Rothenberg, E. V. 2014. "Transcriptional Control of Early T and B Cell Developmental Choices." *Annual Review of Immunology* 32: 283–321. <https://doi.org/10.1146/annurev-immunol-032712-100024>, PubMed: 24471430
- Roy, A. L. 2019. "Transcriptional Regulation in the Immune System: One Cell at a Time." *Frontiers in Immunology* 10: 1355. <https://doi.org/10.3389/fimmu.2019.01355>, PubMed: 31258532
- Rupert, R. D. 2013. "Memory, Natural Kinds, and Cognitive Extension; or, Martians Don't Remember, and Cognitive Science is Not About Cognition." *Review of Philosophy and Psychology* 4(1): 25–47. <https://doi.org/10.1007/s13164-012-0129-9>
- Ryle, G. 1949. *The Concept of Mind*. London: Penguin.
- Sapolsky, R. M. 2017. *Behave: The Biology of Humans at Our Best and Worst*. New York, NY: Penguin Press.
- Segerstrom, S. C. 2000. "Personality and the Immune System: Models, Methods, and Mechanisms." *Annals of Behavioral Medicine* 22(3): 180–190. <https://doi.org/10.1007/BF02895112>, PubMed: 11126462
- Shields, G. S., C. M. Spahr, and G. M. Slavich. 2020. "Psychosocial Interventions and Immune System Function: A Systematic Review and

- Meta-analysis of Randomized Clinical Trials." *JAMA Psychiatry* 77(10): 1031–1043. <https://doi.org/10.1001/jamapsychiatry.2020.0431>, PubMed: 32492090
- Slavich, G. M., M. Giletta, S. W. Helms, P. D. Hastings, K. D. Rudolph, M. K. Nock, and M. J. Prinstein. 2020. "Interpersonal Life Stress, Inflammation, and Depression in Adolescence: Testing Social Signal Transduction Theory of Depression." *Depression and Anxiety* 37(2): 179–193. <https://doi.org/10.1002/da.22987>, PubMed: 31995664
- Slavish, D. C., J. E. Graham-Engeland, J. M. Smyth, and C. G. Engeland. 2015. "Salivary Markers of Inflammation in Response to Acute Stress." *Brain, Behavior, and Immunity* 44: 253–269. <https://doi.org/10.1016/j.bbi.2014.08.008>, PubMed: 25205395
- Slavish, D. C., and Y. Z. Szabo. 2019. "The Effect of Acute Stress on Salivary Markers of Inflammation: A Systematic Review Protocol." *Systematic Reviews* 8(1): 108. <https://doi.org/10.1186/s13643-019-1026-4>, PubMed: 31046830
- Slingerland, E. 2019. *Mind and Body in Early China: Beyond Orientalism and the Myth of Holism*. Oxford: Oxford University Press. <https://doi.org/10.1093/oso/9780190842307.001.0001>
- Smale, S. T., and A. G. Fisher. 2002. "Chromatin Structure and Gene Regulation in the Immune System." *Annual Review of Immunology* 20: 427–462. <https://doi.org/10.1146/annurev.immunol.20.100301.064739>, PubMed: 11861609
- Smale, S. T., A. Tarakhovsky, and G. Natoli. 2014. "Chromatin Contributions to the Regulation of Innate Immunity." *Annual Review of Immunology* 32: 489–511. <https://doi.org/10.1146/annurev-immunol-031210-101303>, PubMed: 24555473
- Swiatczak, B., and A. I. Tauber. 2020. "Philosophy of Immunology." *Stanford Encyclopedia of Philosophy*.
- Tauber, A. I. 2000. "Moving Beyond the Immune Self?" *Seminars in Immunology* 12(3): 241–248; discussion 257–344. <https://doi.org/10.1006/smim.2000.0237>, PubMed: 10910746
- Tauber, A. I. 2013. "Immunology's Theories of Cognition." *History and Philosophy of the Life Sciences* 35(2): 239–264. PubMed: 24466634
- Tauber, A. I. 2017. "Whither Immune Identity?" Pp. 57–88 in *Immunity: The Evolution of an Idea*. New York: Oxford University Press. <https://doi.org/10.1093/acprof:oso/9780190651244.003.0002>
- Taylor, H., and P. Vickers. 2017. "Conceptual Fragmentation and the Rise of Eliminativism." *European Journal for Philosophy of Science* 7(1): 17–40. <https://doi.org/10.1007/s13194-016-0136-2>
- Ulmer-Yaniv, A., R. Avitsur, Y. Kanat-Maymon, I. Schneiderman, O. Zagoory-Sharon, and R. Feldman. 2016. "Affiliation, Reward, and



- Immune Biomarkers Coalesce to Support Social Synchrony During Periods of Bond Formation in Humans.” *Brain, Behavior, and Immunity* 56: 130–139. <https://doi.org/10.1016/j.bbi.2016.02.017>, PubMed: 26902915
- Ulmer-Yaniv, A., A. Djalovski, K. Yirmiya, G. Halevi, O. Zagoory-Sharon, and R. Feldman. 2018. “Maternal Immune and Affiliative Biomarkers and Sensitive Parenting Mediate the Effects of Chronic Early Trauma on Child Anxiety.” *Psychological Medicine* 48(6): 1020–1033. <https://doi.org/10.1017/S0033291717002550>, PubMed: 28889808
- Varela, F. J. 1979. *Principles of Biological Autonomy*. New York: North Holland.
- Varela, F. J., E. Thompson, and E. Rosch. 2016. *The Embodied Mind: Cognitive Science and Human Experience*. Cambridge: MIT Press. <https://doi.org/10.7551/mitpress/9780262529365.001.0001>
- Wang, Z., B. Caughron, and M. R. I. Young. 2017. “Posttraumatic Stress Disorder: An Immunological Disorder?” *Frontiers in Psychiatry* 8: 222. <https://doi.org/10.3389/fpsy.2017.00222>, PubMed: 29163241
- Wilson, R. A., and L. Foglia. 2017. “Embodied Cognition.” *The Stanford Encyclopedia of Philosophy (Spring 2017 Edition)*.
- Yan, Q. 2016. *Psychoneuroimmunology: Systems Biology Approaches to Mind-Body Medicine*. Springer. <https://doi.org/10.1007/978-3-319-45111-4>
- Yirmiya, R. 1996. “Endotoxin Produces a Depressive-like Episode in Rats.” *Brain Research* 711(1–2): 163–174. [https://doi.org/10.1016/0006-8993\(95\)01415-2](https://doi.org/10.1016/0006-8993(95)01415-2), PubMed: 8680860
- Yirmiya, K., A. Djalovski, S. Motsan, O. Zagoory-Sharon, and R. Feldman. 2018. “Stress and Immune Biomarkers Interact with Parenting Behavior to Shape Anxiety Symptoms in Trauma-Exposed Youth.” *Psychoneuroendocrinology* 98: 153–160. <https://doi.org/10.1016/j.psyneuen.2018.08.016>, PubMed: 30149270