of this, it would be premature to rename combined subtype ADHD as "reinforcement/extinction disorder." It is crucial to recognize that multiple pathways may not simply represent alternative routes into ADHD. Rather, it may be the norm for most children to have contributions from several, but not necessarily all, pathways in varying degrees.

Selectionism: Complex outcomes from simple processes

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Abstract: Both the target article and the precommentary demonstrate that relatively simple biobehavioral processes have the cumulative effect of fostering behavioral outcomes characteristic of attention-deficit/hyperactivity disorder (ADHD). As such, the articles illustrate a central theme of Darwinian thinking – basic processes acting over time can produce complex and diverse outcomes. In this commentary, we indicate that tracing the action of processes over time can be facilitated by quantitative methods such as artificial neural networks.

The target article by Sagvolden et al. and the precommentary by Catania illustrate a common general theme: Basic neural and behavioral processes can produce diverse and complex outcomes when they act over time. This theme exemplifies a central insight of Darwinian thinking: namely, complexity can result from repeated action of relatively simple processes (Campbell 1974; Donahoe 2003). Sagvolden et al. describe the cumulative effects of a dysfunction in fronto-striatal circuits involving the neuromodulator dopamine and its resulting impact on the ability of organisms to tolerate temporal delays between behavior and reinforcers. Catania explores further the cumulative effects of differences in the ability to tolerate delay of reinforcement and reveals additional implications for the emergence of complex behavior. Together, these authors provide an interpretation of how a seemingly minor dysfunction can lead to many of the complex characteristics of attention-deficit/hyperactivity disorder (ADHD), including a lack of sustained attention, hyperactivity, and impulsiveness. The target article and precommentary also jointly illustrate a distinction articulated by B. F. Skinner between experimental analysis, whereby basic processes are identified through carefully controlled laboratory observations, and scientific interpretation, whereby the implications of basic processes are explored for phenomena that cannot be studied under circumstances that meet the demands of experimental analysis (Skinner 1957). Sagvolden et al. draw upon experimental analyses at the cellular level of the processes that affect synaptic efficacies, while Catania makes use of experimental analyses at the behavioral level of processes that affect delay of reinforcement. Using these processes, the authors provide plausible interpretations of their effects on ADHD.

Scientific interpretation differs from mere speculation: Interpretation makes use solely of processes and structures that have been identified in prior experimental analyses. Interpretation is the means by which explanation occurs in all historical sciences – evolutionary biology and cosmology as well as behavioral neuroscience. If the cumulative effects of basic processes are sufficient to account for a complex phenomenon, then the interpretation is tentatively accepted as the explanation of that phenomenon. As an example from physical science, if the cumulative effect of gravity on a swirling cloud of primordial dust particles can account for the formation of planetary systems, then the interpretation is accepted as the explanation of planetary formation even though nascent planetary systems have not been subjected to experimental analysis – and likely never will be.

In early Darwinian interpretations of evolution, ordinary language was used to explore the implications of the process of natural selection. However, natural selection provided an interpretation of evolution that even other scientists did not find compelling until ordinary-language interpretations were supplemented by the more formal methods of population genetics (Fisher 1930; Haldane 1931/1966; Wright 1939) and, later, computer simulations (e.g., Maynard-Smith 1982). A similar transition in the nature of scientific interpretation is occurring in biobehavioral research with the advent of such techniques as artificial neural networks. Artificial neural networks that qualify as scientific interpretations must be informed and constrained by experimental analyses of neuroscience and behavior. In this respect, such networks differ fundamentally from superficially similar methods in normative psychology. In normative psychology, the characteristics of neural networks are inferred from the behavioral observations that they seek to explain and are not informed by independent experimental analyses. Behavior analysis is uniquely positioned to interface with neuroscience because, if behavioral processes are to be supplemented by infrabehavioral processes, those processes must be the fruits of independent experimental analysis at the neural level and not mere inferences from behavior (Donahoe 2002).

One approach to artificial neural networks in behavioral neuroscience is selection networks (Donahoe 1997; Donahoe & Palmer 1994; Donahoe et al. 1993). Selection networks consist of two inter-related neural subsystems: a motor subsystem that simulates the effects of the neuromodulator dopamine on synaptic efficacies of neurons in the frontal lobes and a sensory subsystem that simulates the effects of the hippocampus on synaptic efficacies of neurons in the parietal and temporal lobes. Only the motor subsystem is considered in the present interpretation of delay of reinforcement.

Simulated increases in synaptic efficacies in the motor subsystem occur when pre- and postsynaptic units are recently coactive and their coactivity is accompanied by activation of units in the simulated ventral tegmental area (VTA). Coactivity of units in the motor subsystem results from the action of glutamate on postsy-

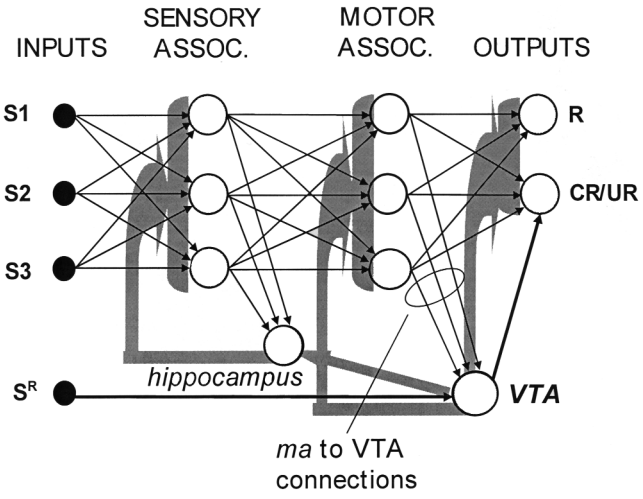

Figure 1 (Donahoe & Burgos). A minimal neural architecture of a selection network. Environmental events stimulate sensory units that probabilistically activate units in the sensory-association subsystem. These units, in turn, probabilistically activate motor-association and output units in the motor subsystem. Activated output units simulate the emission of the operant response (*R*) and the elicitation of the reinforcer-evoked (i.e., unconditioned) response (*UR*). The circled MA-to-VTA connections simulate the fronto-striatal connections that play a central role in the account of delay of reinforcement provided by Sagvolden et al.

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naptic AMPA and NMDA receptors. The pathways that simulate the release of dopamine from neurons in the VTA are represented by the grey regions in the motor subnetwork of Figure 1. The motor subnetwork includes motor-association (ma) units and output units. VTA units are activated by environmental events that stimulate the input unit for the reinforcing stimulus (S^R). The neural processes and structures simulated by selection networks are informed by the research findings of the sort summarized by Sagvolden et al. (see Donahoe 1997; Donahoe & Palmer 1994; Frey 1997). Previous simulation research has shown that selection networks can simulate important aspects of a wide range of behavioral phenomena including acquisition, extinction, discrimination, timing, and revaluation (Donahoe & Burgos 1999; 2000). Of interest here are the effects of the pathways from ma units to VTA units, for these pathways are critical to conditioning with delay of reinforcement.

As Catania noted, delay of reinforcement is inevitable at the cellular level. The intracellular events that are essential for changes in synaptic efficacy endure for only a few hundred milliseconds within the dendritic compartments. This interval is much less than the irreducible delay between the occurrence of an operant (e.g., lever pressing) and the delivery of a reinforcer (e.g., a food pellet). In recognition of this constraint, before operant conditioning is instituted in the laboratory some distinctive stimuli (e.g., a "click" produced by the operation of a pellet feeder) is repeatedly paired with a reinforcer. Then, when operant conditioning begins, the distinctive stimulus is presented immediately after the occurrence of the operant. The neural solution to the problem of delay of reinforcement is accomplished in selection networks by means of connections from ma units to VTA units. These simulate the fronto-striatal pathways identified by Sagvolden et al. as critical for interpreting ADHD. As conditioning proceeds, VTA units become activated not only by the reinforcing stimulus (S^R) but also by ma units that are activated by the effects of environmental stimuli on input units (e.g., *S*1).

Figure 2 displays the results of the simulation of operant conditioning with two different delays of reinforcement – a short delay of three time-steps and a longer delay of six time-steps. During each time-step, the simulated synaptic efficacies were changed according to the learning rule. Changes were a function of the magnitudes of the activations of the pre- and postsynaptic units and of the VTA unit that simulated dopaminergic release during that time-step (see Donahoe et al. 1993 for details.) The input unit for the reinforcer (S^R) was stimulated at the final time-step when the activation level of the operant output unit (R) exceeded zero. The upper panels show simulations when the ma-to-VTA connections were intact. Under these conditions, the VTA could exert its reinforcing effect on synapses in the motor subnetwork when the VTA was activated either by the environmental reinforcer (S^R) or by MA units (via the circled connections) throughout all time-steps. As can be seen, acquisition occurred with both delays of reinforcement, albeit somewhat earlier and more rapidly with the shorter delay. The lower panels show acquisition with the same two delays but with no connections from ma to VTA units; that is, without the conditioned reinforcement supplied by the activity of ma units. With the short delay of three time-steps, acquisition continued to occur although somewhat more slowly and more variably. In contrast, acquisition failed to occur with the longer delay of six time-steps when the ma-to-VTA connections were absent. (Other simulations with as many as 3,000 training trials did not respond above the levels shown in Fig. 2.) Thus, the simulations provide a neural-network interpretation that supports the critical role assigned to fronto-striatal pathways by Sagvolden et al. Neural-network simulations of behavioral phenomena are at an early stage of development and are incomplete in many respects. Nevertheless, they already show promise of providing compelling, biobehaviorally informed interpretations of complex behavior.

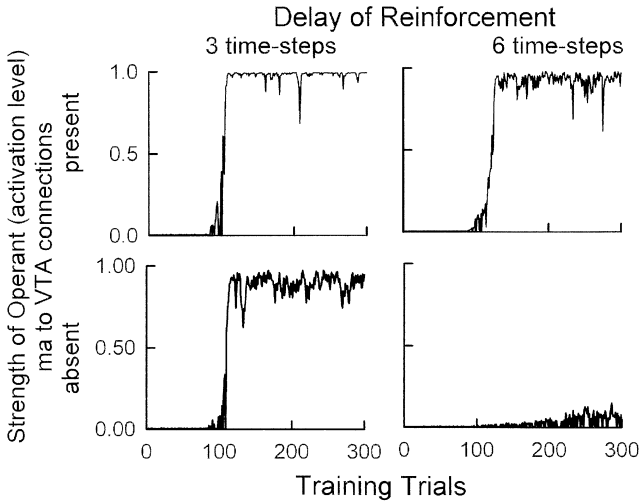

Figure 2 (Donahoe & Burgos). Simulation of the effects of two delays of reinforcement – three time-steps and six time-steps – on the acquisition of an operant response. The activation levels of the *R* output unit on the penultimate time-step are plotted. Conditioning took place when connections from ma to VTA units were present (upper panels) and when they were absent (lower panels). Each acquisition curve is the average of five independent networks with the architecture shown in Figure 1.

A comprehensive and developmental theory of ADHD is tantalizing, but premature

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Abstract: In this commentary, I argue that the theory presented by Sagvolden et al. can be much stronger if its scope is limited, if its developmental aspects are refined, if it can be made to generate testable predictions, and if it can be supported with more data from humans.

The theory presented by Sagvolden et al., based in part on work by Catania, merits attention for several reasons. The field of attention-deficit/hyperactivity disorder (ADHD) sorely needs solid theories from which testable predictions can be derived. The theory promises to be an overarching developmental theory. It tackles an important problem in ADHD. Finally, Sagvolden et al. discourage the use of behavioral intervention techniques (defined as frequent and immediate reinforcement) as impractical and advocate the use of medications for both children and their parents.

Although tantalizing, an overarching and developmental theory of ADHD is premature. In this commentary, I focus on several weaknesses of Sagvolden et al.'s theory, but end with the hope that it will generate more research.

The theory is too comprehensive. The theory is based on the finding that the dopamine (DA) system is implicated in ADHD, and on the premises that DA dysfunction in certain fronto-striatal circuits causes ADHD and that the most important behavioral functions of these circuits are reinforcement of novel behavior and extinction of previously reinforced behavior. Sagvolden et al. argue that these two behavioral processes "cause" ADHD and can explain all the symptoms of ADHD, as well as individual and developmental differences in the nature and severity of symptoms, and can be used to link molecular to societal levels of explanation. However, children diagnosed with ADHD are very heterogeneous, and it is easy to bring up examples of findings that are not easily explained by the theory (patterns of comorbidity across the

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