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The Response Set Theory of Hypnosis: Expectancy and Physiology

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A recent exposition of the response set theory of hypnosis (Kirsch, 2000) contained incorrect and misleading figures. The correct figures illustrated a complementary relation between mental and physiological phenomena. The figures as published erroneously suggested that the author espoused epiphenomenalism. As shown in this corrected version, Kirsch proposes that mind states and body states be considered as two ways of viewing a single psychophysiological phenomenon.

At the request of the former editor of the *American Journal of Clinical Hypnosis*, I wrote a summary of the response set theory of hypnosis, which was published in the January/April, 2000, issue of the journal (Kirsch, 2000). Due to a production error, the figures accompanying the article were not reproduced correctly. Specifically, horizontal lines denoting causal relations between mental states (e.g., negative expectations as a cause of depression) were deleted. The corrected figures are reproduced here, along with the section of the article in which they were contained.

There are two kinds of physiological effects that have been attributed to response expectancies. First, there are effects that are physiological concomitants of expected changes in specific subjective states. For example, people who report increases in expectancy-generated general or sexual arousal may exhibit corresponding changes in blood pressure, penile tumescence, and vaginal blood volume (Kirsch & Weixel, 1988; Lansky & Wilson, 1981; Palace, 1999). Second, global health effects (e.g., enhanced immune functioning) have been hypothesized. For example, Klopfer (1957) reported an amazing case study in which a placebo seemed to profoundly affect the growth of a malignant tumor. It is likely that these two types of effects involve different mechanisms, but both can be best understood by considering the principle of psychophysiological complementarity (Hyland, 1985; Kirsch & Hyland, 1987).

Psychophysiological complementarity rests on the assumption that there is a physiological substrate to all experiential states. The reverse assumption that all physiological states are represented in consciousness is not made. As a heuristic,

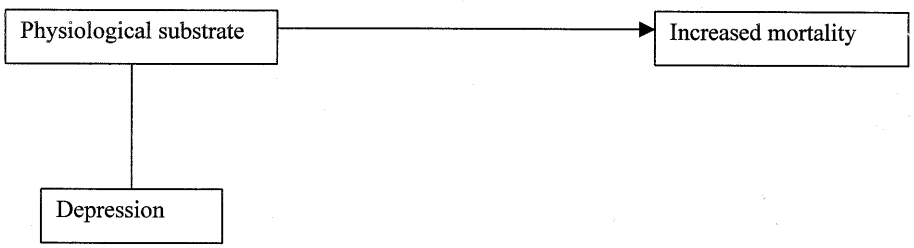
Hyland (1985) proposed that mind states and body states be considered two ways of viewing a single psychological phenomenon, in the same way that light can be viewed as either particles or waves. From this perspective, the relation between a psychological state (e.g., an emotion) and its physiological substrate is that of identity. They are the same state, viewed in two different ways. Although most clearly related to the metaphysical stance of double aspect monism, Kirsch and Hyland (1987) demonstrated that this conclusion can be deduced logically from virtually all monist philosophies of mind. Because it is not tied to a particular metaphysic, Hyland's (1985) position has been characterized as *methodological complementarity*. It is a heuristic framework for psychophysiological theory, rather than a metaphysical argument.

At first glance, rejecting dualism and beginning with the assumption that mind terms and corresponding body terms refer to the same underlying event seems to present a problem, rather than a solution, for psychophysiological theory. If a mental state *is* a physical state, then how can it cause changes in that state? Saying that it does would seem as strange as saying that water was causing a change in H_2O. Yet we have strong evidence of psychological effects on physiological function. How can this be?

The answer lies in recognizing that an accurate causal statement linking a psychological state to a physiological condition is in reality a shorthand summary of a more complex set of relations. These include causal relations between mind states and other mind states, causal relations between physiological states and other physiological states, and identity relations between mind states and physiological states. Figure 1 provides an illustration of a causal network that could be represented by the statement that depression increases mortality. As shown in the figure, it is not the subjective experience of depression that causes the increase in mortality, but rather the physiological state with which the feelings are identified.

The statement "depression increases mortality" is simpler than the corresponding statement (illustrated in Figure 1) that the physiological substrate of depression increases mortality. What is the advantage of the more complex formulation? There are a couple of problems with statements of mental causation of physical events. The

Figure 1: Apparent mental causality as viewed from the perspective of psychophysiological complementarity. The vertical line indicates an identity relation. The horizontal arrow indicates a causal relation.

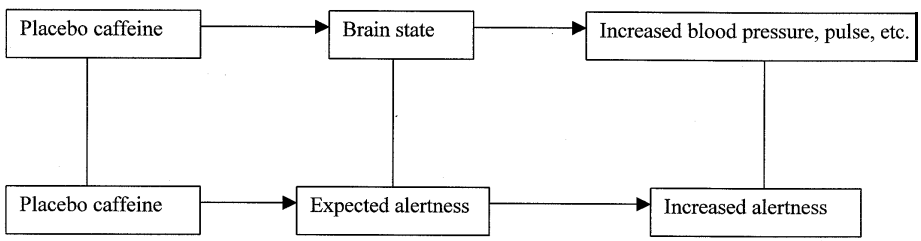


first is that they violate the law of conservation of energy, because the only way to alter the behavior of a physical system is to add energy to it. The second problem is that accepting direct mental causation of physical events leads to an infinite regress. If we accept that depression increases mortality, we are led to ask, "How does depression increase mortality?" The answer to this question will involve either a psychological or physiological mediating variable. We might suppose, for example, that depression adversely affects the function of the immune system and that the increase in mortality is due to the inhibition in immune function. This, however, only leads to the next question, which is, "How does depression inhibit immune function?" Although we can in principle imagine closure to the problem of how one physical event causes another or how one psychological event causes another, it is difficult to conceive of a complete answer to the question of how a mental event causes a physical event without invoking the principle of complementarity. Unless we posit identity relations, answers to that question always involve an unexplained causal connection between a mental state and a physical state.

The principle of psychophysiological complementarity provides a framework for understand both types of response expectancy effects on physiological function: (1) those in which expectancies for specific changes in experience produce produce changes in the physiological substrates of those subjective states and (2) those in which expectancies for positive or negative outcomes produce global health benefits. An example of the first type of effect is illustrated in Figure 2. Here, placebo caffeine produces expectancies for increased alertness, which in turn are associated with reported increases in alertness and with changes in measures of physiological arousal (cf. Kirsch & Weixel, 1988). As subjective states, response expectancies produce changes only in subjective experience. It is the brain state with which the expectancy is identified that alters the physiological concomitants of altered subjective state.

A major task for psychophysiological researchers is to uncover the physiological states with which specific response expectancies are identified. This may seem a daunting task, but an important start has recently been reported. Drevets, Burton Videen, Snyder,

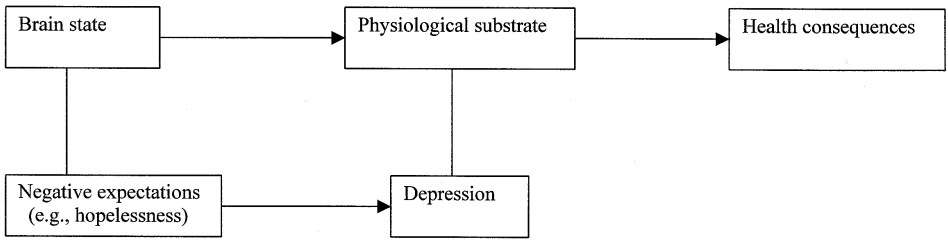
Figure 2: An example of how expectancies produce changes in physiological substrates of psychological states. Vertical lines indicate identity relations. Horizontal arrows indicate causal relations.



Simpson, and Raichle (1995) identified brain state changes associated with expectations of acute pain in specific locations. Specifically, the expectation of a painful electric shock to specific body sites (e.g., particular fingers and toes) produced a significant decrease in blood flow in areas of the somatosensory cortex unrelated to those sites. According to Drevets et al., these data suggest that sensory transmission is suppressed in areas where stimulation is not expected, thereby facilitating the processing of signals in areas where it is anticipated. Although clearly associated with a specific response expectancy, the blood flow changes reported in this study may be a substrate of a consequence of that expectancy, rather than a substrate of the expectancy itself. It may, for example, be a substrate of the shift in attentional focus produced by the anticipated stimulation.

Response expectancy effects on the physiological substrates of psychological states are very specific. For example, the effects of placebos given as tranquilizers are the opposite of those of the same placebos presented as stimulants (Frankenhaeuser, Jarpe, Svan, & Wrangsjö, 1963). This may account for the effects of expectancy on particular symptoms, including the side effects produced by placebos (Pogge, 1963). It may also account for the very specific changes in brain physiology that occur in response to particular hypnotic suggestions. However, it is likely that a more global mechanism is involved in response expectancy effects on physiological functions that are not substrates of particular psychological states. The effects of expectancy on illness, for example, may be mediated by such global psychological states as hopelessness and depression or faith and feelings of well-being. More accurately, it is the physiological substrates of these global psychological states that might produces changes in physical health, as illustrated in Figure 3.

Figure 3: Global psychological states as mediators of response expectancy effects on physical states that are not substrates of psychological states. Vertical lines indicate identity relations. Horizontal arrows indicate causal relations.



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