

# Human Attentional Networks: A Connectionist Model

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## Abstract

■ Recent evidence in cognitive neuroscience has suggested that attention is a complex organ system subserved by at least three attentional networks in the brain, for alerting, orienting, and executive control functions. However, how these different networks work together to give rise to the seemingly unitary mental faculty of attention remains unclear. We describe a connectionist model of human attentional networks to explore the possible interplays among the networks from a computational perspective. This model is developed in the framework of *leabra* (local, error-driven, and associative, biologically realistic

algorithm) and simultaneously involves these attentional networks connected in a biologically inspired way. We evaluate the model by simulating the empirical data collected on normal human subjects using the Attentional Network Test (ANT). The simulation results fit the experimental data well. In addition, we show that the same model, with a single parameter change that affects executive control, is able to simulate the empirical data collected from patients with schizophrenia. This model represents a plausible connectionist explanation for the functional structure and interaction of human attentional networks. ■

## INTRODUCTION

Although the concept of attention is well understood by many, the mechanism by which this function operates remains unclear. Recent advances in cognitive neuroscience have prompted a renewed interest in treating attention as a neural organ system and have bolstered interest in seeking its neural underpinnings (see Posner & Fan, in press; Posner, 2004). One such organ theoretic account advocates that there exist multiple *attentional networks* in the brain, with each responsible for a different aspect of attention (e.g., Raz & Buhle, 2006; Posner & Dehaene, 2000; Posner & Raichle, 1994; Posner & Petersen, 1990). At least three different attentional networks, for *alerting*, *orienting*, and *executive control*, have been distinguished (see Fan, Raz, & Posner, 2003, for a review). Specifically, alerting refers to the function of achieving and maintaining a heightened internal state of arousal in preparation for coming task-related events. The alerting network has been associated with thalamic, frontal, and parietal areas. An example of the alerting network pathology is hemispatial neglect syndrome, a neuropsychological impairment following unilateral superior parietal damage in which patients neglect objects and events in the contralateral hemispace and appear to be blind, deaf, and numb on this side of space. Orienting refers to selectively focusing on one or a few items out of many candidate inputs. The orienting network includes parts of the superior and inferior parietal lobe, frontal eye fields, the subcortical collicular pathway (the superior

colliculus of the midbrain and the pulvinar), and reticular nucleus of the thalamus. A particular important structure in this network is the posterior parietal cortex, a part of the dorsal “where” pathway (Ungerleider & Mishkin, 1982), which is thought to host multiple supramodal spatial representations in egocentric frames and to guide movements (Wang, Johnson, Sun, & Zhang, 2005; Colby & Goldberg, 1999; Egeth & Yantis, 1997). Finally, executive control refers to monitoring and resolving conflicts in planning, decision making, error detection, and overcoming habitual actions. The executive control network includes the midline frontal areas (anterior cingulate cortex [ACC]), lateral prefrontal cortex, and the basal ganglia. These regions are the target areas of the ventral tegmental dopamine system (Montague, Hyman, & Cohen, 2004; Holroyd & Coles, 2002; Braver & Cohen, 1999). The role of executive control is evidential in a range of Stroop-related tasks (see MacLeod & MacDonald, 2000; MacLeod, 1991).

The attentional networks account of human attention has been systematically examined in recent years using a variety of approaches (e.g., see Raz & Buhle, 2006). Genetically, Fan, Wu, Fossella, and Posner (2001) explored the heritability of the networks by comparing attentional performance between monozygotic and dizygotic twins and found that different networks possessed different degrees of heritability. The underlying genetic variation that might contribute to the brain activation related to executive control has also been investigated (Fan, Fossella, Sommer, Wu, & Posner, 2003). Developmentally, evidence has suggested that different attentional networks possess different developmental profiles. Using children aged from 6 to 10 years old, one study has shown that whereas the

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alerting network continues to develop up to and beyond age 10, the executive control network stabilizes after age 7, and the orienting network shows little development during this period (Rueda et al., 2004). Neuropathologically, it has been found that patients with various attentional disorders often show deficiencies in specific attentional networks. For example, it has been demonstrated that the attentional deficits associated with schizophrenia mainly stem from deficits in the executive control network (Wang, Fan, et al., 2005). Selective impairment of attentional networks has also been found in patients with borderline personality disorder (Posner et al., 2002) and attention deficit/hyperactivity disorder (ADHD) (Konrad, Neufang, Hanisch, Fink, & Herpertz-Dahlmann, 2006; Oberlin, Alford, & Marrocco, 2005; Booth, Carlson, & Tucker, 2001). Recently, a neuroimaging study using functional magnetic resonance imaging (fMRI) was conducted to examine the brain activity in an integrated task designed to simultaneously involve different attentional types (Fan, McCandliss, Fossella, Flombaum, & Posner, 2005). The various attentional components of the task were found to differentially activate separable anatomical networks, which is consistent with predictions from the attentional networks theory.

The organ theoretic attentional networks account of attention highlights the notion that attention is a complex and multifaceted system. It remains unclear, however, how different attentional networks work together to achieve the appearance of a unitary mental faculty. For example, conflicting results on how the attentional networks interact have been reported. On the one hand, the claim that different types of attention are subserved by distinctive attentional networks implies that these underlying attentional networks might be independent. This independence hypothesis has been supported by a behavioral study in which no correlation in performance was found between the three networks, measured using a behavioral testing procedure (Fan, McCandliss, Sommer, Raz, & Posner, 2002). However, in a larger study using a similar testing procedure, a significant correlation between the orienting and executive control network was found (Fossella, Posner, Fan, Swanson, & Pfaff, 2002). Interactions among other networks were also observed using a slightly modified task (Callejas, Lupianez, & Tudela, 2004). Although "it would be surprising if the networks did not interact and influence each in other in various tasks, because there are certainly connections between them," as noted by Fan and Posner (2004, p. 212), these inconsistent findings suggest an intriguing relationship among the attentional networks and have profound implications for the use of the organ theoretic account of attention in systematically explaining various normal and abnormal attentional processes.

One approach to addressing these issues is to develop computational models of attention that are capable of simulating the internal workings of relevant neuronal net-

works (Anderson & Lebiere, 2003; Cohen & Tong, 2001; O'Reilly & Munakata, 2000; Churchland & Sejnowski, 1992). As detailed and computationally implementable hypotheses about real neurobiological systems, computational models have the advantage of explicitly exposing the computational theories, representations, and algorithms underlying cognitive operations (Marr, 1982). By systematically manipulating these computational structures and examining how the models work, we hope to infer and better understand, from an information processing perspective, how the real systems work and, more importantly, why they work the way they do. In addition, the models have the potential to make novel predictions. When the function of a model component is disrupted, for example, model performance changes in a predicted way, which can be computationally simulated and then empirically validated.

Developing computational models of human attention has been a fruitful ongoing research endeavor. For example, Cohen, Romero, Servan-Schreiber, and Farah (1994) developed a connectionist model of orienting-based spatial attention. In this model, selective attention is achieved by a "winner-take-all" type of competition among multiple attention units, and the resulting activation of these attention units in turn enhances (or hurts) the corresponding perception units (Desimone & Duncan, 1995). A similar mechanism was used in a well-known connectionist model of executive control in the Stroop task, where the competition was between different pathways with different strengths (Cohen, Dunbar, & McClelland, 1990). This model was extended recently to link more closely the ACC with executive control and conflict monitoring (Botvinick, Cohen, & Carter, 2004; Botvinick, Braver, Barch, Carter, & Cohen, 2001).

Although these models provide important insights on how neural computations realize attentional selection, from the organ theoretic viewpoint it is clear that they often emphasize only one or a few particular aspects of attention. If attention is better viewed as a complex system subserved by multiple attentional networks and manifested through different types of attention, it is essential that we develop computational models that simultaneously involve multiple attentional networks. By doing so, different aspects of attention can be simultaneously simulated, possible interactions among attentional networks can be examined, and a more complete and integrated understanding of the work of human attention can be achieved.

In this study, we have constructed such a computational model of attention. The model is developed in a biologically realistic connectionist framework named *leabra* (local, error-driven, and associative, biologically realistic algorithm) (O'Reilly & Munakata, 2000). One unique feature of our model is that it simultaneously includes multiple attentional networks as advocated by the organ theoretic account described above. As a result, the issue of how these networks work together to

produce various behavioral manifestations can be systematically explored. We believe that the model represents a plausible computational theory and implementation of how different attentional networks function in a coherent organ system. To evaluate the model, we have applied the model to simulate the empirical results acquired in the Attentional Network Test (ANT), from both normal adult subjects (Fan et al., 2002) and schizophrenic patients (Wang, Fan, et al., 2005).

## METHODS

### Attentional Network Test

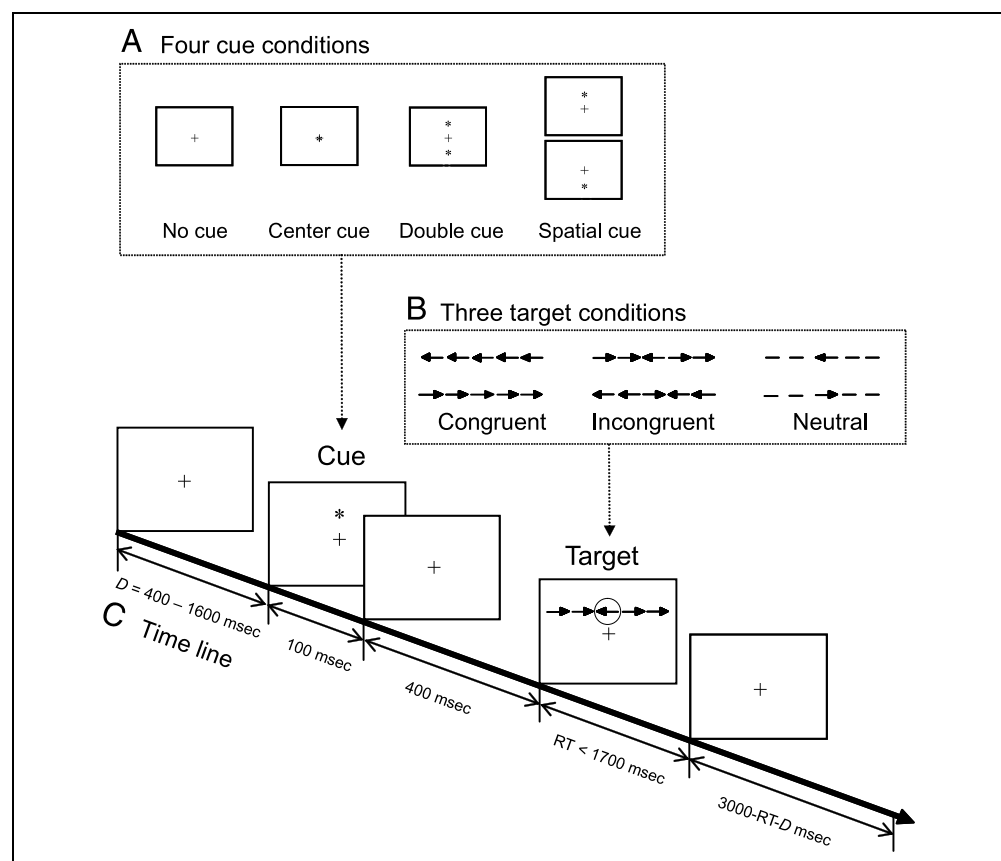
The ANT is an experimental paradigm designed to simultaneously test the effects of the three attentional networks and to evaluate their interrelationships (Fan et al., 2002) (see Figure 1). In the ANT, the *stimulus* consists of a row of five horizontal arrows and the participants' task is to report the direction (left or right) in which the center arrow (the *target*) points. The four arrows surrounding the target (two on each side) serve as flankers and can point either in the same (the *congruent* condition) or opposite (the *incongruent* condition) direction as the center arrow. An additional condition in which the flankers are four lines with no arrowheads is called the *neutral* condition. Presumably additional executive control effort is needed to handle the incongruent condition due to

interference from flankers, leading to a flanker effect (see Bush, Luu, & Posner, 2000; MacLeod & MacDonald, 2000; MacLeod, 1991, for reviews).

To introduce orienting and alerting components, the ANT adopts a spatial cuing technique. First, while the participants are instructed to always focus on center fixation crosshairs ("+" ), the stimulus is to be presented at one of two possible locations, either above the fixation point (top) or below it (bottom). Presumably, the participants have to *reorient* their attention in order to identify the direction of the target. Second, a cue ("\*") may (the *cued* condition) or may not (the *no-cue* condition) be presented before the appearance of the stimulus. If the cue is presented, it essentially *alerts* the participants that the stimulus will soon appear. Third, the alerting cue may be presented at one of three possible locations: the center fixation location (the *center-cue* condition, overlapping with the fixation point), the top or bottom location where the stimulus is to appear (the *spatial-cue* condition), or at both top and bottom locations (the *double-cue* condition, using two "\*" symbols). Note that in the spatial-cue condition, the cue location always validly predicts where the stimulus is to appear. However, this is not the case in the center-cue and the double-cue conditions, in which the participants cannot know the location of the incoming stimulus.

Participants indicate the direction of the target by pressing an appropriate response key and the reaction

**Figure 1.** A sketch of the design of the ANT based on Fan et al. (2002). The goal of the task is to quickly and accurately report, by keypressing, the direction of the center arrow (target) in the stimulus row.



time (RT) is recorded. The following formulae are then adopted to measure the effect of each attentional network:

$$\text{Alerting network effect} = \text{RT}(\text{no cue}) - \text{RT}(\text{double cue})$$

$$\text{Orienting network effect} = \text{RT}(\text{center cue}) - \text{RT}(\text{spatial cue})$$

$$\text{Executive control network effect} = \text{RT}(\text{incongruent}) - \text{RT}(\text{congruent})$$

A study on normal adult participants using the ANT showed an alerting effect of  $47 \pm 18$  msec, an orienting effect of  $51 \pm 21$  msec, and an executive control effect of  $84 \pm 25$  msec (Fan et al., 2002). No evidence of significant correlation was found among the effect measures of the three attentional networks.

It should be noted that the ANT paradigm uses three numbers, in units of time, as the measures of network effects. Because each number actually results from a subtraction of two RT numbers, care should be taken to appropriately interpret them. For example, a large alerting measure may be due to a long RT in the no-cue condition (which indicates difficulty in maintaining alertness without a cue and thus a less efficient alerting attention), or a short RT in the double-cue condition (which indicates a more efficient use of the alerting cue), or a combination of both factors. Similar confounding exists for the orienting and executive control effect measures. Therefore, by the effect numbers alone, it is hard if not impossible to tell for sure how well each network performs and how the system as a whole works together. This is probably one factor that contributes to conflicting findings in term of the interrelationship among different attentional networks. As a result, it has been suggested that to properly interpret the ANT measures, the task, the full range of RTs, and the accuracy data should all be taken into account (Fan & Posner, 2004; Posner et al., 2002).

A small RT difference (about 11 msec) between the center-cue and double-cue conditions has been found (Fan et al., 2002). This may have to do with a subtle difference between the two conditions, which is also reflected by their different uses in the two formulae above calculating the alerting and orienting effects. Specifically, because there are two “\*” symbols appearing simultaneously as the cue at both the “top” and “bottom” locations in the double-cue condition, attention may tend to be diffused a little bit compared to the center-cue condition where only one “\*” symbol appears as the cue at the center fixation location. As a result,  $\text{RT}(\text{center cue}) - \text{RT}(\text{spatial cue})$  seems to be a purer measure for the orienting effect because both the center-cue and spatial-cue conditions involve only one “\*” symbol as the cue.

Similarly,  $\text{RT}(\text{no cue}) - \text{RT}(\text{double cue})$  seems to be a purer measure for the alerting effect. However, because the difference between the two conditions is small, either one may be used in the place of another without significantly affecting the effect measures. This was the procedure used in an fMRI study of attentional networks (Fan et al., 2005), where the double-cue condition was omitted and the center-cue condition was used in calculating both the alerting and orienting effects. That is,

$$\text{Alerting network effect} = \text{RT}(\text{no cue}) - \text{RT}(\text{center cue})$$

We have adopted the same practice in our modeling work reported below.

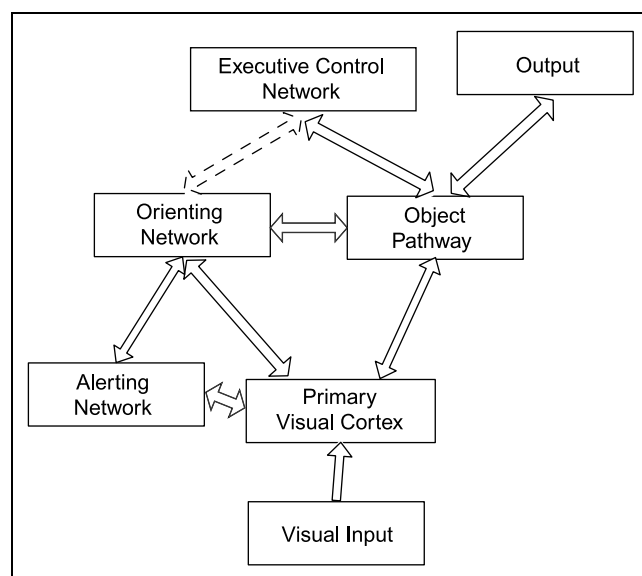
### Computational Model of the ANT

To further explore how different attentional networks work together to give rise to the task performance and network effect measures, we have developed a computational model of the attentional networks based on the ANT.<sup>1</sup> By simultaneously including modules of all three attentional networks and connecting them based on sound biologically realistic principles, we hope the overall model can perform the ANT task and generate effect measures that are comparable with human data. Because this model makes the underlying information representations and algorithms explicit, it has the potential to provide a more detailed and computationally executable explanation for how the task is performed and how different attentional networks interact. In addition, the model makes predictions that can be computationally simulated and empirically tested.

Our model is implemented in a connectionist modeling framework called *leabra*, which is described in (O'Reilly & Munakata, 2000; O'Reilly, 1998). *Leabra* is different from other connectionist modeling frameworks in that it is biologically realistic and possesses several unique features. First, it is constrained by established neurological findings. For example, *leabra* neurons use an activation function that models the known electrophysiology of real neurons as closely as possible while keeping the computation tractable. The connections among neurons in *leabra* cannot freely change signs (i.e., changing from an excitatory link to an inhibitory link, and vice versa), which is possible in earlier artificial neural networks systems and has been shown to be biologically unrealistic. In addition to the biologically inspired Hebbian learning algorithm for model learning, *leabra* uses a special type of Hebbian learning algorithm called contrastive Hebbian learning for error-based task learning, which uses a two-phase mechanism to incorporate error signals. As a result, in *leabra*, it is no longer necessary to backpropagate error signals from output nodes to input nodes, a common criticism for the biologically unrealistic backpropagation learning algorithm.

Second, leabra is a coherently integrated framework. Many distinctions in traditional neural network modeling, including supervised versus unsupervised learning, feedforward versus recurrent networks, and pattern recognition versus self-organization maps, are unified in a single coherent framework. Therefore, for any given task, different information transformation mechanisms and different learning algorithms (Hebbian learning, competitive learning, and error-driven learning) can simultaneously occur and interact. Third, due partly to its biological realism, it is now possible, for example, to develop one leabra network to simulate the primary visual cortex and another network to simulate the posterior parietal cortex, and connect the two networks together with realistic neuroanatomical constraints to form the dorsal “where” pathway. A larger visual cortical system can then be constructed by combining this dorsal network with networks representing the ventral “what” pathway. Each network can have its own properties such as the average activation level and the connection density. As a result, leabra offers us the capability and flexibility to build hierarchies of neural networks to simulate complex cognitive systems.

The conceptual structure of the model is shown in Figure 2. This model contains modules corresponding to



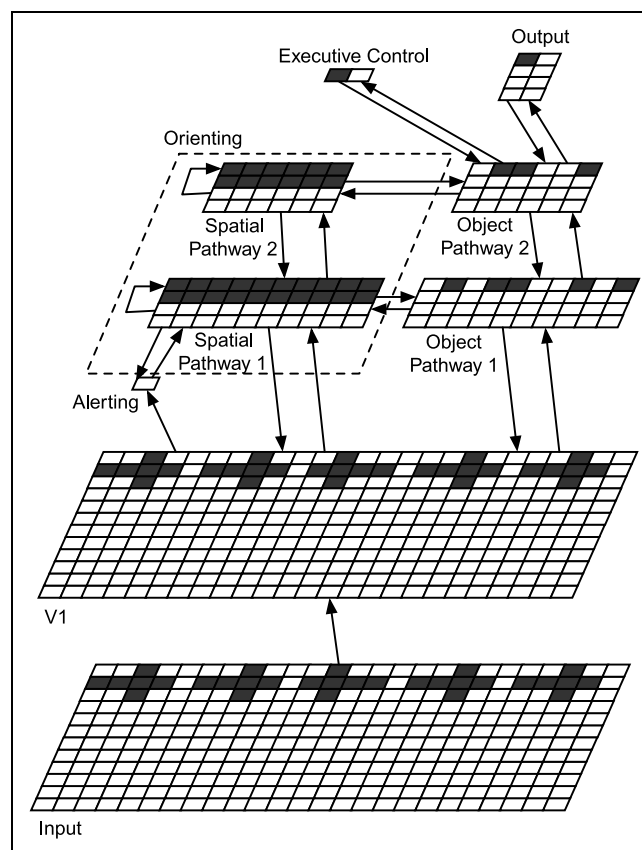
**Figure 2.** The conceptual sketch of the connectionist model of the attentional networks based on the ANT. Each box represents an anatomic or functional neuronal network. The connections among networks are bidirectional (except from visual input to primary visual cortex) and the connection patterns match the function of each network. For the purpose of the modeling work reported in this article, the output network is connected only with the object pathway (i.e., to detect the target arrowhead direction). Similarly, the executive control network is connected only with the object pathway (i.e., to detect and resolve conflicts from distracters). It is possible to also connect the executive control network with the orienting network so to monitor and resolve spatial dimension related conflicts. The dashed connection reflects this possibility.

the three attentional networks, as well as modules for perception (e.g., retina and the primary visual cortex), object recognition (e.g., the what pathway), spatial information processing (e.g., the where pathway), and decision control (output). The module connections conform to various known constraints at both anatomical and functional levels (Farah, 2000; O'Reilly & Munakata, 2000). Specifically, the alerting network receives input mainly from the visual cortex. As soon as an interesting stimulus (e.g., the cue) appears in the visual cortex the alerting network detects it, becomes activated, and then sends its output to other relevant networks (e.g., the orienting network) so as to get them alerted and primed. Note that because the alerting network essentially is just a general-purpose priming/arousal processor, it does not distinguish among different stimuli and/or their spatial locations. Such distinctions occur in modules related to the what and where pathways. The orienting network differentiates spatial locations based on input from the visual cortex. Given the typical retinotopic representations of these networks, any stimulus that appears in a specific location in the visual cortex often only activates specific neurons in the orienting network that monitor that location (i.e., within the neurons' perception fields) (Farah, 2000). The visual cortex network also sends its retinotopy-based representations to networks in the ventral object pathway. The difference between the dorsal orienting network and the ventral object network is that the former magnifies the spatial distinctions among the stimuli and ignores the object-recognition-related features, whereas the latter emphasizes those visual features that are critical for object recognition but collapses on those spatial dimensions. The influence of the orienting network on object recognition (e.g., so that attention can be focused more on the center target instead of peripheral flankers in the ANT) is realized through the connection between the two networks (O'Reilly & Munakata, 2000, Chapter 8). Normally, the output of the object pathway would lead to decisions in the output network (e.g., response module) based on the task goal. However, it is possible that the object pathway's output is too weak or too confusing to afford a decision. In such a case, the executive control network plays a role. Its main function is conflict monitoring *and* resolution (see also Botvinick et al., 2001). It receives input from the object pathway and the orienting network. If task-related conflict (e.g., neurons for “left-pointed arrow” and neurons for “right-pointed arrow” are both receiving input) is detected, the executive control network exerts influence to emphasize the most relevant features (e.g., the center arrow) and deemphasize the less relevant features (e.g., the peripheral arrows). The net result would be that, after the intervention of executive control, decision making becomes possible.

The implementation of the above conceptual model in the leabra framework is basically a straightforward mapping from the conceptual networks to leabra net-

works,<sup>2</sup> constrained by the built-in principles of leabra and the task requirement of the ANT. A snapshot of the implementation is shown in Figure 3. Nine leabra networks are included, with the main characteristics summarized in Table 1.

The input network contains 300 neurons, which are used to represent the visual input at the level of the retina. Each arrow in an ANT stimulus is represented by 15 neurons, organized in a  $5 \times 3$  matrix.<sup>3</sup> At the next level, each stimulus of the ANT, which contains five arrows, is represented by aligning five such  $5 \times 3$  matrices horizontally.<sup>4</sup> Finally, the vertical dimension of the input network



**Figure 3.** A snapshot of the model implementation in leabra. Activation values are grayscale coded (roughly 0 to 1 with light to dark). Visual input (Input) and the primary visual cortex (V1) adopt retinotopic representations (e.g., the incongruent condition is shown here with the center target arrow pointing to the left). The alerting network (Alerting) is implemented by a single neuron network. The orienting network is implemented through a hierarchy of 2 layers of network (Spatial Pathway 1 and Spatial Pathway 2), with the higher level providing a more condensed representation. The same is true for the object pathway (Object Pathway 1 and Object Pathway 2). The executive control network (Executive Control) consists of 2 neurons, responsible for detecting conflict occurring in the object pathway. The activation of either neuron would indicate the occurrence of a conflict, which in turn exerts its influence back to the object pathway (through its feedback connection) to emphasize the center target. This eventually leads to a decision neuron in the output network (Output) to reach the decision threshold and respond.

**Table 1.** Key Parameters in the Leabra Model of ANT

Network	Geometry ( $x \times y \times z$ )	kWTA (No. of Neurons if $>1$ , Percentage if $<1$ )
Input	$5 \times 12 \times 5$	30
V1	$5 \times 12 \times 5$	30
Alerting	$1 \times 1 \times 1$	0.25
Spatial Pathway 1	$2 \times 4 \times 5$	0.25
Spatial Pathway 2	$2 \times 4 \times 3$	0.25
Object Pathway 1	$2 \times 4 \times 5$	5
Object Pathway 2	$2 \times 4 \times 3$	3
Executive Control	$2 \times 1 \times 1$	0.25
Output	$2 \times 4 \times 1$	1

is naturally used to represent the possible cue/stimulus position (top, center, and bottom). Note that the input network actually contains 12 rows of neurons. Because each stimulus requires three rows of neurons for proper representation, the input network actually consists of four “mega” rows. We use the top two mega rows to represent the top location and bottom two mega rows to represent the center location. Within each location’s representation, the top mega row is used only for representing stimuli and the bottom mega row is used only for representing cues.<sup>5</sup> Apparently, because the network only contains 4 mega rows, with 2 for each location (top and center), it does not incorporate the bottom location.

The V1 network, which simulates the visual cortex, adopts a representation scheme that is exactly the same as that of the input network. In other words, neuron activations from the input network are literally copied to the V1 network through a one-to-one connection, resulting in a retinotopic representation in the V1 network. It is important to note, however, that the V1 network, which represents the visual cortex, does not have to be the exact copy of the input network, which represents the retina—nontrivial information processing can occur between the two layers. This is one motivation for us to include two seemingly redundant networks in the current model.

The alerting network contains only one neuron. The function of this network is to monitor the activity in the V1 network and get excited if anything interesting (e.g., a cue) appears there. The alerting network sends its output to the Spatial Pathway 1 network, resulting in a general type of priming effect in the spatial pathway.

The information transformation from the V1 network along the spatial pathway (Spatial Pathway 1 and Spatial Pathway 2) is a result of collapsing on visual features and capitalizing the spatial features. For example, if we

compare the geometric structures of the V1 ( $5 \times 12 \times 5$ ) and Spatial Pathway 1 ( $2 \times 4 \times 5$ ) networks, it is clear that the  $z$  dimension, which represents the five horizontal arrows in the stimulus, does not change, but the  $x$  and  $y$  dimensions, which represent the detailed visual features of arrows, are dramatically reduced. The connection pattern from the V1 network to the Spatial Pathway 1 network demonstrates how the visual feature reduction is done. Figure 4A shows the nonzero connection weights from the V1 network to the top left neuron in the Spatial Pathway 1 network. It is clear that this specific Spatial Pathway 1 neuron's perceptual field is sensitive only to the spatial location (the top left corner of the V1 network) but ignores detailed visual features (i.e., whatever visual stimulus appearing there will tend to activate that Spatial Pathway 1 neuron). Other Spatial Pathway 1 neurons possess similar perceptual fields but with the covering areas shifted rightward or downward (with some overlaps), and therefore the whole V1 network is covered. Further but similar compression is carried out by the projection from the Spatial Pathway 1 network to the Spatial Pathway 2 network.

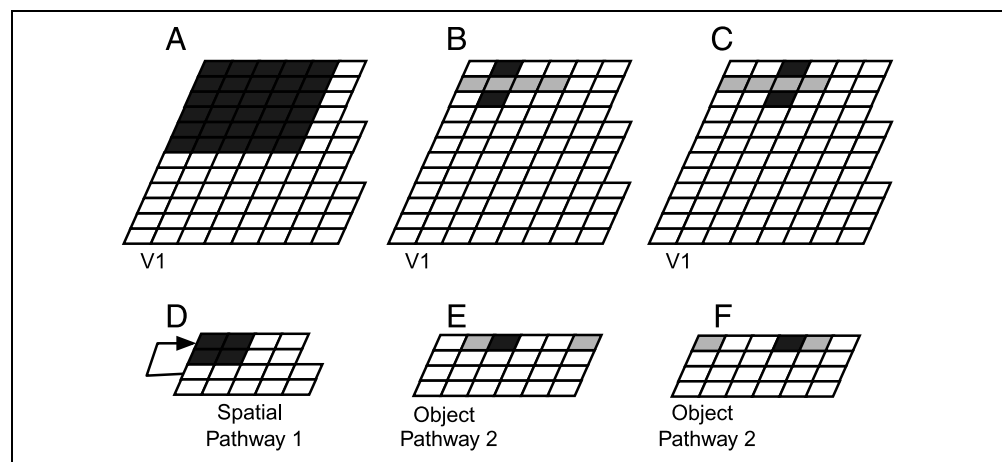
This information transformation pattern is significantly different from that along the object pathway (Object Pathway 1 and Object Pathway 2), where the visual features are emphasized but the spatial distinctions are ignored. Figure 4B shows the nonzero connection weights from the V1 network to the top left neuron in the Object Pathway 1 network. This connection pattern indicates that this specific Object Pathway 1 neuron responds only to the left-pointed arrow. On the contrary, the pattern of connection from the V1 network to the second neuron (to the left of the top left neuron) in the Object Pathway 1 network is quite different. Figure 4C shows that this second neuron responds to the right-pointed arrow. Object recognition is further enhanced through the Object Pathway 2 network, with the left two neurons

representing the left distracters, the center two neurons representing the target, and the right two neurons representing the right distracters. The two neurons in each group represent left-pointed and right-pointed arrows, respectively.

Connections exist between the spatial pathway networks and the object pathway networks to represent the possible interactions between the two pathways. The connection patterns largely follow a retinotopic fashion of mapping. That is, specific areas in a spatial network roughly map to the corresponding areas in the corresponding object network (see Figure 4D for an example). This type of connection allows activations in a spatial pathway network to somehow "prime" the corresponding neurons in an object pathway network even when the visual stimulus is not of the type that the object pathway neurons are designated to recognize. This connection is one of the major reasons why an alerting cue, which activates the alerting network and then the spatial pathway networks, can affect later target detection performance. Similar connections have been adopted in a Stroop effect model (O'Reilly & Munakata, 2000).

The executive control network consists of two neurons, each responsible for detecting a specific type of conflict. Figure 4E represents the connection pattern from the Object Pathway 2 network to the left neuron. It shows that this neuron is activated when a left-pointed target arrow flanked by two right-pointed distracters is detected. Figure 4F represents the connection pattern from the Object Pathway 2 network to the right neuron. It shows that this neuron is activated when a right-pointed target arrow flanked by two left-pointed distracters is detected. In either case, one neuron in the executive control network will be activated. This activation feeds back to the Object Pathway 2 network through the connection from the executive control

**Figure 4.** Key connection patterns used in the model. (A) The receiving weights of the top left neuron in the Spatial Pathway 1 network from the V1 network. (B) The receiving weights of the top left neuron in the Object Pathway 1 network from the V1 network. (C) The receiving weights of the second neuron (the left to the top left neuron) in the Object Pathway 1 network from the V1 network. (D) The receiving weights of the top left neuron in the Object Pathway 1 network from the Spatial Pathway 1 network. (E) The receiving weights of the left neuron in the executive control network from the Object Pathway 2 network. (F) The receiving weights of the right neuron in the executive control network from the Object Pathway 2 network. Weight values are grayscale coded (roughly 0 to 1 with light to dark).



network to the Object Pathway 2 network and strengthens the activation of the two center target neurons, which in effect inhibit the activations of distracter neurons due to the built-in constraints of leabra. As a result, the executive control network performs both conflict detection and conflict resolution functions.

Finally, the output network, through its left and right neuron in each row, is activated by the center target neuron in the Object Pathway 2 network. When the center target neurons are sufficiently activated, one output neuron will in turn be activated. When its activation reaches a set threshold (0.95) a response is said to have been made. The duration, in number of cycles, of the settling process is taken as the measure of model RT.

The “k winners take all” (kWTA) parameter is a critical parameter in leabra. One of its functions is to control the activation level of each network without using explicit inhibitory neurons. In our model, all connections are excitatory and we use leabra’s built-in kWTA and leaking mechanisms to control the networks’ activation levels (see Herd, Banich, & O’Reilly, 2006). As shown in Table 1, the setting of the kWTA parameters is typically straightforward and based on the number of neurons we would like to be activated in each network. Except for these kWTA parameters and the handpicked connection patterns shown in Figure 4, all other parameters used in our model are the default values of leabra (O’Reilly & Munakata, 2000), including various default noise parameters in activation calculation.

## RESULTS

We evaluate the model by first using it to perform the ANT task. A reasonable fit between the model results and the human subjects data lends support to the model as a plausible theory of how the underlying attentional networks function and interact. We then explore whether the model can be used to make testable predictions by applying it to model data from schizophrenic patients.

In a typical simulated ANT trial, stimuli are presented to the model via the input network in a similar way as it would be presented to a human subject (Fan et al., 2002). Depending on the conditions, a cue, which can be either a center cue or a spatial cue, may be presented for a fixed period before the stimulus presentation (note that the double cue condition was not simulated here because the current version of model were not equipped with enough neurons). The number of cycles the output network takes to settle on a stable response after the stimulus presentation serves as the measure of model RT.

The simulation adopts a within-subjects design (i.e., each “simulated subject” performs all different conditions of the task) and 100 simulated subjects have been tested. The simulation results (in cycles) are shown in Table 2, along with the experimental results (in milli-

**Table 2.** The Experimental and Modeling RTs (Mean  $\pm$  Standard Deviation)

Cue	Target	RT		
		Experimental (msec)	Modeling	
			Milliseconds	Cycles
No cue	Neutral	529 $\pm$ 47	536 $\pm$ 7	44.3 $\pm$ 0.6
	Congruent	530 $\pm$ 49	550 $\pm$ 8	45.5 $\pm$ 0.6
	Incongruent	605 $\pm$ 59	629 $\pm$ 10	52.0 $\pm$ 0.8
Center	Neutral	483 $\pm$ 46	490 $\pm$ 6	40.5 $\pm$ 0.5
	Congruent	490 $\pm$ 48	477 $\pm$ 7	39.4 $\pm$ 0.6
	Incongruent	585 $\pm$ 57	540 $\pm$ 9	44.6 $\pm$ 0.7
Spatial	Neutral	442 $\pm$ 39	461 $\pm$ 6	38.1 $\pm$ 0.5
	Congruent	446 $\pm$ 41	444 $\pm$ 7	36.7 $\pm$ 0.6
	Incongruent	515 $\pm$ 58	500 $\pm$ 9	41.4 $\pm$ 0.8

seconds) from Fan et al. (2002). A regression analysis shows that

$$RT(\text{milliseconds}) = 12.1 \times RT(\text{cycle})$$

with an  $R^2$  of .99. We use this regression equation to acquire predicted RTs in milliseconds, which are also shown in Table 2 from easy comparison with experimental results.

From the results in Table 2, we can calculate the average RT in each condition and then estimate the effects of the attentional networks following the formula provided in Fan et al. (2002). We use the center-cue condition in place of the double-cue condition to calculate the alerting effect following the practice in (Fan et al., 2005). The results, together with the experimental results of Fan et al. (2002), are presented in Table 3. One notable difference is a larger alerting effect in modeling results. A further examination shows that this is because the model produced a larger RT in the no-cue condition, which indicates that the model lacks general baseline preparedness (e.g., in absence of a cue). However, the modeling results match the experimental results reasonably well given the fact that we did not change leabra’s default parameter values.

A correlation analysis based on the simulation data is shown in Table 4, along with the experimental data from Fan et al. (2002). It seems that the two sets of data reveal similar correlation structures among the measures. In general, most of the correlations are insignificant. An examination of how the model works suggests that different networks function largely independently, with each contributing to a distinct aspect of the overall task. The significant positive correlation between the executive



**Table 3.** Measures of Attentional Network Effects in Milliseconds (Mean  $\pm$  Standard Deviation)

Effect	Attentional Networks		
	Alerting	Orienting	Executive Control
Experimental	47 $\pm$ 18	51 $\pm$ 21	84 $\pm$ 25
Modeling	70 $\pm$ 7	34 $\pm$ 6	66 $\pm$ 8

control effect and the overall mean RT, shown in both the experiment and the simulation, is likely due to the strong influence of longer RTs in the incongruent condition on the overall mean RT. To a certain degree this correlational structure derived from the modeling results lends support to the claim that there exist multiple functionally distinctive attentional networks as advocated by the organ theoretic account.

There is a notable discrepancy between the modeling results and behavioral results in Table 4. Although the modeling results show a significant negative correlation between the alerting and orienting effect measures no such a correlation was found in the Fan et al. (2002) original empirical study. This discrepancy may be due to the fact that we have used the same center-cue condition to calculate both the alerting and orienting effects in the model whereas, in the Fan et al. study, separate no-cue and center-cue conditions were used in each calculation. In a certain sense this discrepancy serves as an unexpected test about the model's predictive power. That is, can we obtain the same type of correlation if the same center-cue condition is used in the Fan et al. study? This appears to be the case. When we reanalyze the data of Fan et al. using the same formulae we use here, we find a correlation of  $-.20$  between the alerting and orienting effects. Although it is not statistically significant ( $p = .20$ ), the trend toward a negative correlation is evident. The insignificance may be related to the rather large variance in human subjects data.

A careful examination of the model reveals that a possible subtle interplay between the alerting and orienting networks may also play a role in producing the negative correlation. Specifically, the alerting network affects information processing through the spatial pathway networks (see Figure 3). As a result, the spatial cues, which simultaneously activate both the alerting network and the target region in the spatial pathway networks, essentially produce a confounded (alerting + orienting) effect. If there is no interaction during the process, the alerting effect will eventually be canceled out in the calculation of the orienting effect (by subtracting the RT in the spatial cue condition from that in the center-cue condition), leading to no correlation between the alerting and orienting effects. On the other hand, if there is interaction between the two networks then a correlation will arise. It seems that the modeling results support that the latter is likely the case.

**Table 4.** Correlations among Attentional Networks<sup>a</sup>

	Alerting	Orienting	Executive Control
<i>Experiment:</i>			
Orienting	.08		
Executive control	.05	-.12	
Mean RT	.09	.29	.44 <sup>b</sup>
<i>Model</i>			
Orienting	-.47 <sup>b</sup>		
Executive control	.08	-.003	
Mean RT	.09	.06	.24 <sup>b</sup>

<sup>a</sup>Correlations are calculated based on the relevant measures of all subjects (or simulated subjects).

<sup>b</sup>Correlation is significant at the .01 level.

This discrepancy highlights an important aspect of the model's value. The model provides a detailed and executable system that allows us to systematically explore not only how the simulated system may work but also why it works. In addition, because the modeling performance is essentially a function of model structures and parameters, altering these model components may lead to novel and testable predictions. To demonstrate this, we have used the model to simulate schizophrenic patients' attentional networks profile. We recently used the ANT paradigm to test the selective attentional impairment in patients with schizophrenia (Wang, Fan, et al., 2005). One of the major findings is that patients, compared to normal controls, showed a much larger executive control effect ( $153 \pm 10$  msec), indicating a deficit in the executive control network. This result is consistent with a large body of neuropsychological evidence showing that schizophrenia involves attentional deficits in general and dopamine-related executive control deficits in particular (Braver, Barch, & Cohen, 1999; Braver & Cohen, 1999; Fletcher, McKenna, Friston, Frith, & Dolan, 1999). Given that our model is able to simulate normal subjects' ANT performance, we hypothesize that without resorting to more complex algorithms, but with minimal and biologically justified changes, the model should be able to simulate schizophrenic patient's ANT performance as well. To test this hypothesis, we make only one change to our original model. The leaking conductance parameter of the two neurons in the executive control network is changed from the default 0.1 to 1. Because this parameter in leabra summarizes the function of potassium-based leaking channels in real neurons, by increasing it we essentially make our executive control neurons harder to fire. This means that the conflict detection capacity is damaged, which then leads to diminished conflict resolution capacity. We have run 100 "simulated schizophrenic patients" with this change. Our

results show that although this manipulation leaves other conditions largely intact, it greatly increases RTs in the incongruent conditions, which result in a significant increase in the executive control effect measure ( $66 \pm 8$  vs.  $120 \pm 9$  msec).

## DISCUSSION

Traditional wisdom has treated attention as a unitary cognitive faculty. Recent advances in cognitive neuroscience have suggested that attention be viewed as a complex organ system that consists of multiple attentional networks in the brain, with each responsible for a different type of attention. At least three attentional networks (i.e., alerting, orienting, and executive control) have been identified at multiple levels (Posner, 2004; Posner & Dehaene, 2000; Posner & Raichle, 1994). However, how the different networks work together to give rise to the seemingly unitary mental faculty of attention raises a challenge.

One approach to this issue is to develop principled computational models (Cohen & Tong, 2001). These models, often as detailed and executable hypotheses of the real cognitive system, help to fill the gap by adopting a unified information-processing-based language to explain how the system works. More importantly, these models can be “opened” and more thoroughly examined in the sense that they explicitly expose the critical but often hidden computational goals, representations, and algorithms that implement the real system. In the present study, we have developed a connectionist model of human attentional networks in the biologically realistic connectionist modeling framework (O’Reilly & Munakata, 2000). By simultaneously incorporating multiple attentional networks, the model offers a simulation platform for us to hypothesize and infer in explicit detail how information is represented and transformed along different neuronal pathways and how different attentional networks work together to produce observable attention-related behavior.

We believe that our model represents a plausible connectionist explanation for the attentional networks account of attention. By presenting the model stimuli similar to what a human subject in the ANT would see, we show that the model is able to perform the task and produce the RT patterns similar to what human subjects demonstrated. The simulation results suggest that the model captures the various attentional effects that the ANT task was designed to measure. With a single parameter change to the model, we have shown that model is also able to simulate data collected from patients with schizophrenia.

To further clarify how the model can be used as a computational metaphor to explain these attentional effects, we recapitulate the model’s behavior in various ANT conditions. When a cue is presented, the primary visual cortex module is activated, which in turn triggers the alerting network. This cue-induced alerting affects later

stimulus processing because the alerting network will remain excited for a while, which will activate the orienting network, priming it to be ready for the incoming stimulus and thus inducing the alerting effect. In addition, when the cue is a spatial one (i.e., a cue that indicates where the stimulus is to appear), it will further excite the corresponding subregion of the orienting network. This occurs because the orienting network adopts a retinotopy-based spatial representation of the environment. This extra excitation in the subregion of the orienting network will facilitate the corresponding stimulus processing in the object pathway network, due to the connections between them. This accounts for the orienting effect. On the contrary, when the cue is a center one (i.e., the cue appears in the fixation location and does not tell where the stimulus is to appear), a similar facilitation effect occurs; only this time it occurs in the wrong location, which may hurt later stimulus processing due to a possible disengagement process (Posner, 1980). Finally, note that it is the object pathway network that is responsible for the arrow direction detection. When the incongruent stimulus (e.g., a left arrow flanked by four right arrows) is presented, the object pathway network may propose different responses, which compete for final expression in the output network. The executive control network detects the conflict and then activates, enhancing the center arrow and suppressing the flankers. This is where the executive control attention plays a role. If we increase the leaking conductance of neurons in the executive control network, we essentially alter the normal function of the network and impair its conflict monitoring and resolution capacity. As a result, a larger executive control effect is derived, which is consistent with the experimental results from patients with schizophrenia.

Note that the model does not perform conventional neural network learning (e.g., parameter tuning). The connection patterns and weights are all manually set based on known biological constraints and task-specific requirements. Although it is possible in principle to adopt leabra’s built-in learning algorithms to let the model learn these connection weights by itself, the current handcrafted model demonstrates more clearly the concepts and elements that are responsible for the results.

In addition, the complexity of the model, both in terms of the large number of built-in parameters and complex pattern of connections, underscores the caution Fan and Posner (2004) and Fan et al. (2002) advocate in explaining the ANT measures. Because the ANT measures result from multiple RT measures, it is quite possible to change one or a few RT measures by imposing certain constraints in the model, thus dramatically changing the effect measures and the correlation structures. One example is to increase RTs in the center-cue condition (which can be done by altering the orienting network to weaken disengagement, for example). As a result, the alerting effect would be lower and the orienting effect would be higher, leading to even larger negative correlation in the two networks.

Model complexity can be reduced by following more strict biologically realistic principles in model development. Doing so will also permit us to more accurately model and predict how damage in a specific part of the network or connections can lead to specific behavioral patterns. We have demonstrated this by showing that we can alter the model to simulate schizophrenia data. Based on the organ theoretic attentional networks account of attention, attentional impairments in many other psychiatric disorders, such as ADHD, involve specific deficits in one or a few attentional networks. The model developed in this study offers a framework for us to start unfolding the computational structures underlying these phenomena and to make testable predictions.

As another example of the model's predictive power, consider the possibility of invalid spatial cues. Posner's (1980) seminal work using invalid peripheral cues demonstrated the *validity effect*. That is, subjects typically responded faster to a target presented at the validly cued location than that at the invalidly cued location. In a certain sense it would seem that the validity effect is simply a byproduct of the orienting effect. However, we know that the alerting effect is sensitive to the temporal contiguity between the cue and the stimulus, and the executive control effect is sensitive to the degree of cue-target contingency. If we add invalid spatial-cue condition in the ANT task and manipulate the temporal contiguity and cue-target contingency, we may expect new interactions among different effects. If we feed the model with these designed invalid spatial cues, the model would make clear predictions about what will happen, which can then be empirically tested.

Finally, we want to reemphasize the critical challenge that motivates the present study. That is, how do different attentional networks work together to achieve seemingly unitary attentional behavior? Indeed, the problem of linking brain activation found in various neuroimaging studies with observable psychological behavior and avoiding practice of so-called neophrenology has become a challenge for cognitive neuroscience in general (Cohen & Tong, 2001). Although both the genetic and neural "shadows of the mind" reveal important aspects of human cognition, neither alone fully explains cognition (Scerif & Karmiloff-Smith, 2005). We propose a meta-modeling approach to this problem (see also Wang, Fan, & Yang, 2004). According to this approach, we need to develop biologically realistic and psychologically plausible computational models at multiple levels and compare them, contrast them, mutually justify them, and more importantly, probe the link among them. We (Wang, Fan, & Johnson, 2004) have developed a symbolic model of human attentional networks in ACT-R, a primarily rule-based cognitive architecture (Anderson et al., 2004; Anderson & Lebiere, 1998). The connectionist model developed in this study represents another step toward the goal of meta-modeling of human attention. Because both the ACT-R-based model and the leabra-based model

are process models and fit the empirical data well, we can parallel the two models and explore how "attention" manifests itself at different levels along the time dimension. This work is currently ongoing.

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## Notes

1. The model can be downloaded from [www.shis.uth.tmc.edu/Members/hwang](http://www.shis.uth.tmc.edu/Members/hwang).
2. These networks are actually represented by leabra layers in the overall model. We use the two terms (networks and layers) interchangeably in discussing the model.
3. See the top left  $5 \times 3$  neuron matrix of the Input network in Figure 1 as an example, which literally represents a right-pointed arrow.
4. For example, the top three rows in the input network in Figure 1 contain a total of  $5 \times (5 \times 3) = 75$  neurons and represent an incongruent stimulus, with the target pointing to left and four flankers pointing to right.
5. Again, as an example, the input network in Figure 1 shows that an incongruent stimulus is presented at the top location.

## REFERENCES

- Anderson, J. R., Bothell, D., Byrne, M. D., Douglass, S., Lebiere, C., & Qin, Y. (2004). An integrated theory of the mind. *Psychological Review*, 111, 1036–1060.
- Anderson, J. R., & Lebiere, C. (1998). *The atomic components of thought*. Hillsdale, NJ: Erlbaum.
- Anderson, J. R., & Lebiere, C. (2003). The Newell Test for a theory of cognition. *Behavioral and Brain Science*, 26, 587–637.
- Booth, J., Carlson, C. L., & Tucker, D. (2001). *Cognitive inattention in the ADHD subtypes*. Paper presented at the tenth scientific meeting of the International Society for Research in Child and Adolescent Psychopathology (ISRCAP), Vancouver, BC, Canada.
- Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. *Psychological Review*, 108, 624–652.
- Botvinick, M. M., Cohen, J. D., & Carter, C. S. (2004). Conflict monitoring and anterior cingulate cortex: An update. *Trends in Cognitive Sciences*, 8, 539–546.
- Braver, T. S., Barch, D. M., & Cohen, J. D. (1999). Cognition and control in schizophrenia: A computational model of dopamine and prefrontal function. *Biological Psychiatry*, 46, 312–328.
- Braver, T. S., & Cohen, J. D. (1999). Dopamine, cognitive control, and schizophrenia: The gating model. *Progress in Brain Research*, 121, 327–349.

- Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Sciences*, 4, 215–222.
- Callejas, A., Lupianez, J., & Tudela, P. (2004). The three attentional networks: On their independence and interactions. *Brain and Cognition*, 54, 225–227.
- Churchland, P. S., & Sejnowski, T. J. (1992). *The computational brain*. Cambridge: MIT Press.
- Cohen, J. D., Dunbar, K., & McClelland, J. L. (1990). On the control of automatic processes: A parallel distributed processing account of the Stroop effect. *Psychological Review*, 97, 332–361.
- Cohen, J. D., Romero, R. D., Servan-Schreiber, D., & Farah, M. J. (1994). Mechanisms of spatial attention: The relation of macrostructure to microstructure in parietal neglect. *Journal of Cognitive Neuroscience*, 6, 377–387.
- Cohen, J. D., & Tong, F. (2001). The face of controversy. *Science*, 293, 2405–2407.
- Colby, C. L., & Goldberg, M. E. (1999). Space and attention in parietal cortex. *Annual Review of Neuroscience*, 22, 319–349.
- Desimone, R., & Duncan, J. (1995). Neural mechanisms of selective visual attention. *Annual Review of Neuroscience*, 18, 193–222.
- Egeth, H. E., & Yantis, S. (1997). Visual attention: Control, representation, and time course. *Annual Review of Psychology*, 48, 269–297.
- Fan, J., Fossella, J. A., Sommer, T., Wu, Y., & Posner, M. I. (2003). Mapping the genetic variation of executive attention onto brain activity. *Proceedings of the National Academy of Sciences, U.S.A.*, 100, 7406–7411.
- Fan, J., McCandliss, B. D., Fossella, J., Flombaum, J. I., & Posner, M. I. (2005). The activation of attentional networks. *Neuroimage*, 26, 471–479.
- Fan, J., McCandliss, B. D., Sommer, T., Raz, A., & Posner, M. I. (2002). Testing the efficiency and independence of attentional networks. *Journal of Cognitive Neuroscience*, 14, 340–347.
- Fan, J., & Posner, M. I. (2004). Human attentional networks. *Psychiatrische Praxis*, 31, s210–s214.
- Fan, J., Raz, A., & Posner, M. I. (2003). Attentional mechanisms. In M. J. Aminoff & R. B. Daroff (Eds.), *Encyclopedia of neurological sciences* (Vol. 1, pp. 292–299). San Diego, CA: Academic Press.
- Fan, J., Wu, Y., Fossella, J. A., & Posner, M. I. (2001). Assessing the heritability of attentional networks. *BMC Neuroscience*, 2, 14–20.
- Farah, M. J. (2000). *The cognitive neuroscience of vision*. Malden, MA: Blackwell.
- Fletcher, P., McKenna, P. J., Friston, K. J., Frith, C. D., & Dolan, R. J. (1999). Abnormal cingulate modulation of fronto-temporal connectivity in schizophrenia. *Neuroimage*, 9, 337–342.
- Fossella, J., Posner, M. I., Fan, J., Swanson, J. M., & Pfaff, D. W. (2002). Attentional phenotypes for the analysis of higher mental function. *The Scientific World*, 2, 217–223.
- Herd, S. A., Banich, M. T., & O'Reilly, R. C. (2006). Neural mechanisms of cognitive control: Integrative model of Stroop task performance and fMRI data. *Journal of Cognitive Neuroscience*, 18, 22–32.
- Holroyd, C. B., & Coles, M. G. H. (2002). The neural basis of human error processing: Reinforcement learning, dopamine, and the error-related negativity. *Psychological Review*, 109, 679–709.
- Konrad, K., Neufang, S., Hanisch, C., Fink, G. R., & Herpertz-Dahlmann, B. (2006). Dysfunctional attentional networks in children with attention deficit/hyperactivity disorder: Evidence from an event related functional magnetic resonance imaging study. *Biological Psychiatry*, 59, 643–651.
- MacLeod, C. M. (1991). Half a century of research on the Stroop effect: An integrative review. *Psychological Bulletin*, 109, 163–203.
- MacLeod, C. M., & MacDonald, P. A. (2000). Interdimensional interference in the Stroop effect: Uncovering the cognitive and neural anatomy of attention. *Trends in Cognitive Sciences*, 4, 383–391.
- Marr, D. (1982). *Vision*. San Francisco, CA: Freeman.
- Montague, P. R., Hyman, S. E., & Cohen, J. D. (2004). Computational roles for dopamine in behavioural control. *Nature*, 431, 760–767.
- Oberlin, B. G., Alford, J. L., & Marrocco, R. T. (2005). Normal attention orienting but abnormal stimulus alerting and conflict effect in combined subtype of ADHD. *Behavioral Brain Research*, 165, 1–11.
- O'Reilly, R. C. (1998). Six principles for biologically based computational models of cortical cognition. *Trends in Cognitive Sciences*, 2, 455–462.
- O'Reilly, R. C., & Munakata, Y. (2000). *Computational explorations in cognitive neuroscience*. Cambridge: MIT Press.
- Posner, M. I. (1980). Orienting of attention. *Quarterly Journal of Experimental Psychology*, 32, 3–25.
- Posner, M. I. (Ed.). (2004). *Cognitive neuroscience of attention*. New York: Guilford Press.
- Posner, M. I., & Dehaene, S. (2000). Attentional networks. In M. S. Gazzaniga (Ed.), *Cognitive neuroscience: A reader*. Malden, MA: Blackwell.
- Posner, M. I., & Fan, J. (in press). Attention as an organ system. In J. Pomerantz (Ed.), *Neurobiology of perception and communication: From synapse to society. The IVth De Lange Conference*. Cambridge, UK: Cambridge University Press.
- Posner, M. I., & Petersen, S. E. (1990). The attention systems of the human brain. *Annual Review of Neuroscience*, 13, 25–42.
- Posner, M. I., & Raichle, M. E. (1994). *Images of mind*. New York: Scientific American Library.
- Posner, M. I., Rothbart, M. K., Vizueta, N., Levy, K. N., Evans, D. E., Thomas, K. M., et al. (2002). Attentional mechanisms of borderline personality disorder. *Proceedings of the National Academy of Sciences, U.S.A.*, 99, 16366–16370.
- Raz, A., & Buhle, J. (2006). Typologies of attentional networks. *Nature Reviews Neuroscience*, 7, 367–379.
- Rueda, M. R., Fan, J., McCandliss, B. D., Halparin, J. D., Gruber, D. B., Lercari, L. P., et al. (2004). Development of attentional networks in childhood. *Neuropsychologia*, 42, 1029–1040.
- Scerif, G., & Karmiloff-Smith, A. (2005). The dawn of cognitive genetics? Crucial developmental caveats. *Trends in Cognitive Sciences*, 9, 126–135.
- Ungerleider, L. G., & Mishkin, M. (1982). Two cortical visual systems. In D. J. Ingle, M. A. Goodale, & R. J. W. Mansfield (Eds.), *Analysis of visual behavior*. Cambridge, MA: MIT Press.
- Wang, H., Fan, J., & Johnson, T. R. (2004). A symbolic model of human attentional networks. *Cognitive Systems Research*, 5, 119–134.
- Wang, H., Fan, J., & Yang, Y. (2004). Toward a multilevel analysis of human attentional networks. In K. Forbus, D. Gentner, & T. Regier (Eds.), *Proceedings of the Twenty-Sixth Annual Meeting of the Cognitive Science Society* (pp. 1428–1433). Mahwah, NJ: Erlbaum.
- Wang, H., Johnson, T. R., Sun, Y., & Zhang, J. (2005). Object-location memory: The interplay of multiple representations. *Memory & Cognition*, 33, 1147–1159.
- Wang, K., Fan, J., Dong, Y., Wang, C.-Q., Lee, T. M. C., & Posner, M. I. (2005). Selective impairment of attentional networks of orienting and executive control in schizophrenia. *Schizophrenia Research*, 78, 235–241.