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Continuing Commentary

Commentary on **John A. Nevin & Randolph C. Grace (2000). Behavioral momentum and the Law of Effect. BBS 23(1):73–130.**

Abstract of the original article: In the metaphor of behavioral momentum, the rate of a free operant in the presence of a discriminative stimulus is analogous to the velocity of a moving body, and resistance to change measures an aspect of behavior that is analogous to its inertial mass. An extension of the metaphor suggests that preference measures an analog to the gravitational mass of that body. The independent functions relating resistance to change and preference to the conditions of reinforcement may be construed as convergent measures of a single construct, analogous to physical mass, that represents the effects of a history of exposure to the signaled conditions of reinforcement and that unifies the traditionally separate notions of the strength of learning and the value of incentives. Research guided by the momentum metaphor encompasses the effects of reinforcement on response rate, resistance to change, and preference, and has implications for clinical interventions, drug addiction, and self-control. In addition, its principles can be seen as a modern, quantitative version of Thorndike's (1911) Law of Effect, providing a new perspective on some of the challenges to his postulation of strengthening by reinforcement.

Aping Newtonian physics but ignoring brute facts will not transform Skinnerian psychology into genuine science or useful technology

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Abstract: The proposal to add the behavioral momentum metaphor to Skinnerian psychology and the use of other borrowed physical explanatory concepts such as velocity and inertial mass has only superficial value. The basic problem is that, in contrast to Newtonian physics, the "laws" do not apply to a significant proportion of the phenomena to be explained, and these evidential discrepancies are ignored, rather than being used to modify the scientific explanations and improve technological applications that are based on those explanations.

At least since the days of Hull (1949), who referred to "axioms" and introduced quantification into his book in an attempt to associate his psychological theories with those in Newton's *Principia,* some psychologists have tried to "harden" their science by drawing analogies with physics. I view Nevin & Grace's (N&G's) target article as an elegant but fundamentally misguided attempt to perform the same service for Skinnerian psychology, through their introduction of the physical concept of momentum as a metaphor, and by their drawing parallels to such other concepts of Newtonian physics as velocity and inertial mass.

A crucial feature of theories in physics is that they are genuinely testable. As a result of the scientific progress that occurs, the technology based on the science also improves over time. One hint that this may not hold for N&G's psychological metaphor-based theorizing is that N&G discuss only confirmatory evidence, and fail to indicate what disconfirmatory evidence, were it to occur, would falsify their theory.

In contrast, and to take just one example from physics and astronomy, when perturbations in the path of Mercury were found that could not be accounted for in terms of the gravitational influence of the Sun and the other planets, this *tension* between Newtonian theory and apparently contrary evidence was first resolved by positing the existence of another planet inside Mercury's orbit. When "Vulcan" turned out to be an astronomical illusion, Mercury's perturbation became part of the evidence that led to the replacement of Newtonian by Einsteinian physics. Again, an early and genuine test of Einstein's theory was the Michaelson-Morley experiment, which, had it shown that the speed of light varied, would have falsified Einstein's theory. In these and other examples of evidence-based testing of physical theories, contrary evidence is not ignored. The laws of physics are expected to apply to the movement of *all* bodies, and science-based applications (e.g., the development of the atomic bomb) manifest systematic improvements that are based on progress in scientific understanding (e.g., control over nuclear fission was based at least in part on the shift from Newtonian to Einsteinian theorizing).

On the other hand, N&G's central psychological law (that contingency analysis of the relations between the "discriminated operant" and the "reinforcer" and between the "stimulus" and the "reinforcer" fully accounts for the behavior of living organisms) does not account for a substantive set of behavioral phenomena which are unmentioned in their target article. I refer to these phenomena as "brute facts" (Maze 1983) to indicate that their existence is virtually certain and cannot be ignored either by science or by any science-based technology.

The first two facts are related to Skinnerian, operant, or instrumental conditioning (which N&G assert is governed by the contingency between the "discriminated operant" and the "reinforcer"). The second pair of facts pertain to Pavlovian, respondent, or classical conditioning, which N&G, following Rescorla's assertion that Pavlovian conditioning can be "*described*" as the "learning of the relations between events" (Rescorla 1988, p. 151, my emphasis), ascribe solely to the contingency between the stimulus and the reinforcer.

The proverbial version of Skinnerian conditioning is where rats press a bar with food as the reinforcer. Although conditioning occurs in most cases, there are always a few rat-experimenter dyads

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where the "discriminated operant" is never acquired or "established by reinforcement." The reasons for such failures to acquire the behavior are forever hidden, in principle, by the Skinnerian dictum that "shaping is an art." Relegating the acquisition or "shaping" (i.e., learning) of behavior to an "art" and ignoring instances of failures has undesirable consequences both for science and for applications of psychology. The Skinnerian definition of psychology is the prediction and control of behavior. But prediction is undermined if we cannot tell in advance whether a certain rat (perhaps combined with a certain shaper) will acquire the behavior, *and at what rate.* Control is also lessened if one cannot provide scientific principles (and not those of "art") to guide the practitioner in how to administer maximally effective training.

Still, some may object that the above fact about bar-press-for-food Skinnerian conditioning concerns only a few animals. That objection does not apply to a second set of brute facts that emerges when one changes the reinforcer or the to-be-learned target behavior (the discriminated operant). An instance of a change in reinforcer is when one replaces food with shock avoidance. In that case, as is common laboratory knowledge, about 50% of the rats fail to ever acquire the discriminated operant.

An instance of changing the target behavior also illustrates problems for applications – that is, the "control" of behavior rather than its prediction. The example I use is relevant to Skinnerian conditioning's most enthusiastically touted application: "biofeedback" or the instrumental conditioning of autonomic functions.

The central concept behind biofeedback is that supplying "information" about the relations between changes in such autonomically controlled functions as heart rate and blood pressure, on the one hand, and feedback or contingent reinforcement, on the other, will result in an increase of "self-control" over these functions. There was considerable excitement and enthusiasm for such instrumental autonomic conditioning following the report of Miller (1969). But when it comes to such difficult and medically relevant behaviors as heart rate deceleration and blood pressure decrease, there is practically no evidence, not only in the clinic (e.g., Furedy & Shulhan 1987) but even in the laboratory (e.g., Furedy 1987), that the contingency between the reinforcer (feedback) and the target behavior (e.g., blood pressure decrease) has *any* specific beneficial effect on control.

A similar set of lacunae exist for current contingency accounts of the phenomenon of Pavlovian conditioning. For N&G, Pavlovian conditioning is the "contingency space" or the "informativeness of the stimulus with respect to the reinforcement" (p. 79). Essentially, this is the original Rescorla (1967; 1969) formulation, which itself is a resurrection of the Tolmanian idea of sign-significance (S-S) learning (for details of the nature of this sort of cognitive, propositional, current account of Pavlovian conditioning, see Furedy & Riley 1987). No doubt the formulation is "intuitively reasonable," but whether it is also "empirically useful" (p. 79) is open to question, if one includes under the rubric of empirical usefulness the ability of the approach to be sensitive to relevant factual evidence regarding actual Pavlovian conditioning phenomena.

To begin with, it follows from any "contingency-space" account of Pavlovian conditioning that in Pavlovian preparations such as those of eyelid conditioning and human electrodermal conditioning, a stimulus that predicts the absence of the reinforcer (a so-called explicitly-unpaired conditional stimulus [CS]) should produce lower levels of performance than one that is merely uncorrelated with the reinforcer (a so-called truly-random CS). This consequence of the contingency position (first formulated by Prokasy [1965], and then by Rescorla [1967]) has been consistently disconfirmed in the experiments that compared acquisition functions to the "explicitly unpaired" and "truly random" CSs, although more complex post-acquisition assessments (e.g., transfer of training) have yielded some confirmations (see, e.g., Furedy & Riley 1987).

Still, one might object that the above set of facts pertain to relatively esoteric methodological issues about which Pavlovian conditioners still disagree (see, e.g., Furedy et al. 1975 vs. Prokasy 1975). A more obvious and gross violation of the facts by contingency-space accounts is that in eyelid conditioning preparations, the CS-US interval (between CS onset and US or unconditional-stimulus onset) is so crucial that CS-US intervals as short as 2 seconds produce no acquisition conditioning, even though it is patently obvious that the "informativeness" of the stimulus with respect to the reinforcement" (i.e., awareness of the CS/US relation) has been established.

Similarly, with heart-rate deceleration as the target response, when the CS-US interval is extended from 1 to 5 seconds, even repeated sessions fail to produce any acquisition in human subjects who are clearly aware of the contiguity relation between the tone CS and the negative-tilt US (Furedy 1992). This deficiency of the contingency position for predicting behavior is paralleled by a weakness for its control. As with Skinnerian conditioning, the most relevant applied situation for Pavlovian conditioning is that of teaching target behaviors that are beneficial and that are difficult to learn.

Whether the aim is to achieve conditioned heart-rate or blood-pressure decrease or autoimmune increase, any practitioner who is guided by a "contingency-space" account of Pavlovian conditioning will fail because this metaphorical concept ignores the non-propositional stimulus-response (S-R) processes, which, *in addition to propositional sign-significate* (S-S) processes, actually influence the phenomenon of Pavlovian conditioning (see, e.g., Furedy & Riley 1987).

Accordingly, although the "momentum metaphor" may "encompass" the Skinnerian explanatory concepts N&G advance in their target article, and although their analysis may be useful in such fields as economics where the psychology of human preference is a legitimate area of study, in my view the metaphorical explanatory physics-aping emperor has no scientific or applied psychological clothes.

Behavioral momentum in Pavlovian conditioning and the learning/performance distinction

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Abstract: Behavioral momentum theory has evolved within the realm of operant conditioning. The thought-provoking momentum metaphor equates the strength of an operant response with its resistance to change and preference (i.e., choice) for that response over other available responses. Whereas baseline response rate (velocity in the metaphor) is assumed to be largely influenced by the response-reinforcer contingency, resistance to change and preference are assumed to reflect an intervening variable called *behavioral mass*, which is determined primarily by the stimulus-reinforcer relationship. This invites the question of how well the momentum metaphor applies to the stimulus-reinforcer relationships of traditional Pavlovian paradigms. Presumably, a correspondence exists between behavioral mass and the notion of associative strength in the associative learning literature. Although response rate has little meaning in the trialwise structure of classical (i.e., Pavlovian) conditioning, response probability or magnitude might be regarded metaphorically as velocity. Momentum theory suggests that resistance to change (e.g., extinction) is a better indicator of associative strength than is response probability or magnitude. Therefore, variables that strengthen Pavlovian learning should influence resistance to extinction of conditioned responding in a similar manner. Moreover, it is important to assess momentum theory outside of strictly operant paradigms, particularly because in clinical settings many common disorders (e.g., phobias) and their therapies (e.g., cue exposure) are thought to be classically conditioned.

Behavioral momentum theory can be contrasted with the traditional theories of associative learning, which are exemplified by the Rescorla and Wagner (1972) model. Models of associative learning are considered dynamic theories because they have been con-

cerned primarily with the rate at which associative learning takes place. In contrast, momentum theory is typically considered a steady-state model in that it deals with asymptotic levels of responding. Whether the variables that determine behavioral mass also affect the rate at which that asymptote is reached is not explicitly stated by Nevin & Grace (N&G 2000) in their target article. Yet, resistance to change is by definition a dynamic measure and presumably reflects a dynamic process. The analogy to physical mechanics implies that the mechanisms of associative acquisition and extinction should oppose one another. In other words, variables that facilitate acquisition should impede extinction and vice versa. Moreover, the variables that enhance asymptotic behavioral mass should both decelerate extinction and accelerate acquisition. It would be interesting to see whether such a correlation exists. Research in this vein would encourage the development of behavioral momentum theory as a dynamic model of behavior.

Among the variables known to influence associative learning in Pavlovian situations are conditioned stimulus (CS) intensity, reinforcer magnitude, and CS rate within the context. (It is a separate question whether these specific attributes of experience or just their effects on learning are ultimately retained by the organism.) As predicted by the Rescorla-Wagner (1972) model, CS intensity appears to directly affect the rate but not the asymptote of learning, whereas reinforcer magnitude directly influences both rate and asymptote in the expected manner. In the Rescorla-Wagner model, CS intensity is captured by the learning rate parameter α, and reinforcer magnitude modulates λ, the total amount of learning that can occur, and β, a learning rate parameter for the unconditioned stimulus (US).

Although behavioral momentum theory is silent concerning the effect of CS intensity, reinforcer magnitude and rate are the two main variables the theory has used to establish different baselines of behavioral mass. It is with respect to reinforcer magnitude that the two theories contrast most sharply. Although both theories anticipate that greater reinforcer magnitude will increase the asymptotic strength of learning (mass or associative strength), they make different predictions regarding resistance to extinction following training with reinforcers of different magnitude. The Rescorla-Wagner model implies that during extinction β will be larger for a larger (now-omitted) reinforcer. Therefore, extinction (just like acquisition) of associative strength should proceed faster with the cessation of larger reinforcers than smaller reinforcers, even when baselines are normalized. This rather counterintuitive prediction is, of course, opposite to that of a Pavlovian extension of momentum theory, which anticipates that subjects trained with larger reinforcers will exhibit greater resistance to extinction.

The existing data based on traditional Pavlovian paradigms seem to favor the view of behavioral momentum theory. There is a positive relationship between resistance to extinction and magnitude of reinforcement. For example, Wagner et al. (1964) found more rapid salivary conditioning and greater resistance to extinction when the US consisted of six food pellets rather than one. Annau and Kamin (1961) and Kamin and Brimer (1963), varying the intensity of shock used as a US in conditioned suppression, found that higher intensity shocks facilitated acquisition and retarded extinction. Smith (1968) found that conditioning of the nictitating membrane response of rabbits and its resistance to extinction were directly related to the intensity of a shock US.

However, all of these studies used what would be analogous to continuous reinforcement (CRF) schedules; that is, all trials were reinforced. It is well known that in operant situations a direct relationship between reinforcer magnitude and resistance to change only holds for partial reinforcement (PRF) schedules, in which not every response is reinforced. With CRF schedules, greater magnitude of reinforcer often leads to more rapid extinction. This is an example of the partial reinforcement extinction effect with which behavioral momentum theory has struggled over the years (e.g., Nevin 1988). This latest version of momentum theory borrows the concept of generalization decrement, a mechanism that has been used many times before to explain partial reinforcement effects (e.g., Sheffield 1949). In fact, N&G's "augmented model" for resistance to extinction (equation 17 in the target article) is basically a formal statement of Capaldi's (1967) account of the interaction between reinforcement schedule and reward magnitude: Effectively, the generalization decrement involved in the transition from continuous reinforcement to extinction outweighs the "reinforcing power" (mass) gained by using a larger reinforcer. With PRF schedules, the amount of generalization decrement is presumably less, allowing the pattern anticipated by behavioral momentum theory to emerge. But a discrepancy remains: Why was there a positive relationship between resistance and US magnitude when all trials were reinforced in the Pavlovian experiments cited above? There is a literature suggesting that the effect of generalization decrement is less pronounced in Pavlovian tasks (e.g., Gormezano 1966) because the short CS-US interval favors a broad stimulus generalization gradient. Therefore, the short intervals typically found between the CS and the US in Pavlovian conditioning mitigate against the partial reinforcement extinction effect, thereby leaving unopposed the effects of reinforcer magnitude on resistance to change.

Leaving these issues aside, N&G draw a strong distinction between learning and performance, a long-known (e.g., Tolman 1932) but often overlooked (e.g., Rescorla & Wagner 1972) dichotomy that we have examined in our laboratory over many years (e.g., Miller & Matzel 1988). Therefore, we welcome N&G drawing attention to this distinction. However, for them, response rate (metaphorical velocity) is influenced by the operant (response-reinforcer) contingency, whereas resistance to change and preference (metaphorical behavioral mass) are influenced primarily by the Pavlovian (stimulus-reinforcer) contingency. Thus, behavioral momentum (resistance to change) and velocity (response rate) appear differentially sensitive to two different contingencies. This is not necessarily the learning-performance distinction, but rather different measures reflecting two different memories. There is ample evidence that organisms learn about both stimulus-reinforcer and response-reinforcer relationships (e.g., Colwill & Rescorla 1985). Simply noting that resistance to change and response rate are sensitive to different variables may not warrant classifying one measure as an indicator of learning while relegating the other to the status of a performance variable, particularly because the authors acknowledge several exceptions to the assumption that resistance to change is determined solely by stimulus-reinforcer relations.

Authors' Response

Behavioral momentum and Pavlovian conditioning

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Abstract: The constructs of behavioral mass in research on the momentum of operant behavior and associative strength in Pavlovian conditioning have some interesting parallels, as suggested by **Savastano & Miller**. Some recent findings challenge the strict separation of operant and Pavlovian determiners of response rate and resistance to change in behavioral momentum, renewing the need for research on the interaction of processes that have traditionally been studied separately. Relatedly, **Furedy** notes that some autonomic responses may be refractory to conditioning, but a combination of operant contingencies and enriched Pavlovian stimulus-reinforcer relations may prove effective.

Our target article suggested that the components of behavioral momentum – an established rate of responding and its resistance to change, which we take to be analogous to velocity and mass in Newtonian physics – were separately determined by operant response-reinforcer contingencies and Pavlovian stimulus-reinforcer relations. Although separate research traditions have evolved for operant and Pavlovian conditioning, with different theories, methods, and even philosophical orientations, ultimately "what is learned" must be related. The defining feature of Pavlovian conditioning may be the stimulus-reinforcer (i.e., CS-US) contingency, but no learning can be detected in the absence of a response. Conversely, the operant response-reinforcer contingency must always occur in a stimulus context. By emphasizing the importance of stimulus-reinforcer contingencies as determiners of resistance to change, behavioral momentum theory (Nevin & Grace 2000) has always recognized the connection between Pavlovian and operant conditioning.

Savastano & Miller return the favor by drawing a parallel between behavioral mass – which according to momentum theory determines the resistance to change of an operant response – and associative strength, the primary learning construct in Pavlovian conditioning. They cite several studies which show that resistance to extinction of Pavlovian conditioning is positively related to reinforcement magnitude. This is important because if we can show that the same variables affect associative strength and behavioral mass in similar ways, then it is plausible that they represent a single construct. In fact, one of the major goals of our target article was to show that behavioral mass and conditioned reinforcement value could be understood in an expanded metaphor of behavioral momentum. The resulting integration of research on resistance to change and preference implies that behavioral mass and conditioned value represent a single construct. Because Pavlovian processes have long been assumed responsible for the acquisition of conditioned value, Savastano & Miller have closed the circle by linking associative strength and behavioral mass. This may help to build more comprehensive models of conditioning, and we thank them for pointing it out.

Savastano & Miller also note that Pavlovian studies have used continuous reinforcement (CRF) procedures, in contrast to most operant research on resistance to extinction, which has used intermittent or partial reinforcement (PRF). The puzzle then is why CRF often produces the opposite result for operant procedures (i.e., greater resistance for the alternative with the smaller reinforcer; see Case 2000). In our target article (Nevin & Grace 2000), we proposed an augmented model for resistance to extinction:

$$\log \frac{Bx}{Bo} = \frac{-x(c + dr)}{r^a}, \qquad (1)$$

where *Bx* and *Bo* are response rates during extinction and baseline, respectively, *x* is session number, and *r* is reinforcement rate (or magnitude), with sensitivity parameter *a.* The denominator on the right-hand side of Equation (1) is behavioral mass. The disruptive effects of extinction are captured by *c,* which represents the impact of suspending the response-reinforcer contingency, and *d,* which multiplies reinforcement rate (or magnitude) to give generalization decrement. To apply Equation (1) to Pavlovian conditioning, *c* is now interpreted as the effects of suspending the stimulus-reinforcer contingency (e.g., presenting the US with equal probability in the presence and absence of the CS). Equation (1) then predicts increased resistance to extinction for the relatively larger reinforcement magnitude or rate provided that *d* is relatively low, consistent with Savastano & Miller's suggestion that there is a broad stimulus generalization gradient in Pavlovian conditioning.

Savastano & Miller also question whether it is possible to maintain a strict learning-performance distinction, with resistance to change equated with learning (i.e., effects of reinforcement history) and response rate viewed as a performance variable. Although we originally took the strong position that resistance to change depended solely on the stimulus-reinforcer contingency and was independent of response rate, recent studies suggest that in some cases the response-reinforcer contingency and the resulting response rate may affect resistance to change. Nevin et al. (2001) compared resistance to change and preference for variable interval (VI) and variable ratio (VR) schedules with reinforcement rates equated, and found that response rate was greater for VR but resistance to change was greater for VI responding. Moreover, the VI schedule was preferred in a concurrent-chains preference test. Relatedly, Grace et al. (1998) found that an unsignaled delay of reinforcement decreased both response rate and resistance to change relative to immediate reinforcement at the same rate, and, again, immediate reinforcement was preferred (see Nevin & Grace 2000, p. 84). It appears to be that when stimulus-reinforcer contingencies are equated, the response-reinforcer contingency can affect resistance to change in some situations, but when response-reinforcer contingencies are equated (e.g., in a multiple VI VI schedule), differential resistance to change will depend on the stimulus-reinforcer contingency. At present, the relative contributions of operant and Pavlovian contingencies to resistance to change (i.e., behavioral mass or, perhaps, associative strength) are unknown. This issue should be addressed in future research.

Furedy's commentary is also concerned with operant and Pavlovian contingencies, specifically with the failure of certain preparations to establish reliable conditioned responding. However, his "brute facts" simply do not bear on the domain of behavioral momentum theory, which does not attempt to explain the vagaries of acquisition, some of them idiosyncratic and some of them attributable to biological constraints (e.g., the difficulty of maintaining lever pressing by a rat when the reinforcer changes from food to shock avoidance). Our starting point is ongoing operant behavior maintained by an effective reinforcer, and most of the results we cite have been replicated with several species, stimuli, responses, and disruptors. It would be marvelous if we could enunciate a "law" that "fully accounted for the behavior of living organisms" but our goals are more modest.

With respect to biofeedback, **Furedy** asserts that operant and Pavlovian contingencies have no beneficial effect on autonomically controlled cardiac responses. There is, however, evidence that our approach is directly relevant to skeletal muscle relaxation. As described in our target article (Nevin & Grace 2000, p. 86), Tota-Faucette (1991) gave auditory feedback and points to children for meeting a relaxation criterion. In some stimulus conditions, she also gave extra points or candy, independently of how well the

children were performing the relaxation task. These extra reinforcers strengthened the stimulus-reinforcer relation, and when auditory feedback was discontinued (extinction), relaxation was reliably more persistent in the added-reinforcer conditions. The finding exactly parallels results obtained with pigeons, key pecking, and food (Nevin & Grace 2000, p. 77) and replicated many times with other species, responses, and reinforcers, most recently by Ahearn et al. (2003) with autistic children engaged in stereotyped behavior. Thus, our approach can guide effective application. To our knowledge, it has not yet been applied to autonomically mediated cardiac responses but it would be worth trying.

A part of the task of any scientific theory is to identify its domain of applicability, and behavioral momentum theory need not be all-encompassing in order to be useful. For a century, we have known that Newton's laws apply only to macroscopic bodies moving at velocities substantially less than the speed of light, but they still serve superbly for many engineering applications. **Savastano & Miller**'s commentary suggests that our approach may apply to a variety of classical conditioning preparations, and we look forward to expanding the domain and the utility of behavioral momentum theory through convergence with other lines of research and theory.

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Commentary on **Michael Domjan, Brian Cusato, & Ronald Villarreal (2000). Pavlovian feed-forward mechanisms in the control of social behavior. BBS 23(2):235–282.**

Abstract of the original article: The conceptual and investigative tools for the analysis of social behavior can be expanded by integrating biological theory, control systems theory, and Pavlovian conditioning. Biological theory has focused on the costs and benefits of social behavior from ecological and evolutionary perspectives. In contrast, control systems theory is concerned with how machines achieve a particular goal or purpose. The accurate operation of a system often requires feed-forward mechanisms that adjust system performance in anticipation of future inputs. Pavlovian conditioning is ideally suited to subserve this function in behavioral systems. Pavlovian mechanisms have been demonstrated in various aspects of sexual behavior, maternal lactation, and infant suckling. Pavlovian conditioning of agonistic behavior has been also reported, and Pavlovian processes may likewise be involved in social play and social grooming. Several further lines of evidence indicate that Pavlovian conditioning can increase the efficiency and effectiveness of social interactions, thereby improving their cost/benefit ratio. We extend Pavlovian concepts beyond the traditional domain of discrete secretory and other physiological reflexes to complex real-world behavioral interactions and apply abstract laboratory analyses of the mechanisms of associative learning to the daily challenges animals face as they interact with one another in their natural environments.

Learning theory, feed-forward mechanisms, and the adaptiveness of conditioned responding

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Abstract: The specific mechanisms whereby Pavlovian conditioning leads to adaptive behavior need to be elaborated. There is no evidence that it is via reduction in the "destabilizing effect that time lags have on feedback control" (Domjan et al. 2000, sect. 3.3). The adaptive value of Pavlovian conditioning goes well beyond the regulation of social behavior.

From the perspective of researchers who study learning processes, the target article is a highly original method of communicating to researchers in many fields how Pavlovian conditioning (PC) and operant conditioning interact to produce adaptive behavior. The Pavlovian mechanism provides a source of adaptive variants (selected by evolution) that are then honed by operant feedback mechanisms. The target article provides a new and, perhaps, more general language for discussing issues that have been treated by various theories of operant-Pavlovian interactions (Davis & Hurwitz 1977; Locurto 1981; Rescorla & Solomon 1967; Williams 1981). These issues include the influence of feedback on the form of the Pavlovian conditioned responses (Konorski & Miller 1937; Prokasy 1965) and the modulation of operant (Rescorla & Solomon 1967) and unconditioned responses (UR's) (Brown et al. 1983; Kimmel 1966) by Pavlovian conditioned stimuli (CS's). The emphasis in this article is on how the Pavlovian component contributes to the adaptiveness of behavior (cf. Hollis 1997; Prokasy 1965; Zener 1937). What is new in this presentation is the specific suggestion that the adaptive function of Pavlovian conditioning is to reduce the "destabilizing effect that time lags have on feedback control" (Domjan et al. 2000, sect. 3.3).

The target article does indeed present some good examples of the adaptive value of PC in the domain of social behavior. For example, it documents the role of sexual conditioning in reproductive behavior. It is evident that the Pavlovian CS decreases time to copulation (Domjan et al. 1998; Hollis et al. 1989; Zamble et al. 1985) and that CS exposure can even lead to greater competitiveness (Guttierez & Domjan 1996) and reproductive success (Hollis et al. 1997). However, it is not demonstrated that the adaptiveness arises via attenuation of a destabilizing effect of time lags. If there were a destabilizing effect of time lags, then we would expect to find some evidence of destabilization, such as increased behavioral variability, in those animals not exposed to the CS. In all of the examples cited, the average performance is enhanced by presentation of a Pavlovian CS, but there is no evidence of variability differences attributable to the Pavlovian experience.

The adaptive value of PC is broader than described in the target article (cf. Hollis 1997). For example, a very important function of PC is the production of specific outputs that are essential for inducing new behaviors during ontogeny. During behavioral development it is sometimes essential that a behavior occur in roughly accurate form the first time it occurs. Said another way, for feedback to be able to mold behavior into efficient forms it must first occur in a form that is a close enough approximation to the skilled response so that feedback can be effective. PC provides a feed-forward mechanism for this induction. For example, pairings of odor with tactile stimulation seem to underlie initial nipple attachment in rats (Pederson & Blass 1982; Petrov et al. 1999). Successful ingestion of milk seems to further contribute to the effectiveness of attachment (Petrov et al. 1999). The initial Pavlovian contingency gives rise to the behavior on which feedback operates. Another example of this comes from our own work on the development of independent feeding in ring doves. A Pavlovian contingency between the sight of seed and parental feeding induces thrusting at seed (Balsam et al. 1992; Deich & Balsam 1993). Beak opening is induced by the thrusting, and feedback from successful pecks molds the behavior into an adult coordination (Balsam et al. 1993; Deich et al. 1995). These behaviors are induced by PC that takes place during social interactions, and they influence future social feeding (Iskrant & Balsam 1994). In each case, the PC gives rise to a response that is then further modified by feedback.

The target article's unification of biological and learning theories stems from its specification of PC as one of the "proximate mechanisms that increase the utility of social behavior" (sect. 5). The results reviewed in section 4 and the additional examples that we cite above are supportive of this role for PC. But we take issue with the authors' contention that other such proximate mechanisms have yet to be specified by science. First, the adaptiveness of mechanisms that elicit social UR's, modal action patterns, and other species-typical social behaviors has been elaborated in many specific instances (Brown 1975). Second, Pavlovian conditioning is not the lone feed-forward mechanism in behavioral regulation. Habituation, sensitization, and imitation are all feed-forward mechanisms that can give rise to adaptive behavior forms prior to (and subsequent to) the operation of feedback (Balsam & Silver 1994; Balsam et al. 1998; Deich & Balsam 1994).

In conclusion, we agree that PC is an important mechanism of adaptive behavior but we would say that there is not enough evidence to conclude that its adaptiveness arises from a single control process – not even in the domain of social behavior.

Authors' Response

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For the authors' responses to comments similar to those expressed here by **Balsam & Drew**, please see: M. Domjan, B. Cusato, & R. Villarreal (2000). Extensions, elaborations, and explanations of the role of evolution and learning in the control of social behavior. BBS 23(2):269–82. [Authors' Response to first round of commentary.]

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Commentary on **Mike Page (2000). Connectionist modelling in psychology: A localist manifesto. BBS 23(4):443–512.**

Abstract of the original article: Over the last decade, fully distributed models have become dominant in connectionist psychological modelling, whereas the virtues of localist models have been underestimated. This target article illustrates some of the benefits of localist modelling. Localist models are characterized by the presence of localist representations rather than the absence of distributed representations. A generalized localist model is proposed that exhibits many of the properties of fully distributed models. It can be applied to a number of problems that are difficult for fully distributed models and its applicability can be extended through comparisons with a number of classic mathematical models of behaviour. There are reasons why localist models have been underused, though these often misconstrue the localist position. In particular, many conclusions about connectionist representation, based on neuroscientific observation, can be called into question. There are still some problems inherent in the application of fully distributed systems and some inadequacies in proposed solutions to these problems. In the domain of psychological modelling, localist modelling is to be preferred.

The local is running on the express track: Localist models better facilitate understanding of nervous system function

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Abstract: Artificial neural networks have weaknesses as models of cognition. A conventional neural network has limitations of computational power. The localist representation is at least equal to its competition. We contend that locally connected neural networks are perfectly capable of storing and retrieving the individual features, but the process of reconstruction must be otherwise explained. We support the localist position but propose a "hybrid" model that can begin to explain cognition in anatomically plausible terms.

Whether they use localist or distributed representations, artificial neural networks as they are currently conceived have a serious weakness if they are able to be applied as models of natural cognition. It is well known from anatomical and physiological studies that neurons have a finite connection range beyond which the probability of connection of two cells decreases exponentially. This range is about 250 μ in the visual cortex (Krone et al. 1986) and 400 μ in the hippocampus (Traub et al. 1988). On the other hand, neural networks are constructed so that almost all cells in a given layer are connected to all the cells in some other layer. This situation could only be approximately applicable if all the cells fell within a single connection range. Using the figures of Wilson and Cowan (1973), about 10,000 cells could fit within a cylinder of radius of one connection length and thus constitute such a locally *connected* network.

In contrast, the cortex of the edible frog has about 6 million cells (Wilson & Cowan 1973). Making all allowances for the efficiency of scientifically constructed learning algorithms compared with what has been contrived by nature, it is not likely that a conventional neural network operating under the constraints of brain anatomy could have computational power greater than that of a frog. This is not to detract from the demonstrated abilities of neural networks whose nodes and/or activation states are given semantic significance; we simply question their direct relevance to animal psychology.

For the purposes of this discussion, we accept the arguments of the author that the localist representation is at least equal to its competition. The first question we must ask is how much information an actual neuron can store. There might very well be a "grandmother cell" because of the childhood implantation, but it is highly unlikely that anyone other than immediate family has a "Mike Page" cell. Yet we are capable of recognizing numerous relative strangers, largely through the reconstruction of distributively stored details (Bartlett 1932). We contend that locally connected neural networks are perfectly capable of storing and retrieving the individual features, but the process of reconstruction must be otherwise explained.

A rat negotiating a maze, for example, has memory known to be implanted through the action of single hippocampal "place" and "direction" cells (Muller et al. 1996; Rolls et al. 1998). The choice of direction at any point, however, must be assembled from this stored fragmentary data. Of particular significance is the recent work of Fried and colleagues (1997) who measured the activity of single hippocampal neurons during recognition tests. They found that some neurons responded differentially to faces and inanimate objects, some to different emotional expressions, and some to combinations of expression and gender. This would be, in our opinion, the right amount of content a node of an anatomically constrained localist neural network could hold.

What mechanism can connect the elementary localist networks? Perhaps a network that takes Euclidean distance into account can be devised. None exists as far as we know, so questions about how ability is degraded by systematic incompleteness of connections cannot be answered. There have been some simulations incorporating connection range (Krone et al. 1986; Traub et al. 1987; 1988; 1989), but these have not been given any cognitive content; rather, they are concerned with the general behavior of the neural medium.

This area of study was first suggested by Wilson and Cowan (1973), who created what can be called a continuum theory of neural tissue. The differential elements of this medium contain many cells; in modern terms they can be called *neural networks,* although such terms as "cell assemblies" (Hebb 1949) or "groups" have been applied. These elements may or may not be functional wholes; their constituent cells might change with task. However, it is logical to give each, at any time, the computational abilities of a localist neural network.

According to the theory, the elements are interconnected by probabilistically distributed connections of type e-e, e-i, i-e, and i-i, between (literal or figurative) "excitatory" and "inhibitory" "cell" species. The existence of e-e connections signifies an internal source of (electrochemical) energy, so that the medium can support growing disturbances. The conditions of growth give rise to waves with sharply defined wavelength spectra (Koch & Leisman 1990; 1996; 2001; Leisman & Koch 2000). The preferred wavelengths are controlled by the synaptic parameters, and they are always significantly longer than the largest average synaptic connection range.

We postulate that this phenomenon provides a mechanism by which the active nodes of the elementary networks can be "connected." Unlike a neural network, the connection is not causal; instead, two nodes are simultaneously active when the distance between them is an integral multiple of the favored wavelength, which can be changed by changes in the state of the relevant brain region.

We have applied this concept to models of the brain stem, cortex, and hippocampus (Koch & Leisman 1990; 1996; 2001; Leisman & Koch 2000). The last studies are of particular interest for the present discussion. The growth properties of hippocampal waves change as synapses are modified by Hebbian conditioning (Hebb 1949; 1972). Such a process exists in the hippocampus (Eccles 1986), manifested by strengthening in synapses between geometrically and synaptically neighboring cells. This is certainly akin to local weight modification in neural networks, but it has important global effects. Not only does it change the nature of the waves from decay to growth, but it also controls the amplified wavelength and therefore the connections among the networks.

Hence our title: The local ultimately ramifies throughout cognitive regions. We hope to see in the future a "hybrid" model that can begin to explain cognition in anatomically plausible terms.

Local versus distributed: A poor taxonomy of neural coding strategies

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Abstract: Page is to be congratulated for challenging some misconceptions about neural representation. However, his target article, and the commentaries to it, highlight that the terms "local" and "distributed" are open to misinterpretation. These terms provide a poor description of neural coding strategies and a better taxonomy might resolve some of the issues.

Consider a neural network in which individual nodes represent single letters of the alphabet. When a letter is presented to this

network a node responds, providing a localist representation. However, if two (or more) letters are simultaneously presented, two (or more) nodes will be active and the representation would now be described as distributed. The same network thus generates local or distributed representations in different circumstances. In his Response to the commentaries, Page (2000b) makes this point himself: "under the appropriate task demands, such a layer might process a stimulus as a distributed pattern of activation across localist representations"(p. 497). On that ground, local or distributed representations are not a property of the network (alone) and it is questionable whether such labels are useful in describing models. Furthermore, there are two dimensions along which neural coding strategies need to be classified: the tuning properties of the nodes and the number of nodes involved in the representation (Jelasity 2000).

Tuning properties. In the above example of a network representing letters, the preferred stimulus of each individual node has a clear interpretation. Such a node might be narrowly tuned to a specific stimulus or it might be widely tuned, responding over a range of inputs with varying degrees of similarity to the preferred stimulus. Such a node might respond to the same letter written in a variety of fonts or to a specific letter appearing at any location within the receptive field, or to both. However, in all of these cases it is possible to ascribe meaning to the node's activity in isolation: it acts as a feature detector or a matched filter (Barlow & Gardner-Medwin 2000). An alternative response property, which has been described as "(fully/dense) distributed," "compact," or "ensemble" coding, requires unique combinations of active nodes to represent meaningful features of the environment. In such a scheme the responses of isolated nodes do not have an obvious interpretation (Page 2000a). An example is the binary ASCII code in which any individual bit will be ON for an arbitrary set of characters (Barlow & Gardner-Medwin 2000).

A feature detector responds to a set of stimuli to which we can apply a meaningful label, but in a compact code a node responds to stimuli for which there is no such simple classification (Hummel 2000). That the distinction is purely one of interpretation should come as no surprise. Similar activation functions (e.g., weighted summation followed by thresholding) are used in both cases, and similar learning rules can also be used; that is, pseudo-Hebbian learning can generate compact codes such as Principal Components (Oja 1982; Sanger 1989), but is also widely used to learn feature detectors (Földiák 1990; Wallis & Rolls 1997). The arbitrariness of the distinction is further illustrated by differing interpretations of the response properties of simple cells in the primary visual cortex. The receptive fields of these cells have been shown to be similar to the independent components of natural images (Bell & Sejnowski 1997; van Hateren & Ruderman 1998); however, they are also routinely described as edge detectors. Physiological evidence for correlations between the activity of individual neurons and behaviour (Georgopoulos et al. 1986; Newsome et al. 1989) and between the activity of individual neurons and sensory stimuli (Logothetis & Sheinberg 1996; Perrett et al. 1992; Tanaka 1996) suggests that cortical cells are generally tuned to meaningful categories. Such tuning properties may be readily learnt since recurring patterns of stimuli are likely to correspond to features of the environment (Barlow 1972; 1994; Edelman & Duvdevani-Bar 1995).

Coding density. For the network responding to letters, described above, individual letters are explicitly represented whereas combinations of letters are implicitly represented. Similarly, in a hierarchical network the information implicitly represented by the response of many nodes at one level may be explicitly represented by the response of individual nodes at a higher level. A single node therefore explicitly represents a particular feature of the input stimulus and it may do so independently of the activity of other nodes or as part of an implicit representation of the stimulus as a whole.

The cortex explicitly and implicitly encodes information. For example, cells in area V1 provide explicit representations of oriented edge features at specific locations. However, they must also provide an implicit representation of all visual stimuli that can be distinguished by the visual system and which may (or may not) be explicitly represented in higher cortical areas. Since all visual information is available in V1 (and for that matter in the LGN and at the retina), it must be advantageous to recode information so that more abstract entities become more explicitly represented. The appropriate level of abstraction will vary between tasks: it would be equally impractical to explicitly represent every possible event using single nodes as it would be to use a representation in which all events were only implicitly represented using low-level feature detectors (Feldman 1990; Tsotsos 1995; Wilson 1991). Furthermore, such recoding makes learning tractable by transforming complex relational tasks into simpler statistical problems over the recoded data (Clark & Thornton 1997). What information is explicitly represented should depend on the environment and the importance of those stimuli to the task/animal (Logothetis 1998).

Conclusions. Describing networks along a single local versus distributed dimension fails to make explicit the properties of the coding strategy employed. Specifically, Page (2000a) uses the term *local* to describe tuning properties without making a commitment to the coding density, and he uses the term *distributed* to refer to the coding density without specifying tuning properties. He is therefore correct in his assertion that these terms are not dichotomous, but only because they are being used to describe independent properties.

Although most models employ nodes with similar tuning properties, there are significant differences between models in terms of coding density. Constraints on the sparsity or density of activity influence whether information is encoded explicitly or implicitly. For example, a winner-takes-all (WTA) network will need to explicitly encode each stimulus that is to be represented, while a k-WTA network will be biased towards forming implicit representations. Such constraints are commonly employed in unsupervised learning algorithms, resulting in networks unable to cope with tasks that require an arbitrary number of active nodes – even tasks as simple as responding to single or multiple letters. Such constraints result in poor models of cortical coding and may have contributed to the delusion that the cortex is also committed to using either local or distributed representations.

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Commentary on **J. Allan Hobson, Edward F. Pace-Schott, & Richard Stickgold (2000). Dreaming and the brain: Toward a cognitive neuroscience of conscious states. BBS 23(6):793–842.**

Abstract of the original article: Sleep researchers in different disciplines disagree about how fully dreaming can be explained in terms of brain physiology. Debate has focused on whether REM sleep dreaming is qualitatively different from nonREM (NREM) sleep and waking. A review of psychophysiological studies shows clear quantitative differences between REM and NREM mentation and between REM and waking mentation. Recent neuroimaging and neurophysiological studies also differentiate REM, NREM and waking in features with phenomenological implications. Both evidence and theory suggest that there are isomorphisms between the phenomenology and the physiology of dreams. We present a three-dimensional model with specific examples from normally and abnormally changing conscious states.

Drug induced alterations in dreaming: An exploration of the dream data terrain outside activation-synthesis

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Abstract: Two meta-analyses of pharmacological research are presented, demonstrating that psychoactive drugs have consistent effects on EEG and sleep outside of their effects on REM sleep, and demonstrating that drugs other than those affecting sleep neurotransmitter systems and REM sleep can also alter reported nightmare occurrence. These data suggest that the neurobiology data terrain outside activation-synthesis may include sleep and dream electrophysiology, cognitive reports of dreaming, effects of alterations in consciousness on dreaming, immunology and host defense, and clinical therapies for sleep disorders.

The most accepted approach to addressing the obvious complexity of known components of central nervous system (CNS) electrophysiology and neurochemistry is to approach analysis of the system deductively, using selected data from many different areas to support a theoretical construct. Unfortunately, if this approach is utilized to present a purportedly broad-based review for prospective theorists in the field, data that are inconsistent or noncontributory to that theoretical construct (an amended activation-synthesis hypothesis) are excluded and ignored. This approach attempts to guarantee that future researchers and theorists in sleep and dreaming will work within the constraints of that model – a model currently requiring extensive restructuring and remodeling to encompass the experimental data of its supporters (see Nielsen 2003; Hobson et al. 2003). This commentary presents pharmacological data from two of these excluded areas: (i) psychoactive drug alteration of sleep stages and background EEG frequencies inclusive of alterations in REM sleep (REMS), and

Table 1 (Pagel). *Psychoactive drug effects on sleep and EEG frequencies*

Medication Class	EEG Frequency Effects	Sleep Stage Effects
Amphetamines	Increased – beta: Decreased – theta, Delta	Increased sleep latency: Decreased deep sleep
Benzodiazepines	Changes in delta amplitude	Decreased REMS REMS rebound on withdrawal Increased stages 1 & 2
Ethanol	Diffuse slowing	Decreased REMs, sleep latency, REMs rebound on withdrawal
Lithium	Episodic slowing	Increased stage 4
Opiates	Increased delta: Decreased alpha	Increased stage 4
Pheothiazines	Increased theta: Decreased alpha, sigma	(+/−)
Gamma hydroxy butyrate	Increased delta	Decreased stage 1 Increased stages 3 & 4
L-dopa	(0)	Decreased REMS Increased stage 1, sleep latency

Anti-Depressant Class	Drug		
Tricyclic	Trimipramine Nortriptyline Doxepin Amoxapine Amitryptyline Imipramine Amoxapine Protriptyline	Increased beta Decreased delta	Increased REMS latency Decreased REMS (++), sws latency Deep sleep, sleep latency
Non-tricyclic sedating	Desimprinine Maprotiline Mirtazapine	Increased-beta	Increased REMS latency Decreased -sws latency, REMS (++), sleep latency
Maoi	Phenelzine Tranylcypromine	(0)	Increased stage 4 Decreased REMS latency, REMS (+++)
Ssri	Fluoxetine Paroxetine Sertraline Fluvoxamine Citalopram hbr	Increased alpha and eog activity stage 1,	Increased REMS latency, sleep latency, stage 1 Decreased REMS
Ssri + tricyclic	Venlafaxine	(0)	Increased REMS latency Decreased sleep latency, REMS
Da-na-ssri	Bupropion	(0)	Increased REMS latency, sleep latency
Non-tricyclic Non-ssri	Nefazodone	(0)	Increased REMS Decreased sleep latency
5ht 1a agonist	Buspirone	(0)	Increased REMS latency: Decreased REMS

Note: (0) = not studied; (++) = higher levels of effect; (+/−) = reports of both negative and positive effects: delta (1.0–1.5 Hz), theta (7–9 Hz), alpha (8.5–10 Hz), sigma (12–16 Hz), beta (22–27 Hz). (Adapted from Pagel 1993; 1996.)

(ii) medications reported to induce nightmares in clinical trials and case reports. It is hoped that this information will prove useful to both theorists and researchers involved in the study of the neurobiology of the dream state.

Psychoactive drug effects on sleep stages and EEG frequencies. Medications that clinically produce psychoactive effects alter the recording of the electroencephalogram (EEG), the recording of brain electrical potential changes. Specific EEG epiphenomenon (e.g., seizure activity, PGO spikes) are well described, yet the origin of background EEG frequencies, which characterize the EEG particularly during sleep, remain a topic of open debate (Christakos 1986; Elul 1971). In general, drug-induced EEG changes are associated with characteristic behavioral effects (Hermann & Schaerer 1986; Itil 1981). This association has been utilized in developing therapeutic approaches for new medications producing characteristic EEG effects (Mandema et al. 1992). Typically, psychoactive medications alter background EEG frequencies as well as the occurrence, frequency, and latency of the various sleep stages including REM sleep, as charted in Table 1 (Pagel 1993; 1996; Pagel & Helfter 2003). Dream reports can be ob-

Table 2A (Pagel). *Medications Affecting CNS Neurotransmitter Systems Reported to Induce Nightmares in Clinical Trials and Case Studies*

Affected Neuroreceptor Drug	Patient Reports of Nightmares – Evidence Base Clinical Trials (CT) Case Reports (CR)	Probability Assessment of Drug Effect
ACETYLCHOLINE – Cholinergic Agonists		
Donepezil	CT [3/747 report disordered dreaming]	Possible
Rivastigmine	CT [1/100–1/1000 report disordered dreaming	Possible
Tacrine	CT [1/100–1/1000 (2076) report disordered dreaming	Possible
NOREPINEPHINE – beta blockers		
Atenolol	CT [3/20 patients]	Probable
Betaxolol and carbachol [opth.]	CR [1] – de-challenge	Possible
Bisopropol	CT [3/68 patients] : CR [1] – de-challenge	Probable
Labetalol	CT [5/175 patients]	Probable
Oxprenolol	CT [11/130 patients]	Probable
Propranolol	CT [8/107 patients]	Probable
– Norepinephine effecting agents		
Atomoxetine	CT [4/269 abnormal dreams compared to 3/263 placebo group]	Possible
Deserpidine	CT – disordered dreaming listed as side effect	Possible
Guanethidine	CT [4/48 patients] Probable	
Methyl dopa	CT [infrequent reports of nightmares]	Possible
Tramadol	CR [1] – de-challenge Possible	
SEROTONIN – SSRI		
Fluoxetine	CT [1–5% – greater frequency in OCD and bulemic trials: CR [4] – de & re-challenge	Probable
Escitalopram oxylate	CT [Abnormal dreaming – 1 % 999 patients]	Probable
Nefazodone	CT [3% (372) versus 2% control] Probable	
Paroxetine	CT [4% (392) versus 1% control] Significant	
Sertraline CT [1/100–1/1000] Possible		
Agents effecting serotonin & norepinephrine		
Protriptyline	CT – nightmares listed as side effect	Possible
Trazadone	CR [reports abnormal dreams] Doubtful	
Risperidone	CT [1% increased dream activity – 2607 patients]	Probable
Venlafaxine	CT [4% (1033) versus 3% control] Probable	
DOPAMINE – agonists		
Amantadine	CT [5% report abnormal dreams]: CR [1]	Probable+
Bupropion	CR [1] – de-challenge Possible	
Cabergoline	CT [1/188 patients]: CR [1] – de-challenge	Possible
Levodopa	CT [2/9 patients]	Probable
Pergolide	CT [2.7% (189) report abnormal dreams versus 4.5% placebo]	Doubtful
Ropinirole	CT [3% (208) report abnormal dreaming versus 2% placebo]	Probable
Selegiline	CT [2/49 reporting vivid dreams]	Probable
– Amphetamine-like agents		
Bethanidine	CT [2/44 patients]	Probable
Fenfluramine	CT [7/28 patients]: CR [1] de & re-challenge	Probable
Phenmetrazine	CT [3/81 patients]	Probable
GABA		
Flunitrazepam	CT [1/127 patients]	Possible
Gabapentin	CT [1/100–1/1000 (2074) report abnormal dreams]	Possible
Gaba hydroxy buterate	CT [nightmares >1% 473 patients]	Probable
Nitrazepam	CR [2]	Possible
Triazolam	CT [7/21 patients]	Probable
Tiagabine	CT [3/2531 patients]	Possible
Zopiclone	CT [3 – 5/83 patients]	Probable

KEY: (+)Listed under multiple drug classification

tained on awakening from all stages of sleep. Potential electrophysiological correlates for dreaming clearly exist outside adapted versions of the activation-synthesis hypothesis based on the postulate that REM sleep equals dreaming.

Drug-induced nightmares. The effects of medications on dreaming are not generally included in clinical trials and case reports, except as reports of nightmares – vivid and terrifying mental experiences occurring during sleep. Recent pharmacological

Table 2B (Pagel). *Other Drug Classes Reported to Induce Nightmares in Recent Case Reports and Clinical Trials*

Drug Class Drug	Patient Reports of Nightmares Clinical Trials (CT) Case Reports (CR) – Evidence Base	Probability Assessment of Drug Effect
ANESTHETICS		
Ketamine	CR [1]	Possible*
midazolam	CT [<1%]	Possible*
ANTI-INFECTIVES & IMMUNO-SUPRESSANTS		
amantadine	CT [5% reporting abnormal dreams]: CR [1]	Probable+*^
ciprofloxacin	CR [1] – de-challenge	Possible^
erythromycin	CR [2] – de-challenge	Possible
Fleroxacin	CT [7/84 patients]	Probable^
ganciclovir	CR [1] – de & re-challenge	Probable*^
gusperimus	CT [13/36 patient]	Probable
ANTI-EPILEPTICS		
Ethosuximide	CT [reports of night terrors]	Possible*
Lamotrigine	CT [1/100–1/1000 report abnormal dreams]	Possible*^
valproic acid	CR [1] – de-challenge	Possible*^
Zonisamide	CT [1/100–1/1000 report abnormal dreams]	Possible^
ANTI-PSYCHOTICS		
chlorpromazine	CR [1] – de-challenge	Possible*^
Clozapine	CT [4%]	Probable*^
Thiothixene	CR [3] – de-challenge	Possible*^
ANTI-HISTAMINE		
chlorpheniramine	CT [4/80 patients]	Probable*^
ACE INHIBITORS		
Captopril	CR [1]	Possible*^
Enalapril	CT [.5–1% abnormal dreaming – 2987 patients]	Probable*^
Losartin potassium	CT [>1% dream abnormality – 858 patients]	Probable*^
Quinapril	CT	Probable*^
OTHER AGENTS – NO PROPOSED MECHINISM		
buprenorphrine	CR [1] – de-challenge	Possible
Digoxin	CR [1] – de & re-challenge	Probable
Naproxen	CR [1] – de & re-challenge	Probable*^
Verapamil	CR [1] – de & re-challenge	Probable^

KEY – (+)Agents listed in multiple classes: (*) Agents inducing daytime sedation as a side effect to use:
(^) Agents inducing insomnia as a side effect to use.

A qualitative probability assessment is used to determine the probability that nightmares are drug induced by these agents based on the Naranjo et al. (1981) algorithm ranging from definite – probable – possible – doubtful [57]. The association between each medication and described side effect (nightmares or alterations in dreaming) in clinical trial reports is rated from significant ($p < .01$), to probable (reported by >1% of population relative to controls), to possible (less than 1% difference compared to control orin studies without controls) to doubtful (minimal evidence for side-effect/drug association). This study does not include data concerning drugs for which no effects on dreaming or nightmares are reported, because of concerns as to the significance of negative reports. Older (before 1990) clinical trial data is not included since clinical trial reports for agents known to induce nightmares per case reports (examples: tricylic antidepressants, amphetamines, and benzodiazepines often did not include reports of nightmares or disturbed dreaming. [This assessment approach is used for drugs in Tables 2A and 2B.]

literature describing drug effects on dreaming consists primarily of reports of nightmares as a side effect of medication or as a symptom of medication withdrawal (Table 2A).

Data from human clinical trials and case reports (Table 2) indicates that reports of altered dreaming and nightmares are consistently associated with agents exerting pharmacological effects on dopamine, serotonin, and norepinephrine. Beta-blockers are the agents most likely to result in patient complaints of nightmares. The strongest clinical evidence found in this meta-analysis for the association of a drug with nightmare induction is for the SSRI paroxetine (Pagel & Helfter 2003). Most agents affecting dopaminergic neuroreceptors have been reported in clinical trials to induce nightmares in some patients. Medications altering these neurotransmitter systems are likely to induce reports of nightmares and disordered dreaming for patients taking those medications. These neurotransmitters may function in a reciprocal interaction involving a wide spectrum of neurotransmitters interacting in an intricate modulation of the cardinal sleep stages – REM and non-REM sleep (Pace-Schott 2003).

Clinical trial and case report data are less clear in their support

for the association of GABA and acetylcholine receptors with dreaming, and nightmare alteration with the reported nightmare/drug association rated as possible (rather than probable or significant) for the majority of drugs evaluated. The finding that different types of drugs known to affect the GABA receptor (agonists, modulators, and reuptake inhibitors) can result in patient complaints of nightmares and abnormal dreaming is suggestive that GABA may be a modulator of the neuronal populations involved in dreaming as proposed by Pace-Schott (2003) (Mallick 2001, Xi & Morales 1999). Agents that increase acetylcholine levels such as the acetylcholineserase inhibitors routinely utilized in patients with Alzheimer's disease would be expected based on animal models to increase REM sleep. The side effect of nightmares and/or altered dreaming secondary to the use of these agents is rarely reported (only 3 of 747 patients using donepizil in clinical trials reported changes in dreaming) (Pagel; in press).

Agents that alter an individual's conscious relationship to the external environment may alter reported dream and nightmare occurrence. Induction anesthetics induce an electrophysiological state that is not clearly sleep, as well as patient complaints of abnormal dreaming. Some of the agents reported to cause altered dreaming are: profolol, the barbituate thiopental, ketamine, and the opiate tramadol (Krissel et al. 1994; Marsh et al. 1992; Oxorn et al. 1997). The CNS side effects of daytime somnolence and/or insomnia may be an indicator for medications likely to induce disordered dreaming and nightmares (Table 2B).

Infectious diseases are sometimes associated with the complaint of nightmares. Sleep loss affects host defense and cellular immune function (Benca & Quintas 1997; Hall et al. 1998; Moldofsky 1995). These studies suggest that a clear, but currently poorly defined relationship may exist between host defense and infectious disease, and sleep/dreaming. Several of the agents reported in case reports and clinical studies to induce nightmares (fleroxacin, erythromycin, ciprofoxin, and ganciclovir) may induce nightmares by affecting sleep-related immunological response to infectious disease. This meta-analysis indicates that the neuropharmachological agents reported to induce nightmares in human studies differ from the agents modulating REM sleep in animal models. Drugs affecting acetylcholine, the primary modulator of REM sleep in animal models, are not reported to induce nightmares in clinical trials. Pharmachological agents affecting the negative neuro-modulators of REM sleep – norepinephrine and serotonin – are those most likely to induce nightmares in human subjects. Other medications appear to alter dreaming by affecting an individual's conscious relationship to the environment or to host defense and immunology.

Conclusion. The neurobiology of the human CNS is the most complex one yet addressed by theorists and researchers. Activation-synthesis as a theory of sleep and dreaming has been tremendously useful to the field, and its basic tenet – that the physiological events occurring in the brain during dreaming are associated with dreaming – remains absolutely valid. Theories are useful if they can be adapted or altered to achieve consistency with new data. Theories can be harmful if used to misrepresent the extent of knowledge or to limit the breath of study.

In this commentary, two meta-analyses of pharmacological research have been presented. The data on electrophysiological psychoactive drug EEG effects demonstrates that psychoactive drugs have consistent effects on EEG and sleep outside of their effects on REM sleep. The data on drug-induced nightmares demonstrates a complex system characterized poorly by neurotransmitter models limited to the modulation of one or several neurotransmitters at discrete CNS sites. This is experimental data obtained from humans in which the cognitive process of dreaming is addressed, rather than the associated state of REM sleep.

These data suggest that the neurobiology data terrain outside activation-synthesis may include sleep and dream electrophysiology, cognitive reports of dreaming, effects of alterations in consciousness on dreaming, immunology and host defense, and clinical therapies for sleep disorders.[1] Dream neurobiology is a field in which experimental data can be addressed inductively, a field open to new theories developed to explain data lying outside the structured paradigms of current theory. A philosophy that remains cogent for the CNS is that new research almost always shows this system to be more complex than previously thought.

Editors' Note: There is no Authors' Response to this commentary.

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Commentary on **Esther Thelen, Gregor Schöner, Christian Scheier, & Linda B. Smith (2001). The dynamics of embodiment: A field theory of infant perseverative reaching. BBS 24(1):1–86.**

Abstract of the original article: The overall goal of this target article is to demonstrate a mechanism for an embodied cognition. The particular vehicle is a much-studied, but still widely debated phenomenon seen in 7–12-month-old-infants. In Piaget's classic "A-not-B error," infants who have successfully uncovered a toy at location "A" continue to reach to that location even after they watch the toy hidden in a nearby location "B." Here, we question the traditional explanations of the error as an indicator of infants' concepts of objects or other static mental structures. Instead, we demonstrate that the A-not-B error and its previously puzzling contextual variations can be understood by the coupled dynamics of the ordinary processes of goal-directed actions: looking, planning, reaching, and remembering. We offer a formal dynamic theory and model based on cognitive embodiment that both simulates the known A-not-B effects and offers novel predictions that match new experimental results. The demonstration supports an embodied view by casting the mental events involved in perception, planning, deciding, and remembering in the same analogic dynamic language as that used to describe bodily movement, so that they may be continuously meshed. We maintain that this mesh is a pre-eminently cognitive act of "knowing" not only in infancy but also in everyday activities throughout the life span.

Is the concept of object still a suitable notion?

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Abstract: The model and framework presented in the target article by Thelen et al. is an interesting effort that is able to account for the contextual variability in the A-not-B performance of 7–12-month-old infants. In the process of developing their framework, the authors discounted the concept of object as a useful notion in discussions of A-not-B performance. For Piaget and other developmentalists, the main evidence for the acquisition of the concept of object was the disappearance of A-not-B errors after age 12 months. However, the Thelen et al. model makes predictions of A-not-B outcomes over a much shorter, trial-to-trial time scale. Given the mismatch in the time scales over which analyses in the two approaches have been based, we wonder if the challenge to the concept of object has been misplaced.

Thelen et al. (2001) used the inconsistency of the results in the A-not-B paradigm, and the success of their model, to bring into question the utility of Piaget's (1954) *concept of object* as the predominant explanation for A-not-B performance. Nevertheless, we wonder whether the success of their model justifies rejection of the concept of object as an explanation for A-not-B performance. In addition to exploring this issue, we point out that although Piaget did not present a quantitative account of his empirical work, his framework can, at least in part, be considered dynamical. In other words, in many ways Piaget's outlook as a scientist of the developing mind may not differ as much from the viewpoint advocated by Thelen et al. as the authors portrayed.

What is the utility of the "concept of object"? The "concept of object" is generally defined as an awareness that an object continues to exist even when the actor no longer interacts with it, and when sensory input regarding the object has been removed. The A-not-B experimental procedure has been the predominant method used to study the concept of object and to track its changes with age. Briefly, the procedure requires participants to reach for a hidden object at a location A, and then to make a subsequent reach to an object hidden at a cued location B. Continued reaching at location A, committing the A-not-B error, has been taken as evidence of incomplete acquisition of the concept of object, whereas the acquisition of the concept of object has been reflected by the generation of cued reaches to location B. Thus, the level of A-not-B error provides a simple, parsimonious operationalization of the concept of object. Developmentalists, Piaget in particular, were interested in examining performance on this task because it reflected development toward adaptive, adult capabilities. In particular, a transition from the presence of A-not-B error to its disappearance became a landmark for identifying a critical transition in development. Namely, this transition marked the end of Piaget's stage IV to the beginning of stage V, during his sensorimotor period of development.

Between the ages of 7 and 12 months (Piaget's stage IV), variation in task parameters are related to variation in the degree of A-not-B error outcomes, but clearly there is evidence of the A-not-B error. Moreover, the A-not-B error disappears after about age 12 months (Piaget's stage V). Therefore, when considered over the course of development, and taking the literature as a whole, the basic effect and developmental trend *are* reproducible. Early in the Thelen et al. target article (sect. 2), the authors were apparently in agreement with this statement. However, in focusing on the between-experiment variability in A-not-B outcomes during the ages 7–12 months period, the authors (viz., their model) seem to have lost sight of the fact that the trend continues beyond that age, and in fact changes after about age 12 months. It is the appearance of this bifurcation in the developmental function, and not the pre-12-months-of-age between-experiment variability, that would seem to be the main evidence supporting the appearance of the concept of object. Therefore, if the concept of object is to be challenged from an analysis of the extant empirical data, then the authors have based their challenge on what seems to be a wrong view of the data.

In particular, the Thelen et al. model (e.g., Figs. 15–20 in the target article) accounts for how changes in task parameters and internal processes (e.g., memory) act on a short-term, trial-to-trial basis in the prediction of A-not-B performance. This time scale of analysis is much briefer than the time scale with which Piaget and other developmentalists were concerned. Given such a difference, we wonder if the authors were actually interested in addressing developmental processes regarding A-not-B performance. Perhaps Thelen et al. were more interested in adapting an already ex-

isting dynamical model (Schöner et al. 1997) to account for the processes related to the generation of motor output observed under the A-not-B paradigm.

This is not to say that the authors did not achieve success in the problem they considered: accounting for contextual variations in A-not-B outcomes during the age 7–12 months period. Indeed, they provided a quantitative framing of this problem that (1) reorganizes the range of A-not-B results; (2) holds the promise of making predictions of future experimental outcomes; and (3) is cast in a formal language where both the dynamics of mental life and observable behavior can be viewed in the same quantitative framework. The first and second achievements are tenets of good science, in general. The third is an achievement of a science where investigators consider that their subjects have minds, as well as bodies.

Is the concept of object a static construct? The second point of our commentary on Thelen et al.'s portrayal of the Piagetian concept of object is that it appears to have been misrepresented as a static mental structure. It is more reasonable to think of the object concept as representing a global variable that is subject to modulation by a number of sources of input. In Piaget's framework the development of object concept evolves over time through the integration and modification (e.g., assimilation, accommodation) of information coming from the interaction of the infant with the environment. This may also include information from multiple sources of sensory input from the environment, information that is a consequence of the infant's actions, and information available from past experience. In other words, the concept of object arises from the input of multiple, dynamically interacting sources of information. Therefore, we can conclude that Piaget's thinking on the concept of object, if not his general theoretical stance (e.g., see Fig. 11 in Varela 1989), was closer in spirit to the Thelen et al. framework than Thelen et al. have led readers to believe. Although Thelen et al. expressed their point of view with the help of sophisticated quantitative tools, when the Piaget and Thelen et al. approaches are considered together under a broadened view, we can see that the two approaches have common features.

Conclusion. Although there is no question that the Thelen et al. model works to account for the contextual variations in the data observed during ages 7 to12 months, these authors addressed phenomena that occur over a shorter-time scale than that in which Piaget and other developmentalists had interest. The model presented in the target article deals with shorter-term, trial-by-trial predictions. On the other hand, the concept of object is most clearly evidenced by the transition seen when a longer course of development is considered. As a result of the mismatch between the time scale of analysis that Thelen et al. were concerned with, and the time scale that was relevant for an understanding of object concept development, the challenge made by Thelen et al. to the concept of object seems misplaced. We conclude that when the concept of object is considered over the relevant developmental time scale, it, first, retains its usefulness for discussions on development, and, second, is not a static construct but instead evolves through the input from various dynamically interacting information sources.

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There may not be an A-not-B error

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Abstract: In the A-not-B situation children reach toward location A when the object is at location B. Researchers interpret this as an error. I question this interpretation. Reaches are inaccurate only if the intention actually is to obtain the hidden object. If this is not the goal, then reaching for A may be accurate and there may be no error to be explained.

In the A-not-B situation the youngest children do not reach for hidden objects. Between 7 and 12 months of age, children often reach toward A when the object is hidden at B. Finally, older children reach for the hidden object, regardless of its location. Piaget interpreted the first two actions as errors, and the third action as correct. As reviewed by Thelen et al. (2001, sect. 2.1), subsequent accounts of the A-not-B situation also interpret the child's behavior in the second stage as an error. Piaget did not test this interpretation, and his successors do not appear to have done so either. But reaching toward A (when the object is at B) is incorrect only if the child actually intends to reach toward B.

It is risky to assume that we know the goals of prelinguistic children. It is particularly risky to assume that a 9-month-old has the same goals as the experimenter. This may be an example of the psychologist's fallacy (James 1890/1950): the inappropriate assumption that the experimenter's view of the situation is the same as that of the subject. Consider mealtime. At any age, some food may fall to the floor. Adults always consider this to be an error, but children may not. When the child first learns to put food in his or her mouth the skill is poorly developed and food is dropped due to lack of skill; an error. In the mature child (as in the adult), food reaches the floor only by accident. However, in an intermediate stage (at roughly two years of age) the child sometimes intends that the food land on the floor. Adults consider this to be an error but the child regards it as a resounding success. To be sure, there is a developmental change in the child's ability to move food by hand. However, there is also a developmental change in the child's goals (these goal changes are reflected in the fact that we sometimes refer to the food as being dropped, and at other times as being thrown). Researchers appear not to have considered the possibility that a sequential change of goals may occur in the A-not-B situation.

Thelen et al. have sometimes characterized the pattern of reaching as "perseverative," rather than as an error. However, this does not resolve the issue. The model offered by Thelen et al. predicts a divergence between intention (goal) and action (outcome). For all intents and purposes, this is an error, as is shown by the fact that in section 2.1 Thelen et al. have contrasted "perseverative reaching" with "accurate performance."

Perhaps over the course of development children do not make mistakes in the A-not-B situation. Instead, developmental changes in reaching might be produced by developmental changes in the child's goals. Six-month-old children may not want to retrieve the hidden object; maybe they are interested only in having things that they can see. If so, then not reaching is correct at this stage. Between ages 7 and 12 months, perhaps children do not want to retrieve the hidden object at B but do want to reach to A (I don't know why a child might want to do this, but that does not invalidate the point: As any parent knows, a child's goals often differ from the parent's goals, and it is often difficult to understand why children want what they want). If so, then reaching to A would be correct, or accurate, and the model of Thelen et al. would not apply. It is certainly the case that children's behavioral goals change as they develop, and these different goals lead to reliable differences in behavior. The question, then, is whether there might be a regular progression in children's goals that could produce the developmental pattern of reaching reported by Piaget.

Students of the A-not-B situation (including Thelen et al.) offer

no evidence that reaching is incorrect in the ordinary sense of being incompatible with the child's goals or intentions. Evidence might be sought in children's affect, and in their actions. For affect we might determine whether children act as though reaches to A are unsuccessful. Do they appear to be unhappy; do they fuss, or cry? Reactions of this type might be expected if children were attempting to recover the hidden object, especially given the frustration that could build up over a series of failures. If children are not disappointed, perhaps it is because they have not made any mistakes. Separately, behavioral evidence might be sought by examining reach trajectories. A child who begins to reach toward the wrong location may notice this and correct the trajectory online. Sasaki et al. (1995) have documented this with adults in ordinary situations, such as making coffee. A person who wants to add sugar may, by mistake, begin reaching toward the milk, then correct the error in mid-reach so as to arrive at the desired target. Similar corrections occur in infants (Sasaki et al. 1998). If the child intends to reach B, then on some reaches we might expect to see trajectory changes; an initial movement toward A followed by a correction and final arrival at B. A-not-B researchers do not appear to have examined the possibility of mid-course corrections.

Rather than creating models to explain assumed errors in the A-not-B situation, researchers might determine whether the reaching actually diverges from the child's intentions – that is, whether there is any error that needs to be explained. If reaching is accurate, then the phenomenon that needs to be explained is not the child's struggle to generate actions that match his or her intentions but, rather, changes in the child's goals or intentions as a function of development. In this latter case the model of Thelen et al., whatever its virtues, could not be correct because it would be attempting to explain the wrong thing.

Editors' Note: There is no Authors' Response to this commentary.

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Commentary on **Daniel S. Ruchkin, Jordan Grafman, Katherine Cameron, & Rita S. Berndt (2003). Working memory retention systems: A state of activated long-term memory. BBS 26(6):709–777.**

Abstract of the original article: High temporal resolution event-related potential and electroencephalographic coherence studies of the neural substrate of short-term storage in working memory indicate that the sustained coactivation of both prefrontal cortex and the posterior cortical systems that participate in the initial perception and comprehension of the retained information are involved in its storage. These studies further show that short-term storage mechanisms involve an increase in neural synchrony between prefrontal cortex and posterior cortex and the enhanced activation of long-term memory representations of material held in short-term memory. This activation begins during the encoding/comprehension phase and evidently is prolonged into the retention phase by attentional drive from prefrontal cortex control systems. A parsimonious interpretation of these findings is that the long-term memory systems associated with the posterior cortical processors provide the necessary representational basis for working memory, with the property of short-term memory decay being primarily due to the posterior system. In this view, there is no reason to posit specialized neural systems whose functions are limited to those of short-term memory buffers. Prefrontal cortex provides the attentional pointer system for maintaining activation in the appropriate posterior processing systems. Short-term memory capacity and phenomena such as displacement of information in short-term memory are determined by limitations in the number of pointers that can be sustained by the prefrontal controls system.

Hidden operators of mental attention applying on LTM give the illusion of a separate working memory

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Abstract: The authors' results support a functionalist conception of working memory: a manifold repertoire of schemes/schemas (long-term memory) and a small set of general-purpose "hidden operators." Using some of these operators I define mental (i.e., endogenous) attention. Then, analyzing two of the authors' unexplained important findings, I illustrate the mental-attention model's explanatory power. Multivariate methodology that varies developmental, task differences, and individual differences is recommended.

Ruchkin, Grafman, Cameron, & Berndt (Ruchkin et al. 2003) have made a very important contribution by showing that the activity and coherence dynamics in their results contradict conceptions of separate working memory (WM). Their data support a radically distinct, *deconstructed, decentralized, and functionalist* (what the authors call, on p. 711 of the target article, sect. 1.4, an "activation-proceduralist") conception of WM and cortical information processing. "Decentralized" describes an organization in which information does not move from one memory store to another but is mediated/carried by distinct collections of neurons, often distributed over the brain, that are cofunctional (vis-à-vis certain activities) and coactivated in some tasks. These semantic-pragmatically functional collections I call *schemes* or *schemas* (Pascual-Leone 1995; Pascual-Leone & Johnson 1991; 2004; Pascual-Leone et al. 2000). Schemes can be distinctly demarcated as causal determinants that *overdetermine* manifest performance, facilitating process and task analysis. Schemes overdetermine per-

formance because they are *self-propelling.* They tend to apply to the situation (to fire as a collection of neurons) under minimal conditions (cues, related firing) of activation. Ruchkin et al. speak of "brain systems," which in my view are often organized collections of schemes. Long-term memory (LTM) is the only memory organization, as asserted by Ruchkin et al., but LTM can be best explicated as a manifold *repertoire* of schemes sorted in kinds: *executive* schemes (prefrontal), *operative* schemes (frontal), *figurative/declarative* (i.e., perceptual or representational, or linguistic) schemes (occipital, temporal, or parietal), *coordinated or automatized operative/motor* schemes (basal ganglia and cerebellum), *affective/emotion* schemes (broadly defined limbic system), *analytical* schemes *produced with mental effort* (initially left hemisphere), *global or automatized* schemes (right hemisphere), and so forth.

But with this theory of schemes one cannot explain *general* organismic constraints (i.e., unspecific, applying across kinds – modes and modalities – of schemes), such as the so-called central WM capacity limits or central inhibitory mechanisms, or central resolution of schemes' *competition* in the network (caused by their self-propelling nature), or the emergence of truly novel performances via unplanned *dynamic syntheses.* Ruchkin et al. intimate (target article, sect. 5, p. 727) that the prefrontal lobe takes care of all that by controlling processes elsewhere in the cortex (posterior areas), and that its controlling power is limited (the number of attentional "pointers" is restricted). A better solution to these mysterious powers of the brain is to postulate that, in addition to a repertoire of schemes, the brain has a small set of general-purpose functional entities, called in my theory *"hidden*/silent *operators"* (Pascual-Leone 1987; 1995; 1996; 2000a; Pascual-Leone & Johnson 1991; in press; Pascual-Leone et al. 2000). I call them "operators" because they are functional mechanisms of brain "hardware" defined as *molar procedures whose computational details are unspecified,* which apply on (constrain) schemes to change or produce new schemes, or to synthesize truly novel performances. These operators are described as hidden because they lack the substantive content referents that schemes have (i.e., perceptual, motor, representational, etc.); instead, operators express purely relational multivariate constraints – invariant "anomalies" (exhibited under specific empirical circumstances) that a pure theory of schemes cannot explain.

As an example, *mental* (i.e., endogenous) *attention,* a brain system essential for high cognition, results from dynamic interactions among four different sorts of processes, three of which are hidden operators: an attentional scheme-activation resource or *M*-operator, an attentional interrupt ("central" inhibition mechanism) or *I*-operator, a neoGestaltist (i.e., Minimum-Principle, S-R compatibility) closure-producing mechanism or *F*-operator, and the currently-dominant set *E* of attentional-control executive schemes (*E* allocates *M* or *I* to relevant/irrelevant schemes). *M, I,* and *E* are prefrontal functions. I believe *F* results from lateral-inhibition processes in the cortex. This mental-attentional system appears in performance as severely limited parallel-processing constraints, particularly within misleading situations. These constraints become relaxed (yielding more *mental* processing power) with chronological age up to adolescence (Case 1998; Pascual-Leone 1970; 1987; 2000b; Pascual-Leone & Baillargeon 1994; Johnson et al. 1989). I mention only two other hidden operators that are relevant here: content/substantive learning (i.e., *C* operator) and logical/structural/relational learning or *L*-operator (Case 1998; Pascual-Leone 1995, 1996; Pascual-Leone & Goodman 1979).

The construct of hidden operators makes it easier to interpret unexplained findings noted by Ruchkin et al. I give two examples that are interpretable using mental-attentional model $<E, M, I, F>$. The authors do not define what mental attentional processes are, although in their Conclusion they assert that the focus of attention is limited in its "number of pointers." The developmental construct *M*-operator (intuitively, mental "energy" that can be allocated to task-relevant schemes), when considered within the developmental model of mental attention, is better grounded than the authors' congenial metaphor of a "number of pointers." This model serves to interpret a striking finding of Haarmann and Cameron (in press, p. 726). Using coherence analysis, Haarmann and Cameron discovered that retention of sentences with unrelated nouns exhibits a coherence pattern in the 10–14 Hz band, even though during initial processing of the sentences the coherence pattern was found only in the 4–6 Hz band. This can be understood with the mental-attention model by making a sensible assumption: that the band where coherence patterns appear becomes higher in frequency as individuals' mental attention focuses repeatedly on the same schemes, producing a deeper processing. Then we understand the results as expressing a predictable processual difference between the time period when *M*-capacity is being mobilized and applied exploratively to various schemes elicited by the task, versus the period when *M*-capacity is being persistently applied to essential schemes that must be kept strongly activated. Indeed, unpublished experimental research from our lab some years ago found that the "*M*-time psychological moment" (i.e., the time taken by a full allocation of *M*-capacity) is about 100 msec (see, e.g., McCoy 1972). Thus interpreted, the finding supports Ruchkin et al.'s conception of WM. Notice that another finding of Haarmann and Cameron, that coherences in the 10–14 Hz band are generally larger following sentences with semantically unrelated nouns (versus related nouns), is consistent with this interpretation, for sentences using related nouns benefit from previous experience (*L* learning), and so their retention necessitates less *M*-capacity than do sentences with unrelated nouns.

A second example is Ruchkin et al.'s interpretation of results that suggest the existence of central inhibitory processes, although they do not address the issue directly. Discussing brain activation during comprehension and retention of sentences (pp. 724–25), Ruchkin et al. obtain (referring to the lingual gyrus) positive polarity activity in the event-related potential (ERP), and interpret this as expressing operation of a visual-verbal buffer that supports reading. But this idea of a "specialized visual-verbal buffer" is theoretically regressive, for it returns to a metatheory of multiple memories against which the authors have eloquently argued. A different functionalist interpretation is available if we accept the mental attention model with an inhibitory interrupt utility (*I*-operator). Indeed the progressive shifting/decentration from visual to semantic codes necessitates, *due to the paradigm strictures,* active inhibition of visual-phrase schemes (codes) to keep them from interfering with semantic-phrase schemes already activated by reading, which can no longer be boosted by *M*-capacity (because of their excessive number). As the authors may be intimating, it is sensible to think that this semantic shifting takes place in the lingual gyrus. Consistent with the functionalist interpretation that eliminates the need for a buffer is the finding that polarity of the ERP activity in question is positive. Indeed, positive ERP activity seems to index inhibition processes (Bakay et al. 1998; Goode et al. 2002; Halgren 1993; Rovkstroh et al. 1997). The authors' data predominantly exhibit negative ERP polarities, indexing activation processes, because their situations/tasks are basically facilitating. A situation is said to be *facilitating* when it elicits from the subject only schemes relevant for the task; and it is *misleading* when it elicits schemes inconvenient or contradictory with the task (minor instances of that are visual-phrase schemes here). Only misleading situations demand mental attentional interruption of unsuitable schemes; this is why misleading tasks exhibit much more positive ERP waves.

To pursue further the experimental deconstruction of separate WM (or multiple-memory) theories, Ruchkin et al. might find it useful to adopt a functionalist hidden-operators experimental approach. To this end a multivariate perspective becomes important because in this methodology three different but complementary dimensions of variation in the experimental design are necessary. The three dimensions are: (1) Developmental (chronological age, starting with young children), which is needed because *M*-capacity increases in power with chronological age in normal subjects.

(2) Task Differences, in particular contrasting facilitating versus misleading WM situations, necessary because attentional interruption is needed only in the latter (Pascual-Leone 1987; 1995; 1996; 2000a; 2000b; Pascual-Leone & Johnson 2004) and necessary also because only in misleading situations does *M*-capacity alone determine WM capacity; in facilitating situations other operators (e.g., *L* learning, perceptual *F* saliencies) might also increase WM capacity. (3) Stylistic subject differences, like those between Field-Independent versus Field-Dependent subjects (Goode et al. 2002; Pascual-Leone 1970; Pascual-Leone et al. 2000) could affect WM characteristics, because FD subjects tend to mobilize less *M*-capacity and instead rely on the learning and perceptual saliency hidden operators.

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