

Methodological functionalism and the description of natural systems

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ABSTRACT

The primary way that explanations are constructed in cognitive psychology is by methodological functionalism: in short, functionally defined components are proposed in order to explain how inputs (i.e., stimuli from the environment) are turned into behavior. But despite its close association with cognitive psychology, methodological functionalism is a technique that can be used to describe any natural system. I look at how methodological functionalism has fared when used by other special sciences and what lessons can be learned from these cases. Three explanations of chemical and biological systems that were developed using methodological functionalism are examined: Willis's (1684) explanation of fermentation, Farr's (mid-1800s) explanation of cholera, and Mendel's (mid-1800s) explanation of inheritance. The discovery of HIV in the early 1980s, an investigation that rejected methodological functionalism early on, is also discussed. The assessment of methodological functionalism is not positive. This technique has limitations. The implications for cognitive psychology are considered, and one conclusion is that cognitive psychology will eventually cease relying on methodological functionalism.

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1. Introduction

Since the cognitive revolution in the 1950s, a mainstay of psychology has been *methodological functionalism*, the technique used to generate descriptions of the mind without actually looking inside the head. While the application of methodological functionalism in cognitive psychology has clearly been fruitful, the technique and the explanations that it produces deserve a closer look. It is not a technique that is used regularly by any of the other special sciences, and, historically, when it has been used in other disciplines, it was eventually set aside when different investigative techniques became available.

Before going any further, let's review the central features of methodological functionalism. There are many different ways of investigating and explaining the operation of a natural system. Methodological functionalism is a technique that is used when the existence of a system is inferred from its outputs. To investigate such a system, one that cannot be directly inspected, the inputs that the system receives and the outputs that it produces are monitored. A set of components (or intervening variables), each of which performs a particular function, are then proposed. Collectively, these components are treated as the parts of the system. It is by their performance—at least in principle—that the output or outputs are generated. When a description is developed by this technique, the system has been described using

methodological functionalism. The primary alternatives, then, to methodological functionalism are any of the techniques that investigate and track the actual (physical) entities that participate in the system.

Some examples of the application of methodological functionalism in cognitive psychology are discussed in the next section. My main interest, however, is historical: examining how methodological functionalism has been used in biological and chemical investigations and what lessons can be learned from these cases. The takeaway will be largely negative. Despite its prevalence in cognitive psychology, methodological functionalism has a poor track record in the other special sciences. It tends to produce incorrect explanations of natural systems and is not used when the alternative, a direct investigation, is possible.

2. Methodological functionalism and psychology

2.1. Methodological functionalism and function

First, some clarification about how the term *function* is used in methodological functionalism. In other contexts, *function* refers to the job or role that a specific object has—for example, the role that the crankshaft has in a car, the liver in an animal, or a sodium channel in a nerve cell. In the case of the crankshaft, liver, and sodium channel, the physical entity has been identified, and any interest in its function is an interest in the task that the entity performs. With methodological functionalism, the story is somewhat different. There is the function that is assigned to each component, but there is no known physical object that performs that function. The functional role that has been proposed basically creates the component. Conceivably, a physical entity that performs the function might be identified in the future, but as far as methodological functionalism goes, physical entities are not part of the investigation or the resulting account.

As a useful illustration, take Ned Block's simple description of the process by which humans recognize words:

This word-recognizer might be composed of three components, one of which has the task of fetching each incoming word, one at a time, and passing it to a second component. The second component includes a dictionary, i.e., a list of all the words in the vocabulary, together with syntactic and semantic information about each word. This second component compares the target word with words in the vocabulary (perhaps executing many such comparisons simultaneously) until it gets a match. When it finds a match, it sends a signal to a third component whose job it is to retrieve the syntactic and semantic information stored in the dictionary. (1995, pp. 385–386)

In most contexts, of course, a dictionary is a physical object. But in Block's description of the word recognition process, the dictionary and other components are purely functional. They have no physical qualities and exist only for the purpose of explaining how the inputs to the system might be turned into outputs.

Now contrast that description with one that does invoke physical entities. Here is a description of the process that occurs in the small intestine of cholera victims:

Cholera toxin binds to a specific receptor, monosialosyl ganglioside, present on the surface of intestinal mucosal cells, [and] it activates adenylate cyclase in cells of the intestinal mucosa. The net effect of the toxin is to cause cAMP to be produced at an abnormally high rate. This stimulates mucosal cells to pump large amounts of chloride into the intestinal contents. Water, sodium, and other electrolytes follow due to the osmotic and electrical gradients caused by the loss of chloride. Water and electrolytes lost in mucosal cells are replaced from the blood. Thus, the toxin-damaged cells act as pumps for water and electrolytes causing the typical isotonic diarrhea that is characteristic of cholera. (Shetty, Tang, & Andrews, 2009, p. 71)

In this description, there are physical entities, for instance, the cholera toxin (which is a protein complex), the monosialosyl ganglioside, and the mucosal cells. And these entities perform specific functions. The cells in the lining of the small intestine pump chloride into the small intestine, a process that, in turn, depends on the functions performed by the cholera toxin and adenylate cyclase. But despite the importance of each functional role, these are still physical entities with identifiable physical qualities.

This distinction between (i) components that are purely functional and (ii) physical entities that perform various functions is straightforward. It does, however, cut across a common way of thinking

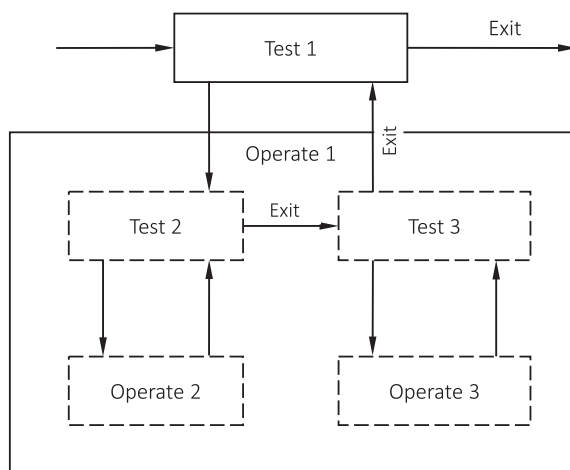


Figure 1. The TOTE unit (test, operate, test, exit), developed by Miller, Galanter, and Pribram (1960), begins with a goal and then proceeds with a series of tests and adjustments until the goal is achieved. In the figure, test 1 and operation 1 are one TOTE unit. Embedded in operation 1 are two additional TOTE units. If, according to test 1, the goal is achieved, the unit is exited. If the goal is not achieved, operation 1 is performed. That involves test 2 and, possibly, an adjustment by operation 2, and then test 3 and, possibly, an adjustment by operation 3. Figure adapted from Wright and Bechtel (2007, p. 41).

about function. Block, again, is helpful here. While distinguishing between functional kinds and structural kinds, he describes *functional kinds* this way:

Functional kinds such as *mouse-trap* or *gene* ... have no essence that is a matter of composition. A certain sort of function, a causal role, is the key to being a mousetrap or a carburetor. What makes a bit of a DNA a gene is its function with respect to mechanisms that can read the information that it encodes and use this information to make a biological product. (Block, 1995, pp. 384–385)

Block is no doubt correct, but the distinction between functional kinds and structural kinds (e.g., *water* or *tiger*) is not the only distinction worth making. Also relevant, and especially relevant here, is the distinction between the *purely functional* and the *functional, but not purely functional* (i.e., entities with known physical characteristics that perform specific functions).

As Block would rightly point out, there are many things that can function as a pump. Nonetheless, in the case of cholera, it is the “toxin-damaged cells”—cells that have specific physical properties and have undergone specific physical changes—that perform this role. And that is important for the description of this process, especially the scientific description. Meanwhile, nothing similar can be said for the dictionary in Block’s word recognition example. Functioning as a dictionary is not just the key for this component; it is all there is. And it is all there ever can be as long as methodological functionalism is employed to generate the description.

2.2. Psychology

That methodological functionalism is used in cognitive psychology is not in doubt. Because it has been so difficult to investigate the system that gives rise to human behavior at any worthwhile level of detail, methodological functionalism is the standard technique used to develop explanations of the mind.¹ In almost all cases, psychological capacities—for example, language comprehension, the emotion process, or the performance of mathematical calculations—are understood in purely functional terms. Such explanations are generated by observing the inputs (that is, stimuli from the environment) that individuals receive and the behaviors that they produce, and then suggesting internal components that can explain how the inputs are turned into behavior. When internal components are proposed to explain a psychological ability, the explanation does not refer to any physical material. The components are just functionally defined placeholders that explain the observable result.

Take, as an example, the emotion process. The outputs of this process have been well studied and delineated into the different emotion types (anger, fear, happiness, etc.). These outputs include facial expressions, certain typical behaviors (i.e., “action tendencies”), and various autonomic responses. But while the outputs are relatively easy to study, the inputs to the emotion process are much less tractable. Not only are other psychological states inputs to the emotion process, but virtually any cue in the environment can, under the right conditions, serve as an input. The psychologists Craig Smith and Leslie Kirby summarize the situation this way:

Simple stimulus-based models have proved untenable, given that different individuals often respond to highly similar situations with very different emotional reactions, and, even more important, the same individual often reacts differently to the same situation across multiple occasions. Thus, rather than being fixed responses to specific patterns of stimulation, emotional reactions appear to be highly context-sensitive and take into account not only the environmental circumstances confronting an individual, but also numerous properties of the individual, including his or her personal needs, goals, desires, abilities, and beliefs. (2000, p. 86)

The task, therefore, is to devise a series of components that can transform these extremely varied inputs into the appropriate kinds of outputs. Consider one part of the account proposed by Smith and Kirby:

A central, distinctive feature of this model is the existence of what we call *appraisal detectors*, which continuously monitor for, and are responsive to appraisal information from multiple sources. ... This information is then combined into an integrated appraisal that initiates processes to generate the various components of the emotional response, including an organized pattern of physiological activity, action tendencies, and the subjective feeling state. (2001, pp. 129–130)

The appraisal information in their account is information such as whether or not the stimulus is desirable for the individual, whether it was created intentionally, and how manageable it is. Those determinations are made earlier in the process, and the appraisal detectors identify the information, synthesize it, and initiate the appropriate emotion response.

What is pertinent here is that the appraisal detectors have these functions. Smith and Kirby are not concerned with finding a physical structure for them, although they do at least mention the brain, saying that “although detailed speculations regarding the neuroanatomy of the appraisal detectors [are] beyond the scope of both this chapter and our expertise, we believe them to be subcortical and to most likely reside in the limbic system” (2000, p. 93). But this is just a gesture, and it is in addition to their explanation. Finding a physical entity that performs the job of an appraisal detector is simply not part of Smith and Kirby’s task.

An example from the study of memory is similar. Fuzzy trace theory was developed to explain how memory contributes to higher-order reasoning—how memory is employed when, for instance, individuals make deductive inferences, probabilistic judgments, or decisions based on the qualitative features of a stimulus (e.g., more versus less). A central feature of the theory is that information about a stimulus is encoded, stored, and accessed in two separate ways: as exact “verbatim” representations and as meaningful but less precise “gist” representations (Brainerd & Reyna, 2002, 2004). Charles Brainerd and Valerie Reyna describe these two types of memories—or *traces*—as follows:

Verbatim traces are integrated representations of a memory target’s surface form, as well as associated item-specific information (e.g., contextual cues that are coincident with a target’s occurrence). Gist traces, on the other hand, are representations of semantic, relational, and other elaborative information about a memory target. ... Thus, verbatim traces may be thought of as representations of rememberers’ “actual” experience, and gist traces may be thought of as representations of rememberers’ “understanding” of their experience. (2004, p. 399)

Defining these two types of traces is a straightforward application of methodological functionalism. Everyday experience tells us that there have to be components inside the head that store information—that is, a memory of some sort. And while there is evidence that information is stored, at least for a period, as verbatim traces, a number of phenomena and experimental data suggest a role for something like gist traces. Two examples, often invoked for fuzzy trace theory, are (a) the ability to make accurate inferences in the absence of accurate memories and (b) false memories, especially false memories that are similar in some way to actual occurrences—for instance, a subject incorrectly recalling the word ‘doctor’ after reading other medical terms or a witness to a crime incorrectly identifying an individual of the same gender and ethnicity as the culprit (Brainerd & Reyna, 2002; Reyna & Brainerd, 1995; Reyna & Kiernan, 1994).

So methodological functionalism is used in cognitive psychology. Note, however, that the technique is used prior to, and is neutral about, the variety of possible commitments pertaining to the resulting explanation. It does not matter whether the psychological explanation is construed as a law, a model, Cummins-style functional analysis, a mechanism, or anything else. Those are types of explanations—or, really, theoretical frameworks that explicate features of the psychological explanation and, generally, set it within a context that includes explanations in other sciences.

Take, for instance, functional analysis:

Functional analysis consists in analyzing a disposition into a number of less problematic dispositions such that programmed manifestation of these analyzing dispositions amounts to a manifestation of the analyzed disposition. By “programmed” here, I simply mean organized in a way that could be specified in a program or flow-chart. (Cummins, 1983, p. 28)

This may be, as Cummins says, “a genuinely distinctive style of explanation” (1975, p. 757), but, when used in cognitive psychology, it does not proceed without the application of methodological functionalism. Consider a favorite example, “the circulatory system’s capacity to transport food, oxygen, wastes, and so on,” contrasted with the emotion process (Cummins, 1975, p. 762). The circulatory system is available for inspection. With a little bit of work, the system can be directly observed, although observing it while it is in operation is trickier. Nonetheless, once it is inspected, the capacity of the system is readily determined. Then that capacity can be decomposed into the simpler capacities of the heart, arteries, capillaries, and veins—all of which are also observable.

For the emotion process, on the other hand, there is no system to observe. There are the stimuli in the environment and the responses produced by the capacity—facial expressions, action tendencies, and various autonomic responses—but that is it. It is in virtue of methodological functionalism that there is any content here at all. Once the capacity is specified and intervening variables are proposed, we can say that the resulting explanation is an instance of functional analysis. Yet that cannot happen until after methodological functionalism has been deployed.

The same point can be made for mechanistic explanations. One issue here, however, is that the canonical definitions of a mechanism stress that it is composed of physical entities, not purely functional components. Bechtel and Richardson put it this way:

A machine is a composite of interrelated parts, each performing its own functions, that are combined in such a way that each contributes to producing a behavior of a system. A mechanistic explanation identifies these parts and their organization, showing how the behavior of the machine is a consequence of the parts and their organization. (2010, p. 17; see also Machamer, Darden, & Craver, 2000, p. 3)

Consequently, this notion of mechanism must be relaxed in order to work in cognitive psychology.

But Wright and Bechtel, for instance, call the TOTE unit, which was conceived in the late 1950s as an explanation of the general process for carrying out purposeful behavior, a “basic cognitive mechanism” (2007, p. 41). They also cite mechanisms for language comprehension and production that were developed in the 1960s using Chomsky’s transformational grammar. But one cannot look inside the head and inspect the TOTE units or the components in a mechanism for language comprehension. The content for the explanation must come from the application of methodological functionalism. In contrast to explanations in psychology, often the physical parts of a system can be observed or, at least in some manner, identified. The common mousetrap is a simple example used by Craver and Bechtel (2005) to illustrate the features of a mechanism. A more complex example, one discussed by Machamer, Darden, and Craver, is the “mechanism by which [positively charged sodium ions] get inside the neuronal membrane”—one of the mechanisms for depolarization (2000, p. 9). In these cases, since the physical entities have been identified, the mechanistic explanation proceeds without methodological functionalism.

In summary, methodological functionalism is a technique used in cognitive psychology, and it is not a competitor to functional analysis or mechanistic explanation. But, in cognitive psychology, these explanations cannot proceed without the application of methodological functionalism. On the other hand, methodological functionalism does not have to be used when explaining, say, the circulatory system, even though both a psychological capacity and the circulatory system can be explained by functional analysis or as mechanisms.

3. Methodological functionalism: three historical cases

I am now going to step away from cognitive psychology and look at how well methodological functionalism has worked in other scientific disciplines. As we will see, when it was used by other sciences—which is less frequent than might have been guessed—it was in the early stages of an investigation when there was no other route to understanding the system of interest. But then, in each of the cases that we will examine, the correct description of the process was achieved once methodological functionalism was replaced with techniques that were able to determine the specific physical entities and their roles in the process.

The first case is Mendel's explanation of inheritance. The second and third cases are less well known. The second is a theory of fermentation from the late 1600s, and the third is a theory from the mid-1800s about how cholera affects its victims.

3.1. Mendel's theory of inheritance

Besides cognitive psychology, the paradigmatic example of methodological functionalism is Gregor Mendel's construction of his theory of inheritance. The story here is familiar. Beginning with true-breeding pea plants, Mendel observed the expression of seven different traits over a number of generations. Each of the traits that he followed took one of two forms—e.g., tall or short stems, round or wrinkled seeds, white or purple flowers. When two pure-breeding plants with different forms of the same trait were crossed, Mendel found that all of the offspring displayed only one form of the trait. This form he termed *dominant*. The alternate form of the trait—the recessive form—reappeared among the offspring of these plants. In this generation (now two generations removed from the original pure-breeding plants), for every three plants with the dominant form, there was one with the recessive form. And half of these plants produced, by self-fertilization, offspring in the same three-to-one ratio, while the other half were true-breeding for either the dominant form or the recessive form of the trait.

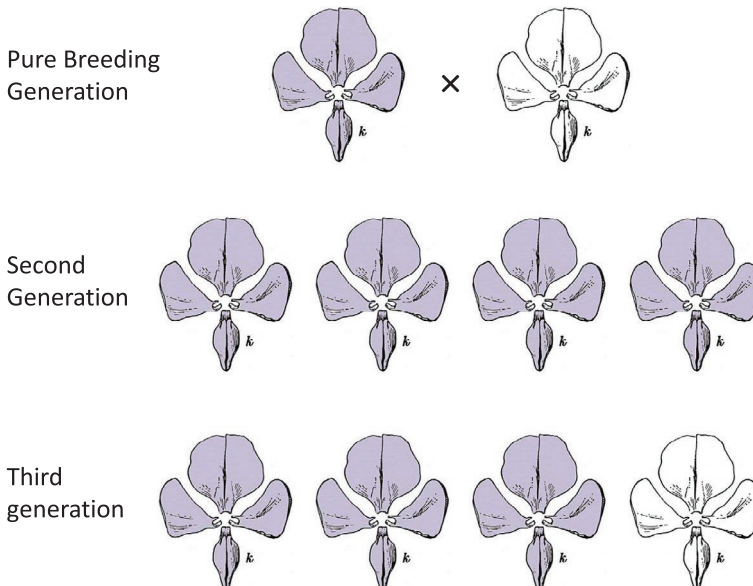


Figure 2. In Mendel's breeding experiments, the plants in the first generation were pure breeding for a trait such as flower color. The plants in the third generation were, he found, split between pure breeding and not. The white flowered plants and about one-third of the purple flowered plants would be pure breeding, and the other two-thirds of the purple flowered plants would produce offspring in the 3:1 ratio.

Based on these observations, Mendel's task was to offer a description of the system that transmits hereditary information from one generation to the next and controls the expression of observable traits. And to do that, he had to devise components that could explain why the particular ratios of traits showed up as they did in each generation.

Mendel started with the idea that a discrete unit carries the hereditary information, what was later termed the *gene*. From there, he suggested that each plant has two copies of the gene for each trait (i.e., two copies of the dominant gene, two copies of the recessive, or one of each, symbolized AA, aa, and Aa, respectively). But each egg and pollen cell that a plant produces has only one copy of each pair. For example, a plant carrying the two gene pairs AaBb would produce an equal number of egg cells containing AB, Ab, aB, and ab. When an egg cell and a pollen cell combine during fertilization, the embryo receives one gene for each trait from each gamete, thereby giving the offspring its own gene pair, which then determines the observed trait.

We now associate the gene with a segment of DNA, but for Mendel, thinking about these carriers of hereditary information was a purely functional endeavor with only the observable traits of the plants for guidance. Hence, it is a clear case where methodological functionalism was used to construct the resulting account.

3.2. Willis's theory of fermentation

In the seventeenth century, the English physician and scientist Thomas Willis developed a wide-ranging theory of fermentation. Willis knew that fermentation occurred during the production of alcohol and bread, but he also thought that it had a role in fevers, digestion, decomposition, and the growth of plants. The basis of his theory—for all these alleged types of fermentation—was the idea that matter is composed of different combinations of spirit, sulfur, salt, water, and earth. Different types of fermentation occur when these particles, in a variety of proportions and combinations, move and interact with each other (Willis, 1684, p. 2).

In the case of beer, after yeast has been added to the wort (the water containing the malt), the particles that Willis thought were active in this process—spirit, salt, and sulfur—were supposed to interact with the particles of earth. Willis describes it this way:

When the liquor of the beer ferments in the vat, the active principles [i.e., spirit, salt, and sulfur] do on every side explicate themselves. [T]hey precipitate in the bottom the more thick, and [the] earthy, being partly driven as it were into flight, and partly sticking to them [i.e., the active particles], they lead them as it were captive to the top, and there make hollow bubbles continually growing up, and bring them as it were to servitude. (1684, p. 18)

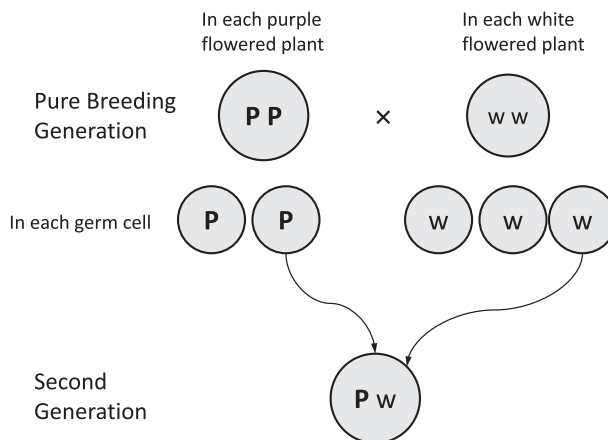


Figure 3. The functionally defined components in the process that Mendel proposed. Each letter represents a dominant or recessive gene.

The process that Willis describes essentially consists of clearing the very murky wort. The components that Willis suggests are the participants in this process—spirit, salt, and sulfur—are given functions that collectively achieve the output: “clear, sweet” beer (1684, p. 18). Of course, in other contexts, salt and sulfur are physical entities, but here there are only the components, referred to as ‘salt’ and ‘sulfur’, that have roles in this alleged process that produces beer.

3.3. *The zymotic theory of cholera*

Of the several theories of cholera that were proposed in the 1800s, the one that went the furthest in trying to describe the process by which cholera acts on the body was developed by the British epidemiologist William Farr, based in large part on the work of the German chemist Justus Liebig. Important to Farr’s account was his belief, common at the time, that the agents that cause disease are found in foul smelling air. As the British social reformer Edwin Chadwick reported to a parliamentary committee in 1846, “all smell is, if it be intense, immediate acute disease; and eventually we may say that, by depressing the system and rendering it susceptible to the action of other causes, all smell is disease” (quoted in Halliday, 2001, p. 1469).

In the case of cholera, Farr suggested that the disease-producing agent was *cholerine*, a “non living organic poison” released when matter decays (Eyler, 1973, p. 82). Decaying bodies that had been infected with cholera were the main source of cholerine, although other decaying organic matter was also sometimes responsible. Once airborne, the cholerine could be inhaled, and it would then pass from the victim’s lungs to the bloodstream. Once in the blood, the cholerine spread and acted as a poison. Farr and Liebig believed that the action of the cholerine in the bloodstream was similar, although not exactly the same as fermentation—the term ‘zymotic’, chosen by Farr, stems from a Greek word meaning “to ferment” (Eyler 1973, 2001; Pelling, 1978). The infection of the blood would then lead to the observable effects of cholera: rapid and intense diarrhea, leathery skin, sunken eyes, and, in almost all cases in the 1800s, death within a day or two.

Interesting from the point of view of an analysis of methodological functionalism, Farr had the input to this process wrong. The agent that causes cholera is waterborne, not airborne, and it is not derived from decaying matter. But he did, of course, get the output correct: the very distinctive type of death described above. And then the intervening process that he described was generated by methodological functionalism; the action of the cholerine in the bloodstream is defined only in terms of what it does.²

3.4. *Lessons from these three historical cases*

In all three of these historical cases, the outputs that each system produce were significant, and it was obvious that unobservable processes were occurring. But the entities and activities that produce the observable phenomena were unknown, and no attempts were made at the time to identify them. Rather, components that act in specific ways—genes (or, to use Mendel’s term, “elements”), cholerine, sulfur, salt, and spirit—were suggested as the participants in the unobservable processes.

It is probably already clear that these descriptions, especially Farr’s and Willis’s, required substantial corrections. But in addition to that observation, a few more relevant points can be made by looking at the historical cases. First, from our present day perspective, it is obvious that Willis’s and Farr’s descriptions needed to be replaced with descriptions that track the physical processes that occur during fermentation and cholera. Mendel’s work has fared better, but the processes he described also had to be recast in terms of biochemical activities. Refusing to do this—that is, clinging to methodological functionalism in the study of genetics—would have been a serious mistake for science and medicine.

These historical cases also highlight two problems. First, looking at only the inputs and the outputs of a system can be misleading and create a bias towards incorrect explanations. Second, even in the best of cases, without knowledge of the processes occurring within the system, it may not be possible to conceive of the correct details. Some participants in the actual physical process are going to be beyond the imagination of any investigator. These are not the only problems raised by the historical

cases, but they are worth examining because they shed light on some of the motivations for giving up methodological functionalism.

As an illustration of the first of these problems, consider Farr's vantage point on cholera. Decaying matter and intense foul smells seem harmful, and since in overcrowded cities they co-occurred with outbreaks of cholera, it was reasonable to assume that they had a role in the spread of the disease. Moreover, watching someone die from cholera might naturally lead to the belief that the person is being poisoned by a fermentation-like process occurring inside the body. And since fermentation was a well-known, if not well-understood, chemical process, using it as a model of changes occurring in the bloodstream was also reasonable. Had the analogy with fermentation turned out to be closer to the truth, Farr's model would have been considered an intellectual achievement.

Nevertheless, Farr was mistaken. It was not until the bacterium *Vibrio cholerae* was identified in 1884 that most scientists accepted that cholera was not caused by something that traveled in the air.³ Moreover, *Vibrio cholerae*, which produces the cholera toxin, had to be discovered in the intestines of victims before it could be determined that the action of this agent occurred there, not in the bloodstream (Howard-Jones, 1984).

A similar moral can be drawn from Willis's description of fermentation. Although it is a historical curiosity at this point, Willis's account is a somewhat plausible story about how murky wort might be transformed into a clear liquid. (In approach and style, it is surprisingly close to Block's word recognition system. Both Block and Willis focus on the macro-level task, the problem as we perceive it. And both propose solutions for that task.⁴) But focusing on clearing the liquid misses what is significant about fermentation. The correct description could not proceed until glucose, fructose, and the enzymes in yeast were discovered in the late nineteenth century (Bechtel, 2005a, 2005b).

Mendel is less guilty of this first problem. He was correct that the outputs he observed—the observable traits that were passed down from the previous generation—suggest that hereditary information is carried by discrete units that occur in pairs. And he knew that the relevant inputs came from the parents. But this just brings us to the second lesson that can be learned from these historical cases. Without knowledge of the physical processes occurring in the system, it is difficult or even impossible to conceive of the correct details.

Regarding these details, anything approaching the full story is too long to relate here.⁵ Nonetheless, while Mendel's notion of the gene as a discrete unit has survived, in almost all cases, individual genes do not control the expression of individual traits. That genes encode the information needed to produce proteins is closer to the truth.⁶ And, typically, more than one gene controls the expression of a trait. This correction is not just a simple switch from *trait* to *protein*, however. Gene expression simply cannot be explained with Mendel's genes—or with the notion of the gene adopted by the early twentieth-century "Mendelian" genetics. At the biochemical level, there are multiple steps between the initiation of transcription and the synthesis of a protein, and the control of these steps by proteins and RNA in the cell determines which proteins are created and when. If DNA, RNA, and the proteins that participate in the process are not invoked, then it is not possible to explain how the hereditary information encoded in the genome is expressed. So, as successful as Mendel was at employing methodological functionalism, the process that he sought to describe in purely functional terms must be described in terms of biochemical entities and activities.

4. The discovery of HIV

The historical cases demonstrate that when methodological functionalism is used for a period, it gets replaced as direct investigations of the processes become possible. A different perspective on the same issue comes from looking at a case where methodological functionalism could have been used but was not. The discovery of HIV is one such case. The agent that causes AIDS and how that agent acts on the individual could have been described with (purely) functionally defined components that produce the visible effects of this syndrome. Or, if that is impractical to imagine, a process conceived

in purely functional terms could have been developed, after which the realizers of this functionally defined process could have been sought. This, however, is not the way in which the case unfolded.

The first scientific reports of what was later recognized as AIDS were by the Centers for Disease Control (now the Centers for Disease Control and Prevention) in June and July of 1981 (CDC, 1981). These were reports of two rare diseases—a type of pneumonia, *Pneumocystis carinii* pneumonia⁷, and a skin cancer, Kaposi's sarcoma—that had occurred, separately and together, in 30 previously healthy, homosexual men in New York City, Los Angeles, and San Francisco (Connor & Kingman, 1988). By August, the CDC was prepared to suggest that “the apparent clustering of both *Pneumocystis carinii* pneumonia and KS among homosexual men suggests a common underlying factor” (CDC, August 1981, p. 410). And rare though it is, *Pneumocystis* pneumonia typically occurs in patients who have received treatments that depleted their white blood cells. Thus, the ability to suppress the immune system seemed to be the critical feature of this “common underlying factor.”

So, the immediate understanding of the system that produces AIDS was in the manner of methodological functionalism: (i) active homosexual men were on the input side of the system, (ii) the outputs were two, normally rare, diseases, and (iii) intervening was a single agent that appears to suppress the immune system. Notice that this agent is defined in purely functional terms; the only feature attributed to it is its ability to suppress the immune system, allowing *Pneumocystis* pneumonia or Kaposi's sarcoma to develop.

One year later, the CDC had two hypotheses about how the agent is introduced into the patient's body:

One hypothesis consistent with the observations reported here is that infectious agents are being sexually transmitted among homosexually active males. Infectious agents not yet identified may cause the acquired cellular immunodeficiency that appears to underlie KS and/or PCP among homosexual males.

Another hypothesis to be considered is that sexual contact with patients with KS or PCP does not lead directly to acquired cellular immunodeficiency, but simply indicates a certain style of life. The number of homosexually

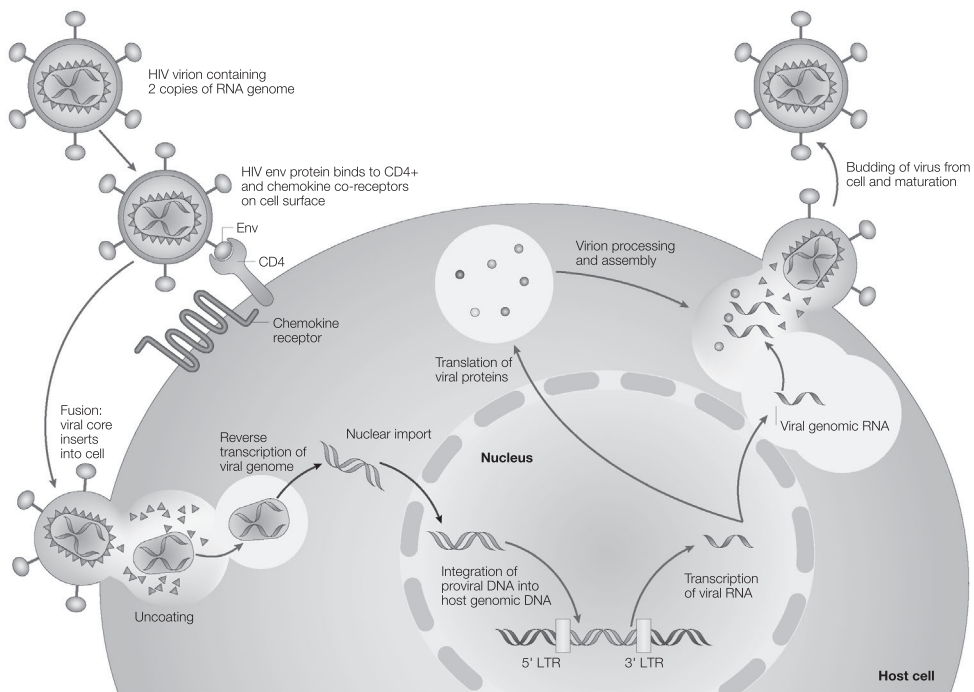


Figure 4. The process by which the HIV retrovirus enters and reproduces in a CD4+ T-cell. Figure from Rambaut, A., Posada, D., Crandall, K. A., and Holmes, E. C. (2004). Used with permission.

active males who share this lifestyle may be much smaller than the number of homosexual males in the general population. ... For example, Marmor et al. recently reported that exposure to amyl nitrite was associated with an increased risk of KS in New York City. Exposure to inhalant sexual stimulants, central-nervous-system stimulants, and a variety of other “street” drugs was common among males belonging to the cluster of cases of KS and PCP in Los Angeles and Orange counties. (CDC, June 1982, pp. 306–307)

But finding, later that year, that intravenous drug users, hemophiliacs, and Haitians were also contracting AIDS complicated these hypotheses (CDC, December 1982). The occurrence of additional “opportunistic infections”—that is, infections that thrive when the immune system is suppressed—in previously healthy patients, however, supported the notion that the infectious agent was acting on the immune system (CDC, September 1982).

By the end of 1982, the CDC believed that AIDS was caused by “an infectious agent transmitted sexually or through exposure to blood or blood products” (CDC, December 1982, p. 653). A week after that statement, they reported four cases of infants who had AIDS-like symptoms, but had not received blood transfusions. One infant’s mother had died, apparently of AIDS, one year after the child’s birth. The mother of the second infant was a prostitute and intravenous drug user in San Francisco, and the other two infants were Haitian.

Having fairly thorough information about both the input side and the output of this illness, investigators could have employed methodological functionalism to fill in details about this infectious agent and how it invites these opportunistic infections. But, even by this point, and before anything else was known about the infectious agent, it had been found that AIDS patients had depleted levels of T-cells, one of the several types of white blood cells (CDC, November 1982). Hence, the purely functional route was never really pursued. And investigations were about to begin in earnest at the cellular and molecular level.

On January 3, 1983, a team in France lead by Luc Montagnier obtained tissue from a lymph node biopsy of a young homosexual male with the early symptoms of AIDS. T-cells from the lymph nodes were cultured, and two weeks later Montagnier found evidence indicating the presence of a retrovirus—a virus whose genome is a single strand of RNA rather than DNA. Further tests demonstrated that this retrovirus was different from HTLV, the retrovirus that causes adult T-cell leukemia, and which was, at the time, the only known retrovirus to infect humans. In May 1983, Montagnier’s group reported their discovery of this retrovirus, later to be named the human immunodeficiency virus (HIV). Within another year, both Montagnier and a team lead by Robert Gallo in the United States established that this retrovirus causes AIDS.⁸ And at the end of 1984, it was reported that one of the first steps of the infection is the retrovirus binding to the CD4 protein of CD4⁺ T-cells (Montagnier, 2002).

It has since been found that, on T-cells, the virus binds to the CD4 protein and either the CXCR4 or CCR5 chemokine receptor, allowing a fusion of the viral and cellular membranes. Then, having gained entry to the cell,

the viral RNA is reverse-transcribed into DNA and randomly integrated into the host cell genome. Activation of the host cell enhances the production of viral proteins, which assemble upon budding out of the cell, utilizing the plasma membrane as the viral envelope. (Alimonti, Ball, & Fowke, 2003, p. 1652)

Thus, the virus spreads (see figure 4). What kills the T-cells is less clear. There are probably several different routes by which this occurs.

Side effects of the virus replicating may be a factor—for instance, damage to the cell membrane from the budding of the virus probably compromises some cells. But the primary way that HIV kills T-cells seems to be by inducing apoptosis, the programmed cell death that happens normally to cull damaged or unneeded cells. In infected T-cells, the integration of the HIV’s DNA into the host cell’s genome activates DNA-dependent protein kinase (DNA-PK). Normally, this protein has a role in repairing broken strands of DNA, but, when HIV’s DNA integrates with the host cell’s, DNA-PK along with the protein p53 triggers apoptosis, killing the T-cell. Beyond the host cells, HIV can also cause apoptosis in uninfected “bystander” T-cells. Several different proteins—gp120, Tat, and Nef—that are encoded by the HIV genome and produced in the infected cells trigger the apoptosis once they are released and migrate to nearby healthy T-cells (Alimonti, Ball, & Fowke, 2003; Cooper et al., 2013; Cummins &

Badley, 2010). It is by the depletion of these T-cells that HIV becomes AIDS. To put it precisely, AIDS is present when the number of CD4⁺ T-cells drops below 200 cells per cubic millimeter of blood, or simply when an opportunistic infection occurs in an individual who has HIV.

In the end, this is a clear case in which methodological functionalism was *not* used to describe the system. Rather the investigation proceeded by seeking out the physical entities that participate in this process—and, of course, determining the role that each has. Given what is already known about this process and the desire to have a full causal (and physical and mechanistic) account of how AIDS develops in an infected individual, it is a little odd to speculate about what a description of the process would look like if methodological functionalism had been employed.

But let's speculate for a moment. Recall that, at the point right before Montagnier began his investigation, it was known that the infectious agent was introduced to the body by sexual contact, by blood transfusion, or from mother to infant during pregnancy or shortly after birth. It was also known that, in individuals with AIDS, the immune system is suppressed, which allows certain infections to thrive. If generating an explanation of how AIDS develops proceeded using methodological functionalism, and if little or nothing was known about the immune system, then a reasonable explanation would be something like the one Farr developed for cholera: the infectious agent poisons the blood. To an extent, that is encouragingly close—although notice that it is the explanation that comes from the direct investigation, not the one generated by methodological functionalism, that determines what counts as close or as correct. But it is still obviously lacking as a complete explanation, and it is useless if one wants to create drugs and treatments that effectively prevent or manage HIV infections. And the latter point is not merely academic. Between 1996 and 2012, 6.6 million deaths were prevented by antiretroviral therapies, a combination of drugs that target different steps in the reproduction of the virus in T-cells (UNAIDS, 2013).

5. Conclusion

Nothing that has been said here guarantees that employing methodological functionalism is always without merit. But there has been a recurring theme. When the physical entities that participate in a process can be studied, the preferred explanation is the one that includes those physical entities and their activities. The cases involving Mendel, Willis, and Farr demonstrate that methodological functionalism is prone to errors. Looking at only the inputs and the outputs of a system can be misleading and create a bias towards incorrect explanations. Even when an investigation is on the right track, without probing the actual process, it is difficult to conceive of all of the correct details. The HIV case, meanwhile, points to the importance of (and our apparent interest in) the complete, physical explanation. One conclusion drawn from looking at these cases, then, is that methodological functionalism has significant limitations.

But even with its flaws, it may be possible to justify the use of methodological functionalism in cognitive psychology. The more benign option is to claim that cognitive psychology uses methodological functionalism because the alternative is not currently feasible, though in the future methodological functionalism will likely be replaced. Since the primary alternative to methodological functionalism is investigating the physical entities and activities that participate in a process, the consequence of eventually giving up methodological functionalism is that cognitive psychology gets reduced to neurobiology.

A different route is to claim that methodological functionalism is, and always will be, the appropriate technique for generating descriptions of what goes on inside the head. The justification for this stance is not obvious though. The cases that have been examined here undermine the notion that there is inductive support from across the sciences for methodological functionalism. And to say that cognitive psychology should use methodological functionalism because doing so gives us the types of explanations that we want looks question begging. (In other words, the argument is this: our finished explanations need to include components such as appraisal detectors, gist traces, beliefs, desires, and the like; methodological functionalism gives us these things; therefore, we need to use methodological functionalism.)

A slightly different and non-question begging rationale is along the lines of Jerry Fodor's claim that cognitive psychology exists as it is, "not because of the nature of our epistemic relation to the world, but because of the way the world is put together" (1974, p. 113). This gets murky, however. If there is one thing that we have seen, it is that methodological functionalism does not track the "way the world is put together" very well. Direct investigations into various phenomena tell us how the world is put together. That said, one issue that I have not directly addressed is "How correct (or explanatory, or predictive) is cognitive psychology?" And although I am not going to take up that question now, the picture that has developed here suggests that there are errors in cognitive psychology that cannot be overcome unless methodological functionalism is given up.

The purported multiple realizability of psychological states (qua their purely functional descriptions) is another possible justification for maintaining methodological functionalism but, perhaps, not a very robust one.⁹ The justification would go as follows: psychological states, as they are described using methodological functionalism, are multiply realizable; hence we are prevented from giving up methodological functionalism because these states are multiply realizable. But that aside, again, the cases that have been examined here are not supportive. When methodological functionalism is used, any of the intervening variables that are invoked could be multiply realized. Farr's cholera, Willis' spirit, salt, and sulfur, and Mendel's gene are all, in principle, multiply realizable. But there does not seem to have been any interest in blocking a direct investigation—that is, a reduction—on the grounds that the purely functional component might be multiply realized. Presumably, the thought never even occurred to anyone. Moreover, in those cases, the proposed components are not realized at all simply because the explanations in which they figure are false. Thus, multiple realizability is beside the point. Of course, we can hope that cognitive psychology fairs better, but, as just mentioned, the implication is that cognitive psychology contains errors that are not correctable as long as methodological functionalism is used. Thus, prior to making a claim about multiple realizability, it should be demonstrated that the descriptions generated by cognitive psychology do not require the types of corrections that, in the examples from Farr, Willis, and Mendel, were made by direct investigations.¹⁰

Since giving up methodological functionalism amounts to reducing cognitive psychology to neurobiology, there is a lot at stake here. But not giving up methodological functionalism when doing so is possible is, perhaps, even less appealing. Recall Paul Churchland's (1981) argument that if alchemy had been recast as a functional theory, it could have avoided being replaced by the chemistry of Lavoisier. This move, which a "cunning and determined defender of the alchemical vision" could have undertaken, Churchland calls the *functionalist stratagem*: the participants in the theory become purely functional states, the level of description of the theory is put at a suitably abstract level, and it is claimed that the potential realizers of the functional states are multiple physical substances (1981, p. 80). Now the defender of alchemy can claim "it is the particular orchestration of the syndromes of occurrent and causal properties which makes a piece of matter gold, not the idiosyncratic details of its corpuscularian substrate," and correction by a corpuscularian chemistry is no longer possible (Churchland, 1981, p. 80). The purely functional theory is safe because the physical details are—so it is claimed—irrelevant to the explanation. Phlogiston theory, medieval medicine's four humors, and the vital essence of early biology could also, Churchland suggests, have avoided being eliminated by using the functionalist stratagem.

Churchland's point is that a functional theory—of the mind or anything else—has resources that allow it to resist the idea that the system being investigated may be more satisfactorily explained in terms of the relevant physical entities and their activities. Claiming that mental states are functional, abstract, multiply realizable, and, therefore, irreducible may—for a time anyway—allow a functional theory of mind to avoid being corrected (or replaced) by neurobiology. But the functional stratagem, whether employed on behalf of alchemy or folk psychology, is, Churchland says, "a smokescreen for the preservation of error and confusion" (1981, p. 81).¹¹ My point is simply that theories developed using methodological functionalism, which, of course, includes cognitive psychology, are in a precarious position: they are prone to errors that cannot be corrected without giving up methodological functionalism. But not facing up to that problem seems to risk, whether it is intentional or not, the adoption of the functionalist stratagem.

In any event, having examined how methodological functionalism is used to generate explanations and how it fared in historical cases, we can certainly conclude that cognitive psychology is on a trajectory in which it uses methodological functionalism for a period of time, after which it will turn to other investigative techniques as they become available. It may even be that the shift away from methodological functionalism has already begun. Although he does not discuss methodological functionalism as such, John Bickle argues that the shift has been underway for some time. Near the end of the chapter “Reduction-in-practice in current mainstream neuroscience” in his book *Philosophy and Neuroscience: A Ruthlessly Reductive Account*, Bickle reports:

Psychoneural reductionism is alive and thriving in current cellular and molecular neuroscience, as revealed in the attitudes of its practitioners and their choices of experimental manipulations and investigations. And the specific nature of the reductionism encapsulated within these practices and results is usefully illustrated by the “structuring” of psychology’s purely functional posits into specific sequences and combinations of cellular and molecular entities, processes, and causal interactions. That is reductionism-in-practice in current mainstream neuroscience and its metascience. (2003, p. 102)

But if cognitive psychology is not on such a trajectory and a justification for maintaining methodological functionalism is available, then the conclusion is that cognitive psychology is unique among the special sciences insofar as it is able to successfully employ methodological functionalism.

Notes

1. I date the beginning of psychology’s use of methodological functionalism in the 1950s—the point at which behaviorism was displaced by cognitive psychology and cognitive science. That may be overlooking the use of methodological functionalism prior to the 1950s, but starting with the cognitive revolution is sufficient for the purposes here because, ultimately, my interest is in cognitive psychology.

Behaviorism, at least in its strictest versions, sought to avoid postulating intervening variables, and so did not employ methodological functionalism. But, strict behaviorism aside, something similar to methodological functionalism may have been used before the 1950s, although introspection as an “investigative technique” should be kept separate from methodological functionalism.

2. In its state outside of the body, cholera might not be considered purely functional—it is presumed to come from decaying organic matter—even though no attempt seems to have been made to perform a physical study of it. Inside the body, however, it has all the characteristics of a purely functional component.

A similar concern can be raised about Willis’s use of spirit, salt, and sulfur in the fermentation of beer. If Willis thought that those particles were interacting with the particles of earth (which is, presumably, what he thought made the wort so murky), then it is not much of a step to attribute size and location to the spirit, salt, and sulfur. But, nonetheless, the account is one that was, quite clearly, generated using methodological functionalism.

Any seeming ambiguity in these cases arises because the explanations are offered within the boundaries of materialism (Willis’s “spirit” aside), and so terminology consistent with materialism is used. We see that these explanations are the product of methodological functionalism, however, when we look at how they were generated: inputs and outputs were known, and an intervening process was proposed but never directly investigated.

3. The British physician John Snow, a contemporary of Farr’s, claimed that cholera was caused by an agent found in the water, not the air. Unfortunately, this view was not accepted while Snow was alive. Interestingly though, Farr became sympathetic to it at the end of his life (Halliday, 2001).

The German scientist Robert Koch discovered *Vibrio cholerae* while studying patients with cholera in 1883 and 1884, and his discovery became immediately well known. The bacterium had, however, already been found (and reported) by the Italian scientist Filippo Pacini in 1854 (Howard-Jones, 1984).

4. This manner of describing a psychological capacity is what William Lycan (1987) calls *homuncular functionalism* (see also Dennett, 1975).

5. It should be stressed that the previous section discussed Gregor Mendel’s account. The classical (or “Mendelian”) genetics of the first decades of the twentieth century did, to an extent, move beyond purely functional descriptions. Kenneth Waters explains:

[Classical genetics] has a cytological interpretation. Gene chains are identified as chromosomes. Meiosis, the process in which chromosomes are distributed to gametes, offers an explanation of segregation and assortment. During the first division of this process, homologous chromosomes pair and then separate as two daughter cells are produced. The lack of complete linkage of genes located on the same chromosome is explained in terms of the crossing over (the exchange) of chromosomal segments. (1990, p. 127)

The importance of this interpretation was probably limited, however, during the development of that theory. In 1915, Morgan, Sturtevant, Muller, and Bridges took this view:

But it should not pass unnoticed that even if the chromosome theory be denied, there is no result dealt with in the following pages that may not be treated independently of chromosomes; for, we have made no assumption concerning heredity that cannot be made abstractly without the chromosomes as bearers of the postulated hereditary factors. (1915, p. viii)

6. This too is open to revision though. See, for instance, Pearson (2006).
7. This form of pneumonia is now called pneumocystis jiroveci pneumonia.
8. The causal connection was found, not by determining the mechanism at the molecular level, but by testing for antibodies to Mantagnier's retrovirus in two groups of subjects: (i) individuals with AIDS or individuals who were believed to have AIDS and (ii) individuals who were not at risk and did not have any symptoms of AIDS.
9. The case has also been made for a while now that it cannot be taken for granted that mental states are multiply realized. Multiple realization may, it seems, be a much less robust phenomenon than was once assumed. See, for instance, Bechtel and Mundale (1999), Bickle (2003), Polger (2004), and Shapiro (2000).
10. This presents another difficulty for cognitive psychology. In the cases examined, the ultimate arbiter of what is correct and incorrect was the direct investigations. Thus, if methodological functionalism is not given up, there is, potentially, a barrier to even establishing that the explanations in cognitive psychology are correct.
11. The functionalism that concerns Churchland reaches beyond the use of methodological functionalism. Adopting the functionalist stratagem is committing to a certain theoretical framework—functional analysis, for instance, or the generic functionalism that Churchland outlines in section 3 of "Eliminative Materialism and the Propositional Attitudes." (This was the issue discussed at the end of section two.)

Also, the historical cases that I have discussed were, more or less, legitimate investigations that used methodological functionalism. Churchland uses alchemy, phlogiston theory, medieval medicine, and pre-modern biology because they are theories that are, in hindsight, clearly false. That said, alchemy, phlogiston theory, medieval medicine, and pre-modern biology may have employed, and probably did employ, methodological functionalism. But adopting the functionalist stratagem would be a further step, one that requires committing to a full functionalist framework (say, Cummins-style functional analysis) for the purpose of avoiding correction or replacement by new scientific developments.

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References

- Alimonti, J. B., Ball, T. B., & Fowke, K. R. (2003). Mechanisms of CD4⁺ T lymphocyte cell death in human immunodeficiency virus infection and AIDS. *Journal of General Virology*, 84, 1649–1661.
- Bechtel, W. (2005a). Mental mechanisms: What are the operations? *Proceedings of the 27th Annual Meeting of the Cognitive Science Society* (pp. 208–213). Mahwah, NJ: Erlbaum.
- Bechtel, W. (2005b). The challenge of characterizing operations in the mechanisms underlying behavior. *Journal of the Experimental Analysis of Behavior*, 84, 313–325.
- Bechtel, W., & Mundale, J. (1999). Multiple realizability revisited: Linking cognitive and neural states. *Philosophy of Science*, 66, 175–207.
- Bechtel, W., & Richardson, R. C. (2010). *Discovering complexity: Decomposition and localization as strategies in scientific research*. Cambridge, MA: MIT Press.
- Bickle, J. (2003). *Philosophy and neuroscience: A ruthlessly reductive account*. Boston, MA: Kluwer.
- Block, N. (1995). The mind as the software of the brain. In D. N. Osherson & L. R. Gleitman (Eds.), *An invitation to cognitive science: Thinking* (pp. 377–425). Cambridge, MA: MIT Press.
- Brainerd, C. J., & Reyna, V. F. (2002). Fuzzy-trace theory and false memory. *Current Directions in Psychological Science*, 11, 164–169.
- Brainerd, C. J., & Reyna, V. F. (2004). Fuzzy-trace theory and memory development. *Developmental Review*, 24, 396–439.
- Centers for Disease Control (CDC) (1981, August). Follow-up on Kaposi's sarcoma and *pneumocystis* pneumonia. *Morbidity and Mortality Weekly Reports*, 30, 409–410.
- Centers for Disease Control (CDC) (1982, June). A cluster of Kaposi's sarcoma and *pneumocystis carinii* pneumonia among homosexual male residents of Los Angeles and Orange counties, California. *Morbidity and Mortality Weekly Reports*, 31, 305–307.
- Centers for Disease Control (CDC). (1982, September). Current trends update on acquired immune deficiency syndrome (AIDS)—United States. *Morbidity and Mortality Weekly Reports*, 31, 507–508, 513–514.

- Centers for Disease Control (CDC) (1982, November). Cryptosporidiosis: Assessment of chemotherapy of males with acquired immune deficiency syndrome (AIDS). *Morbidity and Mortality Weekly Reports*, 31, 589–592.
- Centers for Disease Control (CDC). (1982, December). Possible transfusion-associated acquired immune deficiency syndrome (AIDS)—California. *Morbidity and Mortality Weekly Reports*, 31, 652–654.
- Churchland, P. M. (1981). Eliminative materialism and the propositional attitudes. *Journal of Philosophy*, 78, 67–90.
- Connor, S., & Kingman, S. (1988). *The search for the virus*. New York, NY: Penguin.
- Cooper, A., Garcia, M., Petrovas, C., Yamamoto, T., Koup, R. A., & Nabel, G. J. (2013). HIV-1 causes CD4 cell death through DNA-dependent protein kinase during viral integration. *Nature*, 498, 376–379.
- Craver, C., & Bechtel, W. (2005). Mechanism. In S. Sarkar & J. Pfeifer (Eds.), *The philosophy of science: An encyclopedia* (pp. 469–478). New York, NY: Routledge.
- Cummins, N. W., & Badley, A. D. (2010). Mechanisms of HIV-associated lymphocyte apoptosis: 2010. *Cell Death & Disease*, 1, e99.
- Cummins, R. (1975). Functional analysis. *Journal of Philosophy*, 72, 741–765.
- Cummins, R. (1983). *The nature of psychological explanation*. Cambridge, MA: MIT Press.
- Dennett, D. C. (1975). Why the law of effect will not go away. *Journal for the Theory of Social Behaviour*, 5, 169–188.
- Eyler, J. M. (1973). William Farr on the cholera: The sanitarian's disease theory and the statistician's method. *Journal of the History of Medicine and Allied Sciences*, 28, 79–100.
- Eyler, J. M. (2001). The changing assessments of John Snow's and William Farr's cholera studies. *Sozial- und Präventivmedizin/Social and Preventive Medicine*, 46, 225–232.
- Fodor, J. A. (1974). Special Sciences (Or: The Disunity of Science as a Working Hypothesis). *Synthese*, 28, 97–115.
- Halliday, S. (2001). Death and miasma in Victorian London: An obstinate belief. *British Medical Journal*, 323, 1469–1471.
- Howard-Jones, N. (1984). Robert Koch and the cholera vibrio: A centenary. *British Medical Journal (Clinical Research Edition)*, 288, 379–381.
- Lycan, W. G. (1987). *Consciousness*. Cambridge, MA: MIT Press.
- Machamer, P., Darden, L., & Craver, C. (2000). Thinking about mechanisms. *Philosophy of Science*, 67, 1–25.
- Mendel, G. (1996). Experiments in plant hybridization (W. Bateson, Trans.). *Electronic Scholarly Publishing Project*. (Original work published 1865). Retrieved from www.esp.org/foundations/genetics/classical/gm-65.pdf
- Miller, G. A., Galanter, E., & Pribram, K. H. (1960). *Plans and the structure of behavior*. New York, NY: Holt.
- Montagnier, L. (2002). A history of HIV discovery. *Science*, 298, 1727–1728.
- Morgan, T. H., Sturtevant, A. H., Muller, H. J., & Bridges, C. B. (1915). *The mechanism of Mendelian heredity*. New York, NY: Holt.
- Pearson, H. (2006). Genetics: What is a gene? *Nature*, 441, 398–401.
- Pelling, M. (1978). *Cholera, fever and English medicine, 1825–1865*. Oxford: Oxford University Press.
- Polger, T. W. (2004). *Natural minds*. Cambridge, MA: MIT Press.
- Rambaut, A., Posada, D., Crandall, K. A., & Holmes, E. C. (2004). The causes and consequences of HIV evolution. *Nature Reviews Genetics*, 5, 52–61.
- Reyna, V. F., & Brainerd, C. J. (1995). Fuzzy-trace theory: An interim synthesis. *Learning and Individual Differences*, 7, 1–75.
- Reyna, V. F., & Kiernan, B. (1994). Development of gist versus verbatim memory in sentence recognition: Effects of lexical familiarity, semantic content, encoding instructions, and retention interval. *Developmental Psychology*, 30, 178–191.
- Shapiro, L. A. (2000). Multiple realizations. *Journal of Philosophy*, 97, 635–654.
- Shetty, N., Tang, J. W., & Andrews, J. (2009). *Infectious disease: Pathogenesis, prevention and case studies*. Chichester, England: Wiley-Blackwell.
- Smith, C. A., & Kirby, L. D. (2000). Consequences require antecedents: Toward a process model of emotion elicitation. In J. P. Forgas (Ed.), *Feeling and thinking: The role of affect in social cognition* (pp. 83–106). Cambridge UK: Cambridge University Press.
- Smith, C. A., & Kirby, L. D. (2001). Toward delivering on the promise of appraisal theory. In K. R. Scherer, A. Schorr, & T. Johnstone (Eds.), *Appraisal processes in emotion: Theory, methods, research* (pp. 121–138). New York, NY: Oxford University Press.
- United Nations Joint Programme on HIV/AIDS (UNAIDS) (2013). *Global report: UNAIDS report on the global AIDS epidemic 2013*. Geneva, Switzerland: UNAIDS.
- Waters, C. K. (1990). Why the anti-reductionist consensus won't survive: The case of classical mendelian genetics. *PSA: Proceedings of the Biennial Meeting of the Philosophy of Science Association 1990*, 1, 125–139.
- Willis, T. (1684). *Dr. Willis's practice of physick* (S. Pordage, Trans.). London: Printed for T. Dring, C. Harper, and J. Leigh.
- Wright, C., & Bechtel, W. (2007). Mechanisms and psychological explanation. In P. Thagard (Ed.), *Philosophy of psychology and cognitive science: A volume of the handbook of the philosophy of science series* (pp. 31–79). Boston, MA: Elsevier.