Dopamine and the Mechanisms of Cognition: Part I. A Neural Network Model Predicting Dopamine Effects on Selective Attention

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Background: Dopamine affects neural information processing, cognition, and behavior; however, the mechanisms through which these three levels of function are affected have remained unspecified. We present a parallel-distributed processing model of dopamine effects on neural ensembles that accounts for effects on human performance in a selective attention task.

Methods: Task performance is simulated using principles and mechanisms that capture salient aspects of information processing in neural ensembles. Dopamine effects are simulated as a change in gain of neural assemblies in the area of release.

Results: The model leads to different predictions as a function of the hypothesized location of dopamine effects. Motor system effects are simulated as a change in gain over the response layer of the model. This induces speeding of reaction times but an impairment of accuracy. Cognitive attentional effects are simulated as a change in gain over the attention layer. This induces a speeding of reaction times and an improvement of accuracy, especially at very fast reaction times and when processing of the stimulus requires selective attention.

Conclusions: A computer simulation using widely accepted principles of processing in neural ensembles can account for reaction time distributions and time-accuracy curves in a selective attention task. The simulation can be used to generate predictions about the effects of dopamine agonists on performance. An empirical study evaluating these predictions is described in a companion paper. Biol Psychiatry 1998;43:713–722 © 1998 Society of Biological Psychiatry

Key Words: Computer simulation models, dopamine, information processing, cognitive science, gain, Eriksen task, selective attention

Introduction

Psychopharmacology has strong scientific foundations in pharmacokinetics, pharmacodynamics, and molecular biology, where good models of underlying mechanisms are available. However, models that can predict behavioral effects of particular drugs from what is known of their physiological effects are notably lacking (Callaway et al. 1994). In many instances, investigators have relied on almost circular explanations of drug effects by using terms that are poorly defined at the psychological level such as “arousal,” “attention,” or “motivation.” Yet, both the animal and human literatures have suggested specific effects of drugs on cognition and behavior—e.g., norepinephrine and acetylcholine effects on sensory processing (Callaway et al. 1992; Halliday et al. 1994), serotonin effects on impulsivity of responding (Soubrie 1989), and dopamine effects on motor initiation (Carlisle et al. 1989)—and this calls for models of the detailed mechanisms underlying information processing in the brain that have the potential to link drug effects with specific aspects of behavior (e.g., Oades 1985).

Until the late 1980s, however, explicit models of human information processing have generally relied on the digital computer metaphor (e.g., Anderson 1983). This metaphor does not easily capture the variability in storage or in computational characteristics under different environmental or internal states that is typical of biological systems. Yet, this variability is critical to understanding drug effects on cognition. In previous work, we have shown that a class of simple neural network models—generally known as parallel distributed processing (PDP) models—can be used to account for the effects of D-amphetamine (D-AMP) on signal detection performance in terms of the physiological effects of dopamine at the neural level (Servan-Schreiber et al. 1990; Cohen and Servan-Schreiber 1992, 1993). In the present paper, we show how a PDP model can be used to generate new predictions about dopamine (DA) effects on behavior. By manipulating the same parameter that captured the effect of DA in the signal detection task, the model predicts the effect of DA on a different aspect of performance—speed and time—accu-
racy curve—in a different task known as the “Eriksen response competition task.”

The Eriksen task requires subjects to ignore salient parts of a stimulus that are irrelevant to the task (i.e., respond to the center letter and ignore the flankers in the array “HHSSH”). It thus taps into processes of selective attention and response competition on which the effects of DA are not known. Furthermore, the task has been studied extensively in psychological and psychophysiological laboratories. As a result, extensive information is available on the mechanisms of information processing that are implicated in performance.

In this first part of a two-part report, we present the modeling study that led to the predictions about DA effects in the Eriksen task. The companion paper describes the results of an empirical study in human subjects that tested these predictions using D-AMP as a probe of the DA system.

Methods and Materials

The model we developed is based on the PDP framework (Rumelhart and McClelland 1986)—which provides a set of principles for simulating neural information processing—and combines the elements of two previously described models: our model of DA effects on neural processing (Servan-Schreiber et al 1990), and our model of the Eriksen task (Servan-Schreiber 1990; Cohen et al 1992). We explain here what these different models are based on and how they were combined.

PDP Framework

The PDP framework provides a set of information-processing mechanisms that can be implemented in computer models that simulate human performance in experimental tasks, and, at the same time, capture salient information-processing characteristics of biological systems (see McClelland 1993 for a review). Processing takes place in networks of interconnected units, each of which is a simple nonlinear device that captures the dynamic characteristics of populations of neurons coding for the same information. A unit accumulates inputs from other units, and adjusts its output in response to these inputs (Figure 1). Typically, units are grouped into modules (e.g., input, output, and intermediate or associative), and modules are connected to each other. Information is represented as the pattern of activation over the units in a module. The activation of each unit is a real valued number varying continuously between a minimum and maximum value. Processing occurs by the propagation of signals (spread of activation) across the connections between units and is subject to random distortion by noise. The connections between units in different modules constitute processing pathways.

Because activation accumulates gradually and in parallel from input modules to output modules, the model displays “processing in cascade”—in which activation accumulates and overflows from one module to the next—as originally imagined by McClelland (1979) and recently supported by the results of event-related potential studies in humans (Osman et al 1992) and neurophysiological experiments in nonhuman primates (Miller et al 1992). Hence, in contrast to the serial model of Sternberg (1969)—which has been used to address drug effects on cognition—assumptions about discrete serial stages of processing are not necessary. Operations of the model can be described in terms of separate mechanisms of processing, such as the response characteristics of units, or the flow of activation from one module to another. In addition, systematic perturbations of the mechanisms of processing incorporated in the model produce changes in the model’s behavior that can be measured using the same dependent variables that are used in humans, such as reaction time distributions and time-accuracy curves. The model can therefore be used to derive specific predictions about how a drug affecting a particular neural mechanism would affect behavior as measured by these dependent variables.

Simulation of the Physiological Effects of Dopamine

To simulate the effects of DA in the PDP framework, we have relied on evidence suggesting that—at physiological concentrations—the release of DA on target neurons does not have an excitatory or inhibitory effect, but rather modulates how target neurons respond to other neurotransmitters (Schneider et al 1984; Aou et al 1983; Sawaguchi and Matsumura 1985; Chiodo and Berger 1986; Barrionuevo, personal communication). Specifically, we have assumed that DA release produces an increase in the responsibility of units to their afferent inputs. This is presumed to take place diffusely in the vicinity of where DA is released (viz., not a topographic but a diffuse regional effect; Stricker and Zigmond 1986). [Though some studies have suggested that DA may have primarily an inhibitory effect on cortical neurons (e.g., Bunney and Aghajanian 1976; Bunney and Sesack 1987; Ferron et al 1984; Reader et al 1979), these studies have relied on rates of stimulation or iontophoresis that are likely to be above those relevant to physiological function.]

This increase in responsibility is simulated in the model by increasing the gain parameter of the activation function of all units in areas where DA transmission is presumed to be increased. The activation function determines how a particular unit becomes activated or inhibited as a function of the inputs it
receives. The gain parameter controls the sensitivity of this response.

The effect of gain on the activation function is illustrated in Figure 2. D-AMP is expected to produce the same effect on individual neurons because at low doses (e.g., 0.1–0.25 mg/kg), D-AMP is thought to have a selective effect on DA release. Indeed, Schneider et al (1984) have found that systemic administration of D-AMP induces an increase in the sensitivity of neurons to excitatory and inhibitory neurons similar to that of the local release of DA.

Note that a gainlike effect in a given cell may be a direct effect of DA on membrane properties of the target cell, or it may be arising from changes in circuit-level interactions, such as those that may arise from inhibition of inhibitory interneurons. At the level of PDP modeling, we are not concerned with the details of how an overall gainlike effect is implemented in particular neural circuits. The focus of the endeavor is to determine whether a change in gain in the model can account for specific drug effects on behavior.

**Simulation of the Eriksen Task**

We first developed a model of a cognitive task that provides a window on the dynamics of information processing and selective attention: the Eriksen response competition task. The effects on this task of drugs enhancing or reducing DA release were not known, which allowed us to generate new behavioral predictions.

In this task, subjects are asked to respond with a different hand to two different target letters (e.g., S or H) that appear in the center of a stimulus array. In the compatible condition, all letters are identical (e.g., HHHHH or SSSSS). In the incompatible condition the surrounding letters are different from the central letter (e.g., HHHHH or SSHSS). Instructions emphasize the importance of speeded responses, and subjects are encouraged to make approximately 15–30% errors. Subjects make more errors and are slower in the incompatible condition.

![Figure 2](https://example.com/figure2.png)

**Figure 2.** The influence of the gain parameter on the activation function of an individual unit. Note that, with an increase in gain, the effect of the net input on the unit’s activation is increased both for excitation and for inhibition. These effects simulate the consequences of increased dopaminergic transmission.

![Figure 3](https://example.com/figure3.png)

**Figure 3.** Example of empirical results in the Eriksen task: time–accuracy curves and reaction time distributions for the compatible (top panel) and incompatible (bottom panel) conditions, averaged over 8 subjects.

Combined measures of reaction time (RT), accuracy, electromyograms (EMGs), and event-related potentials (ERPs) (e.g., Gratton et al 1988) have helped define the mechanisms involved in the Eriksen task. ERPs and EMG studies—using the lateralized readiness potential (LRP) to make inferences about covert activity in response channels—have shown that processing occurs through a continuous, parallel flow of information (rather than in discrete stages as had been postulated previously), that there is a competitive interaction between response channels, and that a response is emitted when activation of the motor cortices exceeds a fixed threshold. Furthermore, the data provide evidence regarding the time course of different processing mechanisms; when accuracy of responses is plotted against reaction time bins, performance can be characterized by a time–accuracy curve (Figure 3). This time–accuracy curve is not the same for the compatible and incompatible conditions. In the compatible condition, accuracy is high (around 70%) even at very fast reaction times and rises to asymptote almost immediately. In the
incompatible condition, accuracy is below 50% at very fast reaction times and rises more progressively to asymptote. The fact that accuracy is below chance level initially suggests that, early in processing, unattended but salient stimuli (flankers) tend to control activity in the response channels, more so than the central stimulus.

We designed a connectionist model that accounts for the different aspects of subjects’ performance in the Eriksen task (Servan-Schreiber 1990; Cohen et al 1992). The network is comprised of three modules of units (Figure 4) with inhibitory connections between all units of a given module (i.e., direct competition), and excitatory connections between modules (information flow). There is an input module to represent features of the stimulus array and a response module in which each unit represents one of the two response alternatives. In addition, an “attention” module provides information about the locus of spatial attention (e.g., center of the array) by enhancing the processing of letters in a particular location over others. Note that for simplicity only one flanker letter on each side of the target letter (center) was included in this simulation of the Eriksen task; however, the simulation results are identical with two flanker letters on each side of the target.

Excitatory connections between the input, object, and attention module are bilateral, supporting interactive processing between these modules. The model was implemented using the C code provided by McClelland and Rumelhart (1988) for interactive activation competition (IAC).

We assume that information about which location should be preferentially attended (i.e., which unit should receive activation in the attention module) is available as the output of some other module following the encoding and interpretation of task instructions. We do not attempt to simulate the process through which task instructions are interpreted; we simply assume that, as a result, the center unit in the attention module receives additional input.

A stimulus is presented by providing some external input to the relevant input units. As activation progressively accumulates at the level of input units, it flows to the response units. When activation of one of the response units exceeds a fixed threshold, a response is recorded, and a new trial is initiated. Random variability in the network’s performance arises from a noise term sampled from a Gaussian distribution and added to the net input of each unit at each cycle of processing. Attentional effects rely on the attention module. Each of the units in the attention module favors one of the possible locations in the stimulus array. Each has bidirectional excitatory connections with the input units that represent particular letters appearing in a particular location (e.g., “H” and “S” at each of the three locations). The “center” attention unit receives external input from the beginning of a trial. This biases processing of the stimulus array in the input module in favor of any letter presented in the center of the array (whether “H” or “S”).

In this network, all stimuli on the input module also tend to attract attention to themselves through the bidirectional connections with the attention module. Input units in the center of the array have an advantage over flanker input units because the “center” attention unit has a higher activation than other attention units through the external input it receives; however, since all input units are connected to the response units, some influence of flanking letters on the response module takes place before the effect of attention on the center letter has fully developed, and this tends to produce inaccurate responses at fast reaction times in the incompatible condition.

Reaction time in the network is measured in terms of the number of cycles necessary for one of the response units to reach an arbitrary threshold (e.g., activation greater than 0.3). A “correct” response is recorded when the response unit reaching threshold corresponds to the center letter of the array. Because the noise term added to each unit at each cycle, the network’s behavior varies from trial to trial, even when the same stimulus is presented. We tested the network’s behavior over 50,000 trials for each condition, compatible and incompatible. Following the method of Gratton et al, we then divided the trials of the stimulation into RT bins (on the basis of the number of cycles) and plotted accuracy separately for each bin. This procedure yields reaction time distributions and time–accuracy curves for the compatible and incompatible conditions.

Figure 5 shows that the simulation simultaneously captures many aspects of the empirical data: 1) the monotonic approach to asymptote of the accuracy curve in the compatible condition; 2) the dip in the early part of the accuracy curve of the incompatible condition; and 3) the overall shape of RT distributions. The relation of these four curves to each other is an example of the constraints that usefully limit the parameter search involved in designing the model. Whereas many different parameter configurations may produce accuracy curves or RT curves comparable to those observed empirically, few will capture the shape of all four curves simultaneously as well as the relation of the curves to each other. [Note that traditional statistical tests are not appropriate to evaluate the quality of the fit between the model and the empirical data. This is because such tests are designed to measure a difference rather than a fit (the null hypothesis). We have subjected the quality of fit of the model to a more recently developed statistical method based on maximum likelihood estimation (Bruno et al in submission), and this procedure...
Figure 5. Results of the simulation of normal performance in the Eriksen task: time–accuracy curves and reaction time distributions for the compatible (top) and incompatible (bottom) conditions (compare to human empirical data in Figure 3). Error bars indicate the 95% confidence interval for the accuracy of each bin.

provided support for all the statements made about quality of fit in this article.

Results

To predict DA effects in the Eriksen task, we explored two hypotheses about the main locus of DA release in the central nervous system following systemic administration of D-AMP.

The first hypothesis follows authors emphasizing the significance of DA release from nigrostriatal fibers and their related effects on probability and execution of motor responses (e.g., Kuczenski et al 1991; Lyon and Robbins 1975). The second hypothesis follows authors emphasizing DA release from the mesolimbic and mesocortical fibers and effects on cognition (e.g., Yamamuro et al 1994; Weinberger et al 1988; Cohen and Servan-Schreiber 1992, 1993). To explore these two hypotheses in the model, we either increased gain over the response module alone—to simulate the consequences of striatal release of DA—or over the attention module—to simulate the consequences of mesocortical release of DA. Of course, D-AMP most likely induces DA release in both of these systems, even if it does so to a different degree in each (e.g., Moghadam et al 1993) and differently so as a function of dose (e.g., Carboni et al 1989; Kuczenski et al 1991); however, to derive predictions related clearly to one mechanism or the other, we decided to explore these two hypotheses separately.

Figure 6 illustrates the results of the simulation after increasing gain only over the response module. There was a large shift to the left of the RT distribution (faster responses) and a deterioration of the time–accuracy curve, resulting in a drop of accuracy independent of the speeding of reaction times (i.e., responses taking the same number of cycles were on average less accurate).

As shown in Figure 7, increasing gain only over the attention module produced a moderate shift of the reaction time distribution to the left (i.e., faster RTs overall) in both conditions. In addition, it resulted in an improvement of the time–accuracy curve that induced an improvement of accuracy, though only in the incompatible condition and only at fast reaction times. This is a nonintuitive prediction, because the existing literature on D-AMP effects on reaction time tasks emphasizes changes in speed, but not in accuracy (e.g., Halliday et al 1987, 1994; Lyon and Robbins 1975; Frowein and Sanders 1978, Frowein 1981). Indeed, in the compatible condition—which resembles most usual reaction time tasks, in which there is no competition between stimuli—this is exactly what is predicted by the model; however, in the incompatible condition, the hypothesis derived from the existing literature—emphasizing a D-AMP-induced shift of the RT distribution to the left—would predict that subjects would make more errors as their performance is shifted toward the region of the time–accuracy curve where accuracy is low. Yet, the model predicts the opposite: an improvement in performance even in the incompatible condition, because the time–accuracy curve shifts upward in the early bins.

We experimented with different values of gain (from 0.5 to 2.5 at 0.05 increments) over the two different modules. Though the extent of the shift of the RT and time–accuracy curves differed, different values all had a similar qualitative effect on the direction of shift for both curves, and the effect was monotonic (i.e., larger values of
gain always shifted the curves further in the same direction.

We also experimented with a combined increase in gain over both the attention and the response module. This resulted in additive effects: greater shift to the left of the RT distribution than either change in gain alone, and no change in time-accuracy curve compared to gain = 1.0.

[Note that the estimates of standard errors for the result of the computer simulation are very low because the model generated 50,000 trials for each condition and each value of gain that we explored. Estimates are less reliable in the first two bins, because the number of trials in these bins is very low. It is important to realize, however, that whether two curves differ statistically cannot be determined entirely from differences between individual data points. This is a limitation of using standard statistical tests, such as t tests, chi-square tests, or even an analysis of variance to evaluate differences between curves. A statistical test applied to two specific points of the curve does not take into account the contribution of neighboring points. A method has been developed for evaluating such differences statistically, and it confirmed the impression conveyed by the figures and described in the text. Details]
are described in Bruno et al in submission; the manuscript is available upon request.]

**Discussion**

Using the framework of parallel-distributed processing, we designed a computer simulation model of human subjects’ performance in the selective attention task of Eriksen. By integrating a model of dopamine effects on target neurons to this simulation, we derived two alternative hypotheses about the effects of the dopamine-releasing drug D-amphetamine on reaction time and accuracy in the Eriksen task. Specifically, the model predicts that if D-amphetamine primarily affects attention processes (i.e., induces a release of dopamine primarily in mesocortical DA neurons), subjects should show a moderate speeding of reaction time together with an improvement of the time–accuracy function in the compatible condition of the task at fast reaction times. If D-amphetamine primarily affects response processes (i.e., induces a release of dopamine primarily in nigrostriatal DA neurons), subjects should show a speeding of reaction time but a deterioration of the time–accuracy function.

**Empirical Nature of Modeling Studies**

We should emphasize the experimental aspect of this modeling study. Although we designed and implemented the different components of the model, the outcome of running the final simulation—i.e., combining the increase in gain with the Eriksen task—was not foreseeable from a knowledge of the individual components. Furthermore, the complexity of interactions between the different elements of the model is such that it is not even possible to derive exact mathematical formulas that could predict the behavior of the model. Predictions require running the computer simulation over many trials and recording reaction times and accuracy scores, much like one does in a study of animal or human subjects. This is because the system is nonlinear and dynamical and that such systems are known to display “emergent properties” from the interaction of many low-level computational elements (Rumelhart and McClelland 1986); however, the model does not become a black box. It can be understood in terms of specific mechanisms by testing it with a variety of different parameter values—such as gain—that are applied to different components—such as specific modules. Such tests characterize the contribution of specific components to the emerging properties of the system.

**Levels of Modeling**

In this current version, the model does not specify how DA produces an increase in gain at the level of individual neural elements. It does not address, for example, whether this may be related to changes in ion channels or other membrane properties of the target neurons, or whether a gain effect may arise from circuit level phenomena, such as an inhibition of inhibitory interneurons. The purpose of the current model is simply to provide a bridge between a physiological effect of DA—modulation of the response characteristic of neurons to neurotransmitters—and its effects at the behavioral level—changes in speed and accuracy of responses in different task conditions. As the results show, it is not necessary to include a detailed account of how dopamine may affect lower-level mechanisms (such as voltage-dependent ion channels, membrane potentials, protein phosphorylation, or gene expression) to do so.

More generally, the contribution of models situated at this bridging level is that they simultaneously integrate principles of neural organization and processing on one hand, and principles of information processing psychology on the other.

The principles rooted in neuroscience can be summarized as follows:

- Information processing in the brain relies on ensembles of interconnected neurons (“units” in the model).
- Neurons are organized in anatomically distinct modules that have different information-processing functions.
- Neurons respond to other neurons by increasing or decreasing their rate of firing (“activation”).
- Communication between neurons is subject to distortion by random noise.

At the same time, the model incorporates the following principles from information-processing psychology:

- Organized behavior arises from the association of specific stimuli to specific responses, mediated and modulated by internal representations.
- A response occurs when sufficient evidence has accrued to rise above a predetermined threshold.
- The accumulation of evidence is subject to distortion by noise, which leads to distributions of reaction times.
- This accumulation of evidence is progressive over time, which gives rise to time–accuracy curves.
- Finally, attention can modulate which stimuli or aspects of a stimulus are selected for accumulation of evidence.

Connectionist models demonstrate how the information-processing principle exhibited at the level of behavior
can be explained in terms of the principles that characterize the neural level, without having to reproduce all the details of the neural level. The specific contribution of the present model is to use these principles to offer a mecha-
nistic explanation for how known physiological effects of DA at the neural level induce an improvement of performance at the behavioral level. The resulting explanation relies on broadly accepted mechanisms of neural processing, and it can replace vague or circular explanations that rely on terms such as “increased arousal,” “improvement of vigilance,” or “improved focusing of attention,” for which no grounding in lower-level mechanisms is provided.

Animal vs. Computer Models

The most common “models” used in psychopharmacology to make predictions about drug effects on human behavior are animal models. Animals are assumed to share with humans much of the relevant biological apparatus. It is thus further assumed that changes in their behavior or physiology following a change in neurotransmitter function do predict comparable changes in humans; however, these assumptions can be misleading when the behavior of interest is related to cognitive functions that may not be as fully developed in animals (such as selective attention), or when the neurotransmitter system of interest is distributed to brain regions in humans that may not have an equivalent in other species. This may be particularly relevant to the role of DA in human cognition, given the important mesocortical projections of tegmental nuclei, especially to the prefrontal cortex (e.g., Lewis 1987, 1992), which is the region of brain anatomy that most significantly differentiates humans from other species. Perhaps even more importantly, animal models can provide important information on neural mechanisms but often remain “cognitively impenetrable” (Pylyshyn 1980) as they still beg the question of how changes at the neural level combine to produce a specific behavior.

Generalizability

The type of attention implicated by our model of the Eriksen task is primarily spatial selective attention; however, we believe that our model can be used to address other forms of attention. Indeed, in other work we have addressed how changes in gain over the attention module can affect behavior in tasks such as the Stroop task and the continuous performance test (Servan-Schreiber et al 1990; Cohen and Servan-Schreiber 1992). In the Stroop task, two aspects of the stimulus can induce competing responses (ink color and word meaning, as in the word RED written in blue ink); and in the continuous performance test, attention is necessary to maintain information about stimulus identity. Neither of these implicate spatial processing, yet the same mechanisms are used to implement response selection and attention. It is thus possible to generate predictions about DA effects in these, and possibly other, paradigms using changes in the gain parameter and observing the consequences on the behavior of the model.

Limitations of the Model and Conclusion

The model also has significant limitations. Most importantly, the response module combines in a single module the function of letter recognition and response selection. On neuroanatomical grounds, it is clear that these two functions are supported by different brain regions and circuits. It is therefore possible that our simulation of increased gain on the output module does not adequately predict the specific effect of DA motor systems alone. Our understanding of the mechanisms at work in the model leads us to believe that dissociating the current response module into two modules—an object recognition and a response selection module—would not qualitatively affect the results; however, this remains to be demonstrated through an implementation of a more elaborate version of the model.

Another limitation arises from the lack of neurophysiological detail. The “units” on which the model is based are only coarse approximations of neural assemblies. They do not capture the detailed function of individual neurons or even circuits of neurons, in which the rate of firing is only one of many changes in neural function occurring during task performance. It is conceivable that implementing these more detailed aspects of neural function may lead to new and different insights about the mechanisms at play in the effects of DA on information processing.

Because they do not capture all the details of biology, computational models can be said to be “biologically impenetrable.” It can legitimately be argued that a good fit to empirical data does not imply that the mechanisms of the model are analogous to those implemented in biology. Other mechanisms may have produced the same results; however, Lakatos (1978) has proposed criteria according to which a new theory or model can be judged to contribute to scientific progress. These criteria do not depend on the level of homology with natural mechanisms. Rather, the test of a model’s strengths and limitations is whether it generates predictions that can be evaluated empirically and whether this validation leads to new interpretations of accepted empirical results. In a companion paper (Servan-Schreiber et al this issue), we report on an empirical study of D-amphetamine effects in the Eriksen task that addresses these issues.
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