NEURAL DYNAMICS OF AUTISTIC BEHAVIORS:

Learning, Recognition, Attention, Emotion, Timing, and Social Cognition

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Definitions of Key Terms and Facts

Category learning. This process enables variations on the same object or event to learn how to activate the same category, or symbol, that represents this object or event in the brain. Activating such a learned category enables the brain to recognize the object or event through a process that includes read-out of the category's learned top-down expectation, which focuses attention upon the critical features that define the category, and helps to stabilize the category learning process.

Vigilance. This process concerns the sensitivity of the brain to the mismatch between an exemplar that represents an object or event in the world and the top-down expectation that is read-out by a currently active recognition category. Vigilance is a gain parameter that can change through time to control whether a category will learn concrete or abstract information about the

world. High vigilance leads to the learning of a concrete category (e.g., one that is activated by particular view of a familiar person's face), whereas low vigilance leads to the learning of abstract category (e.g., one that is activated by any face). During high vigilance, attention focuses on fine details. Lower vigilance enables attention to focus on more holistic aspects of an object or scene.

Attention. This process concerns the brain's ability to selectively process a subset of available information about the world. Object attention focuses on predictively relevant combinations of features, such as the critical features that define a learned category. Spatial attention focuses on locations of particular interest in a given situation. Object and spatial attention often work together to learn about objects and events in the world and to act upon them.

Cognitive-emotional processing. This process enables the brain to combine cognitive and emotional information to determine what events in the world predict desired or undesired outcomes. Cognitive-emotional processing also enables the brain to learn how to respond to objects and events with contextually appropriate feelings, and to motivate the brain to focus attention and appropriate actions towards desired or undesired objects and events.

Opponent emotions. Emotions are often organized in opponent pairs, one with positive affect, and the other with negative affect. Learning to balance these opponent affects enables the learning and unlearning of emotional responses based upon their predictive success. Opponent emotional circuits also enable appropriate emotions to be felt in different situations, and help to control the release of motivationally appropriate actions.

Adaptive timing. This process enables the brain to learn how to respond to environmental contingencies with appropriately timed actions. Adaptively timed learning enables the brain to detect and learn about rewards and punishments that occur after their predictive cues terminate. Such learning occurs in several parts of the brain, including the cerebellum, hippocampus, and basal ganglia. A lesion of the hippocampus can, for example, prevent trace conditioning, which is the kind of conditioning that occurs when a cue and reward or punishment are separated in time. Neural models suggest how adaptively timed learning in the hippocampus may help to maintain motivated attention upon a task while it is being carried out; in the cerebellum may help to control correctly timed read-out of learned actions; and in the basal ganglia may help to control learning in response to unexpected rewards and punishments.

Social cognition. This process concerns the collection of knowledge and skills whereby individuals can learn to successfully adapt to the many challenges that society poses for its members. Social cognitive learning includes the ability of an individual to learn from a teacher, despite the fact that the learner and teacher experience the world from different perspectives. Learning how to use a tool from a teacher illustrates this capability.

Joint attention. This process concerns the ability of a student to learn how to attend to where a teacher is looking, despite the fact that student and teacher experience the world from different perspectives.

Imitation learning. This process concerns the ability of a student to learn a skill from a teacher, despite having observed it from a different perspective than the teacher does when performing the skill.

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Summary Points

This chapter describes how the Adaptive Resonance Theory (ART), Cognitive-Emotional-Motor (CogEM), and Spectral Timing neural models link brain mechanisms to behavioral functions. These models describe brain processes in sensory, temporal, and prefrontal cortex; amygdala; hippocampus; cerebellum; and basal ganglia, among other brain regions.

The Spectrally Timed ART (START) model joins together these models into a unified brain architecture.

These models were developed to explain how the brain generates normal behaviors.

Specific imbalances in model brain mechanisms in the imbalanced START (iSTART) model lead to behavioral symptoms of autism.

The iSTART model predicts how problems in learned categorization and attention, cognitive-emotional responsiveness, and adaptive timing can lead to autistic symptoms, and how these processes can reinforce each other through interactions within the brain and with environmental feedback.

The following mechanisms are predicted by iSTART to influence autistic behaviors:

Attentive vigilance in inferotemporal cortex may be stuck at a high level in some autistic individuals, thereby causing concrete category learning and narrow attentional focusing.

The Synchronous Matching ART (SMART) model proposes how ART mechanisms may be realized by identified cells in the laminar circuits of neocortex and thalamus, and predicts how vigilance may be controlled by acetylcholine release via the nucleus basalis of Meynert.

Tonic arousal of amygdala and other emotion-representing brain areas may be abnormally low in some autistic individuals, leading to a high threshold for reacting to cues, but hypersensitivity to suprathreshold cue increments. Small arousal bursts to slight changes in sensory cues may enhance ongoing positive affect, but larger arousal bursts due to larger changes in cues may cause unusually strong negative affective rebounds. This combination of affective properties in affectively underaroused individuals may clarify the autistic preference for sameness.

Adaptive timing by cerebellum, hippocampus, and basal ganglia may be deficient, thereby preventing effective reinforcement learning and social interactions.

Interactions between recognition learning and attention under high vigilance, underaroused emotional depression, and faculty adaptive timing may lead to a variety of behavioral and social problems.

More complex behaviors that build upon these recognition, emotional, and timing processes may also be impaired, such as language learning.

The Circular Reactions for Imitative Behavior (CRIB) model proposes how a student may learn from a teacher who experiences the world from a different perspective, and how learning of joint attention and other social cognitive skills may be impaired by the above brain properties.

ABSTRACT

A full understanding of a complex spectrum of mental conditions such as autism would show how the brain dynamics of autistic individuals give rise to their behavioral symptoms, and how the brain dynamics of normal individuals are changed in autism. This article summarizes neural models that contribute to both goals. One of these neural models is called the iSTART (imbalanced Spectrally Timed Adaptive Resonance Theory) model. iSTART proposes how cognitive, emotional, timing, and motor processes that involve brain regions like sensory, temporal, and prefrontal cortex, amygdala, hippocampus, cerebellum, and basal ganglia may interact together to create and perpetuate autistic symptoms. These model processes were originally developed as part of the START model to explain data concerning how the brain controls normal behaviors. The iSTART model is a synthesis of three models that shows how autistic behavioral symptoms may arise from prescribed breakdowns in several types of brain processes: underaroused emotional depression in the amygdala and related affective brain regions (Cognitive-Emotional-Motor, or CogEM, model) and how it influences Theory of Mind and hyperreactivity to novel events; high vigilance attentional processing and how it influences the learning of hyperspecific recognition categories and narrow attention in temporal and prefrontal cortices (Adaptive Resonance Theory, or ART, model); and breakdowns of adaptively-timed attentional and motor circuits in the hippocampal system, cerebellum, and basal ganglia (Spectral Timing model) and how they may hinder social development and language development. iSTART clarifies how malfunctions in a subset of these mechanisms can, though environmentally mediated feedback, cause and maintain problems with them all. The SMART (Synchronous Matching ART) model additionally proposes how chronically high vigilance may be traced to how acetycholine is released from the nucleus basalis of Meynert. Finally, the CRIB (Circular Reactions for Imitative Behavior) model clarifies how imitation learning can occur between a student and a teacher who experience the world from different perspectives, and how the development of social cognitive capabilities such as joint attention and imitation learning with such a teacher may be impaired by the above problems.

1. Introduction

This article reviews neural models that explain data about normal brain and behavior, as well as autistic behavioral symptoms when their neural mechanisms become imbalanced in specific ways. This linkage between normal and abnormal brain and behavior provides many constraints for evaluating proposals about the causes of autism, since data about normal brain and behavior are more plentiful and parametrically organized than data about clinical symptoms.

The article summarizes key organizational principles, neural mechanisms, and emergent behavioral properties of the imbalanced Spectrally Timed Adaptive Resonance Theory (iSTART; Grossberg and Seidman, 2006), the Synchronous Matching ART (SMART; Grossberg and Versace, 2008), and the Circular Reactions for Imitative Behaviors (CRIB; Grossberg and Vladusich, 2010) model. The iSTART model arose from the START model (Grossberg and Merrill, 1992, 1996) that was developed to quantitatively explain data about normal brain and behavior. Particular imbalances of START processes in iSTART give rise to behaviors that strikingly resemble autistic properties of attention and categorization, cognitive-emotional processing, and adaptive timing of behavior. Table 1 summarizes some of these symptoms.

The START model provides a foundation for discussing the role of the basal forebrain and acetylcholine in attention and learning (as modeled by SMART); the way in which a student can learn to share joint attention with, and to imitate behaviors of a, teacher who experiences the world from a different perspective (as modeled by CRIB); and how these competences may break down in autism.

2. iSTART: A Neural Model of How Autistic Symptoms Can Arise

START unifies into a single neural architecture three component models: Adaptive Resonance Theory, or ART; the Cognitive-Emotional-Motor model, or CogEM; and the Spectral Timing model.

ART proposes how the brain learns to categorize and recognize objects and events, pays attention to important events, and predicts future events. This occurs through matching of bottom-up perceptually-driven inputs with learned top-down expectations. A top-down expectation uses learned prototypes to focus attention on critical feature patterns that define its recognition category, and to prime the brain to anticipate expected

Table 1
Some Key Behavioral Symptoms of Autism

Social skill impairments
Verbal and nonverbal communication deficiencies
Restricted and repetitive patterns of behavior, interests, and activities
Imitation skill deficiencies
Uneven cognitive skills including a concrete learning style with impaired abstract thought
Variable levels of cognitive ability from mentally retarded to normal or above average
Diminished emotional reactivity to many (particularly socially salient) stimuli
Strong negative emotional reactivity to some lower-order stimuli or to variations in routine

Impaired joint attention and difficulties in flexibly disengaging and shifting attention
Deficient adaptive timing of motor behaviors
Subtle abnormalities during first year becoming more dramatic during years two and three

feature patterns. When a sufficiently good match occurs, the system locks into an attentive resonant state that drives the recognition learning process; hence the term *adaptive* resonance.

ART predicts that *all conscious events are resonant events*. The degree of match that is required for resonance and sustained attention to occur is set by a *vigilance* parameter. Vigilance controls whether a particular learned representation will be concrete or abstract. Low vigilance allows the learning of general and abstract recognition categories. High vigilance forces the learning of specific and concrete categories. In the limit of very high vigilance, the learned prototype may code an individual exemplar. An increase in vigilance after a predictive failure causes a reset of the currently active category, and enables the brain to search for, attend, and learn the specific features that caused the predictive failure.

The iSTART model predicted that the vigilance of various autistic individuals is abnormally high, thereby forcing concrete categorization and sharply focused attention; that is, *hypervigilance leads to hyperspecific learning*. This property cause problems with learning, cognition, and attention in the thalamo-cortical-hippocampal system.

Two sets of psychophysical experiments have reported data consistent with hypervigilant category learning in autism (Church et al., 2010; Vladusich et al., 2010).

Grossberg and Versace (2008) developed the SMART model to propose how visual stimuli may be processed by networks of spiking neurons in laminar cortical circuits as they interact with specific and nonspecific thalamic nuclei to learn recognition categories. This model proposes how vigilance may be controlled by mismatch-activated cells in the nonspecific thalamus, which activate the nucleus basalis of Meynert, whose output signals release acetylcholine (ACh) broadly through the cortex, thereby raising vigilance. Consistent data by Weinberger, Miasnikov, and Chen (2006) shows that the specificity of auditory associative memory covaries with the amount of nucleus basalis stimulation. Palma, Grossberg, and Versace (2012) summarize additional supportive data. Perry et al. (2001) have reported abnormalities in cholinergic processing by the nucleus basalis in autistic individuals. ACh may also modulate saccade selection in the superior colliculus (Winkowski and Knudsen, 2008), which has been modeled by ART (Grossberg et al., 1996). Thus, attention, recognition, and movement control may all be impaired by this mechanism during autism.

CogEM extends ART to learning of cognitive-emotional associations that link external objects and events to the internal emotions that give them value. These emotions also activate motivational pathways that energize actions aimed at attaining valued goals. Resonance within CogEM circuits occurs between sensory/cognitive representations of what is possible and emotional representations of what is valued, and focuses attention upon objects and events that promise to satisfy emotional needs. CogEM model clarifies thalamo-cortico-amygdala interactions.

The emotional representations are proposed to be organized into opponent affects, such as fear vs. relief. These opponent affective circuits are energized by internal sources of arousal. Under normal circumstances, arousal is set at an intermediate level, or Golden Mean, during wakefulness. Either underarousal or overarousal can cause abnormal emotional reactions and, with them, abnormal cognitive-emotional learning. In particular, there is *an Inverted-U* in emotional reactivity in these opponent circuits: If the emotional center is overaroused, then the threshold to activate an emotion is abnormally low, but the intensity of emotion is abnormally small. If the emotional circuits are underaroused, then the threshold for activating an emotion is abnormally high but, when this threshold is exceeded, the emotional response is hyperreactive. The iSTART model proposes that various individuals with autism experience underaroused emotional depression, which helps to explain their baseline of reduced emotional expression, as well as their emotional outbursts in response to particular stimulus triggers.

The Spectral Timing model clarifies how the brain adaptively times responses in order to acquire rewards and other goal objects. Such adaptive timing is essential for all terrestrial animals, since rewards and other goals are often delayed in time relative to actions that are aimed at acquiring them. For example, if reward is usually delivered in ten seconds, how does the brain prevent us from getting frustrated and leaving the reward-predictive environment before that time elapses? The Spectral Timing model predicts how the brain distinguishes *expected non-occurrences* of rewards, which should not be allowed to interfere with acquiring a delayed goal, from *unexpected non-occurrences* of rewards, which can trigger the usual consequences of predictive failure, including reset of working memory, attention shifts, emotional rebounds, and exploratory behaviors. The Spectral Timing model proposes how various individuals with autism experience failures of adaptive timing, thus leading to premature release of behaviors in a context-inappropriate manner that can prevent these behaviors from being rewarded during social interactions. The Spectral Timing model clarifies thalamo-cortico-hippocampal-cerebellar-(basal ganglia) interactions.

The next section reviews these models in greater detail.

3. Adaptive Resonance Theory

3.1. Perceptual and Cognitive Learning, Expectation, Attention, and Fantasy

ART proposes a solution to the *stability-plasticity dilemma*; namely, how the brain can learn quickly throughout life without being forced to unselectively forget previously learned memories just as quickly (Grossberg, 1980, 1999b). This problem has also been called the *catastrophic forgetting* problem (Carpenter, 2001; French, 1999; Page, 2000). ART proposes how normal learning and memory may be stabilized through the use of learned top-down expectations. (Figure 1a) In other words, we are "intentional" beings so that we can learn quickly without suffering catastrophic forgetting. Top-down expectations have been predicted to operate at multiple cortical and thalamic levels, including top-down expectations from higher cortical areas, such as from prefrontal to inferotemporal cortex, and also at lower cortical areas, such as from striate visual cortex to the lateral geniculate nucleus (Gove, Grossberg, and Mingolla, 1995; Grossberg, 1999b, 2003a; Raizada and Grossberg, 2003).

Top-down expectations learn prototypes that are capable of focusing attention (Figure 1b) upon the combinations of features that comprise conscious perceptual experiences. When top-down expectations are active in a priming situation in the absence

of bottom-up information, they can modulate or sensitize their target cells to respond more effectively to future bottom-up information that matches the prototype. Such expectations cannot, however, fully activate these target cells under most circumstances. When bottom-up inputs do occur, an active top-down expectation selects the cells whose input features are consistent with the active prototype, and suppresses those that are not, thereby generating an attentional focus on the combinations of features that are expected. This matching and attentional process can synchronize and amplify the activities of selected cells, leading to a context-sensitive "resonance". Attentionally relevant stimuli are selected for learning, while irrelevant stimuli are suppressed and hence prevented from destabilizing existing representations.

Thus, ART solves the the stability-plasticity dilemma by allowing neural representations to be rapidly modified only by incoming stimuli with which they form a sufficiently close match. If the match is close enough, then learning occurs. Precisely because the match is sufficiently close, this learning fine-tunes an existing representation, rather than radically overwriting it. Matching gets started by initially endowing the top-down matching pathways with broadly distributed adaptive weights, so that a newly chosen category can match whatever feature pattern activated it. Learning prunes these weights and makes the prototype more selective.

Top-down expectations and attention achieve these matching properties using top-down on-center off-surround networks (Figure 1c). A balance between top-down excitation and inhibition in the on-center enables attention to modulate, but not fully activate, on-center cell responses to bottom-up inputs, while cells are strongly inhibited in the off-surround. Such a matching process has been mathematically proved necessary to stabilize memories of learned representations in response to a complex input environment (Carpenter and Grossberg, 1991). These circuits realize the ART Matching Rule. The LAMINART model predicts how identified cells within laminar neocortical circuits carry out the ART Matching Rule (Grossberg, 1999a, 2003a; Raizada and Grossberg, 2003): top-down signals from layer 6 of a higher cortical area reach layer 6 of a lower cortical area, where they are relayed to layer 4 of that area via modulatory on-center off-surround interactions. The term "biased competition" (Desimone, 1998; Kastner and Ungerleider, 2001) heuristically summarizes that attention biases off-surround competition.

The ART model predicts how the brain exploits the modulatory on-center to support internal fantasy, imagery, and planning (Grossberg, 2000a). In particular, phasic *volitional signals* from the basal ganglia can shift the balance in favor of excitation in the modulatory on-center (Figure 1c), thereby enabling top-down expectations, in the absence of supportive bottom-up inputs, to cause conscious experiences of imagery and inner speech. If these volitional signals become tonically hyperactive during a mental disorder, top-down expectations cause hallucinations.

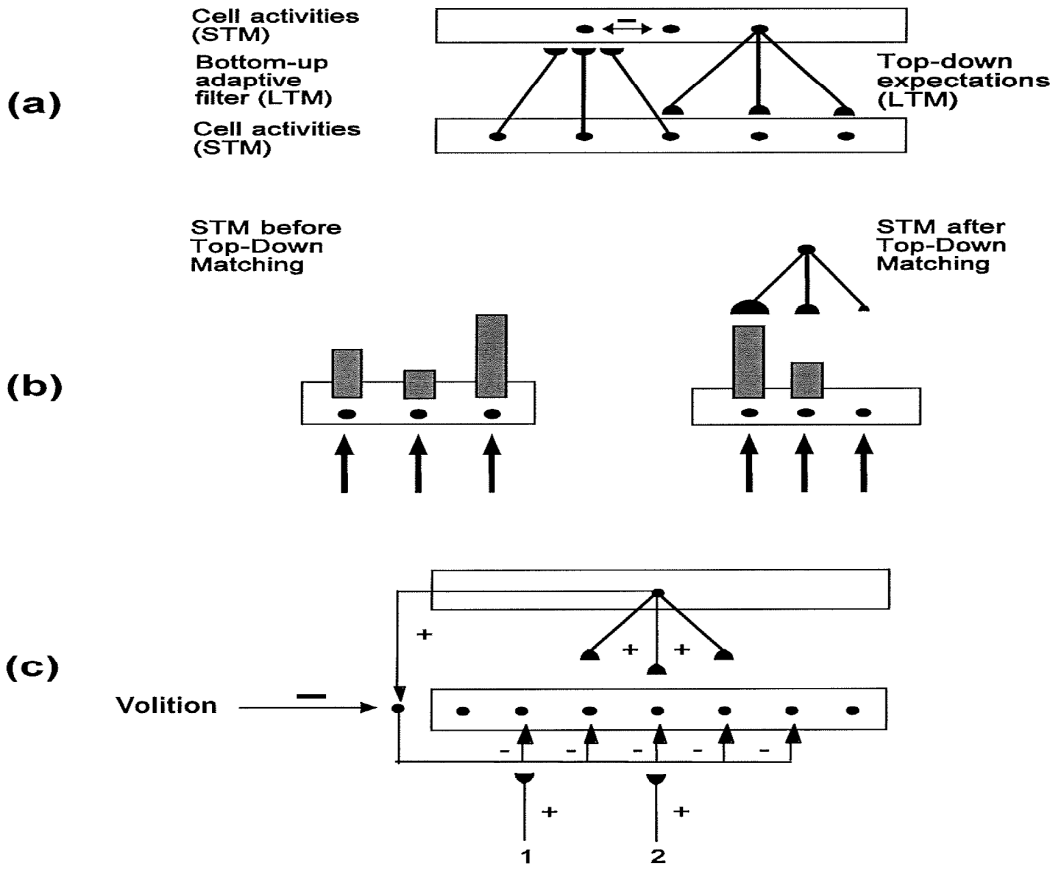


Figure 1. Data-driven bottom-up and attentive top-down interactions between short-term and long-term memories. (a) Patterns of activation, or short-term memory (STM), across feature-selective cells at a lower processing level send signals via bottom-up pathways to a higher processing level. Cells at the higher level respond selectively to prescribed combinations of features at the lower level. For example, such cells may represent recognition categories, as in inferotemporal cortex. The selective activation of category cells is achieved by multiplying the bottom-up signals with adaptive weights, or learned long-term memory (LTM) traces at the ends of the bottom-up pathways, before these learning-gated signals activate target category cells. These category cells compete among themselves to select a small number of winning cells. The combination of bottom-up adaptive filtering and competition are the basic ones for defining a self-organizing map. The active category cells, in turn, activate top-down pathways that read-out learned expectations via their own LTM traces. These top-down expectations are matched against the STM pattern that is active at the lower featural level. (b) This matching process confirms, synchronizes, and amplifies STM activities of features that are supported by large LTM traces in an active top-down expectation, and suppresses STM activities of features that do not get top-down support. The size of the hemidisks at the end of the top-down pathways represents the strength of the learned LTM trace that is stored in that pathway. (c) The ART Matching Rule may be realized by a modulatory top-down on-center off-surround network. In particular, bottom-up inputs, such as in pathways 1 and 2, can activate their feature-selective cells when no top-down expectation is active. When a

top-down expectation is active whose prototype (the learned on-center with excitatory pathways) does not include the feature activated by pathway 1, then the top-down off-surround cancels the bottom-up input, thereby suppressing activation of that feature. Since the feature that is activated by pathway 2 is included in the top-down prototype, the top-down excitation and inhibition approximately cancel (typically, with a small positive priming bias), so that activation of the corresponding feature-selective cell is preserved, synchronized, and even amplified. [Reprinted with permission from Grossberg (1999b).]

3.2. How Vigilance May Control the Generality of Knowledge

What information is bound together into object or event representations? ART proposes how the brain learns to attend to *critical feature patterns*, or combinations of relevant features, that are sufficient to achieve predictive success. Critical feature patterns replace the prototypes of classical recognition models. They may be incrementally learned through time from sequences of experienced exemplars with or without external supervision.

As mentioned above, the generality of learned prototypes is determined by the network's *vigilance* parameter, which is controlled by environmental feedback or internal volition. ART proposes how, in a normal brain, vigilance can track the demands of a particular environment, creating specific or general categories as needed to solve environmental problems; see below.

Spitzer, Desimone, and Moran (1988) have reported neurophysiological data from monkey inferotemporal cortex that are consistent with the predicted link between vigilance control, top-down matching, and attention. These authors write that "In the difficult condition the animals adopted a stricter internal criterion for discriminating matching from non-matching stimuli. The animal's internal representations of the stimuli were better separated…increased effort appeared to cause enhancement of the responses and sharpened selectivity for attended stimuli."

If vigilance is fixed through time at an abnormally high level, the system would be "hypervigilant," and environmental events would be classified concretely, with learned categories coding exemplar-like information. The iSTART model predicts that many individuals with autism have their vigilance fixed at a high level.

Other mental disorders also seem to include defective vigilance control. Carpenter and Grossberg (1993) predicted how a lesion of the hippocampus, which includes part of the ART orienting system (Figure 2), creates permanently low vigilance. The model then exhibits symptoms of medial temporal amnesia.

The flexible control of vigilance is thus a topic that warrants more neurobiological investigation.

3.3. Learning, Attention, Memory Search, Hypervigilance, and Hyperspecificity

Figure 2 summarizes the ART proposal of how an *attentional system* and an *orienting system* normally work together to discover and learn effective recognition categories without experiencing catastrophic forgetting.

Vigilance regulates the matching process that takes place between an exemplar input and the top-down expectation that is read out by an active recognition category. A sufficiently bad mismatch resets an active category and initiates a memory search, or hypothesis testing, cycle that can lead to the selection of a new category. Such a

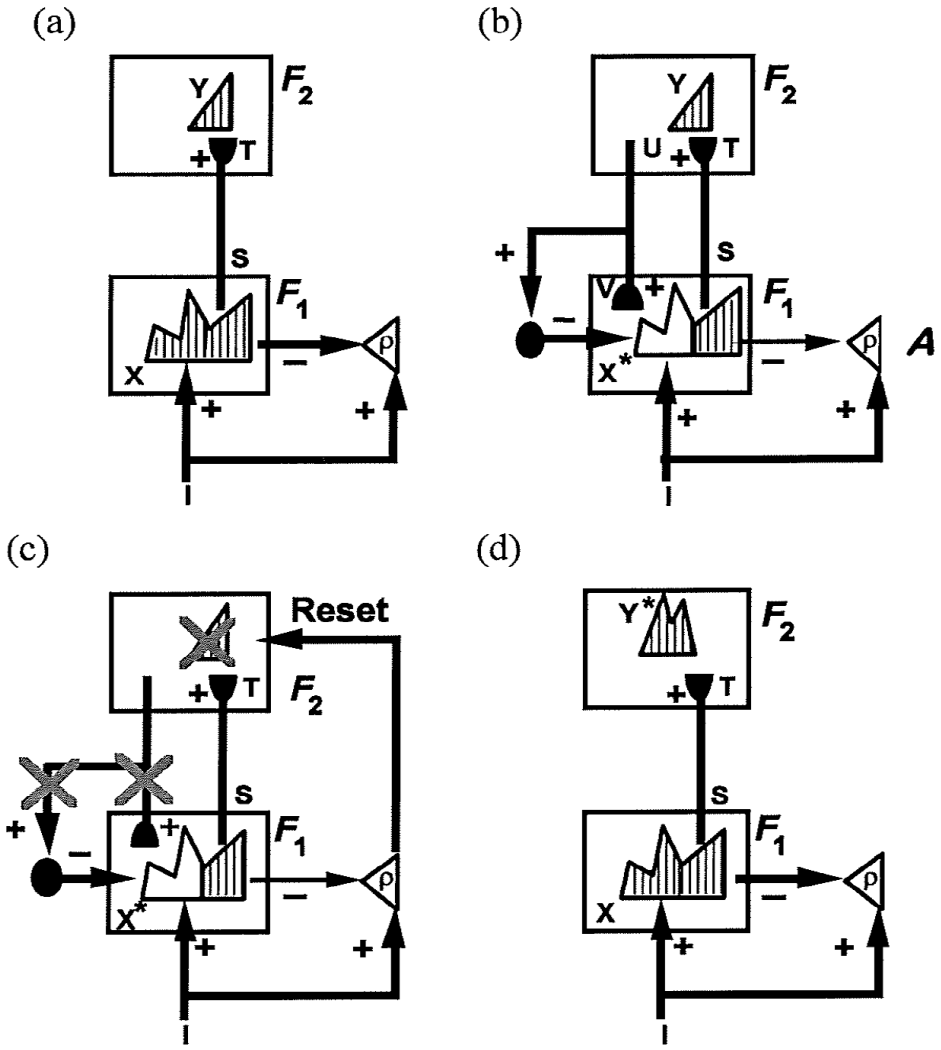


Figure 2. Search for and learning of a recognition code within Adaptive Resonance Theory. (a) The input pattern I is instated across the feature detectors at level F_1 as a short term memory (STM) activity pattern X. Input I also nonspecifically activates the orienting system A. STM pattern X is represented by the gray pattern across F_1. Pattern X both inhibits A and generates the output pattern S. Pattern S is multiplied by long term memory (LTM) traces, or learned adaptive weights. These LTM-gated signals are added at F_2 nodes to form the input pattern T, which activates the contrast-enhanced STM pattern Y across the recognition categories coded at level F_2. (b) Pattern Y generates the top-down output pattern U which is multiplied by top-down LTM traces and added at F_1 nodes to form the prototype pattern V that encodes the learned expectation of the active F_2 nodes. If V mismatches I at F_1, then a new STM activity pattern X^* is generated at F_1. X^* is represented by the gray pattern. It includes the features of I that are confirmed by the top-down expectation V. Mismatched features are inhibited. The inactivated nodes

corresponding to unconfirmed features of X are unhatched. The reduction in total STM activity which occurs when X is transformed into X^* causes a decrease in the total inhibition from F_1 to A. (c) If inhibition decreases sufficiently, A releases a nonspecific arousal wave to F_2, which resets the categorical STM pattern Y at F_2. (d) After Y is inhibited, its top-down prototype signal is eliminated, and activity pattern X can be reinstated at F_1. Enduring traces of the prior reset lead X to activate a different STM pattern Y^* at F_2. If the top-down prototype due to Y^* also mismatches I at F_1, then the search for an F_2 code continues until a more appropriate F_2 representation is selected. Then an attentive resonance develops and learning of the attended data is initiated. [Adapted with permission from Carpenter and Grossberg (1993).]

mismatch can occur, say, because the exemplar input represents an unfamiliar type of experience. A mismatch within the attentional system activates the complementary orienting system. Novelty-activated nonspecific arousal signals from the orienting system rapidly reset the recognition category within the attentional system that has been reading out the poorly matching top-down expectation (Figures 2b and 2c). The cause of the mismatch is hereby removed, thereby freeing the system to activate a different recognition category (Figure 2d), and the cycle continues. If no matching category exists, say because the bottom-up input represents a novel experience, then the search process automatically activates an as yet uncommitted cell population with which to represent the novel information. Event-related potential data from humans (Banquet and Grossberg, 1987) and neurophysiological data from monkeys (Miller, Li, and Desimone. 1991) support predicted properties of ART search.

ART proposes how vigilance can be adjusted up and down within the orienting system to learn more specific or general information, respectively, in response to predictive failures within each environment. Every increase in vigilance implies that a more specific category will be learned. If a predictive failure causes vigilance to increase to the smallest value that can correct a predictive error, then the most general categories can be learned that can eliminate predictive errors. Such *match tracking* clarifies how the brain can learn categories sensitive to the statistical structure of each situation, thereby enabling some cells in inferotemporal cortex learn to code specific information (e.g., a view of a familiar face), whereas other cells learn to code more general information.

If vigilance gets "stuck" at a high level, then concrete, hyperspecific learning will ensue in both in the bottom-up adaptive filtering pathways that define a recognition category and in the top-down prototypes that focus attention. Hyperspecific prototypes will mismatch and reset attention in response to even small environmental variations.

3.4. All Conscious States are Resonant States

As noted in Figure 2, if the top-down expectation is close enough to the bottom-up input pattern, then the pattern X^* of attended features reactivates the category Y which, in turn, reactivates X^*. The network hereby locks into a resonant state through a positive feedback loop that dynamically links, or binds, the attended features across X^* with their category, or symbol, Y. The resonance between distributed feature patterns and recognition categories converts the *pattern* of attended features into a coherent context-sensitive state that is linked to its category through feedback. It is this coherent state, that joins together distributed features and symbolic categories into a unified bound state,

which can enter consciousness. ART hereby predicts that *all conscious states are resonant states*.

4. Cognitive-Emotional-Motor Model

4.1. Three Types of Representations and Learning

The CogEM model (Grossberg, 1982a, 1984a, 2000b) proposes how emotional centers of the brain, such as the amygdala, interact with sensory and prefrontal cortices — notably ventral, or orbital, prefrontal cortex — to generate affective states, attend to motivationally salient sensory events, and elicit motivated behaviors (Figures 3 and 4). Experiments support the predicted role of these amygdala and orbitofrontal interactions (Baxter *et al*. 2000; Schoenbaum *et al*., 2003). Activating the feedback loop between cognitive and emotional centers generates a cognitive-emotional resonance that support conscious awareness of events and how we feel about them; that is, core consciousness (Damasio, 2000).

Figure 3a summarizes the hypothesis that (at least) three types of internal representation interact during reinforcement learning: sensory and cognitive representations S, drive representations D, and motor representations M. Sensory representations S temporarily store internal representations of sensory events in working memory. Drive representations D are sites where reinforcing and homeostatic, or drive, cues converge to activate emotional responses. Motor representations M control the read-out of actions.

The S representations are thalamocortical representations of external events, including the ART recognition categories that are learned by inferotemporal and prefrontal cortical interactions. Sensory representations temporarily store internal representations of sensory events in short-term memory via recurrent on-center off-surround networks; see Figure 3. The D representations include hypothalamic and amygdala circuits at which reinforcing and homeostatic, or drive, cues converge to generate emotional reactions and motivational decisions. The M representations include cortical and cerebellar circuits that control discrete adaptive responses.

Three types of learning take place among these representations: Conditioned reinforcer learning (CRL) enables sensory events to activate emotional reactions at drive representations. Incentive motivational learning (IML) enables emotions to generate a motivational set that biases the system to process cognitive information consistent with that emotion. Motor learning allows sensory and cognitive representations to generate actions.

In particular, learning within the S $\rightarrow$ D conditioned reinforcer pathways converts a CS into a reinforcer when activation of its sensory representation S occurs just before the drive representation D is activated by an unconditioned stimulus (US), or other previously conditioned reinforcer CSs. The ability of the CS to subsequently activate D via this learned pathway is a key property of a conditioned reinforcer. As these S $\rightarrow$ D associations are learned, incentive motivational learning within the D $\rightarrow$ S incentive motivational pathways also occurs, also due to pairing CS and US. Incentive motivational learning enables an active drive representation D to prime, or modulate, the sensory representations S of all cues, including the CSs, that have consistently been correlated with it. That is how activating D generates a "motivational set": it primes all of the sensory and cognitive representations that have been associated with that drive in the past. These incentive motivational signals are a type of motivationally-biased attention.

The S → M motor, or habit, learning enables the sensorimotor maps, vectors, and gains

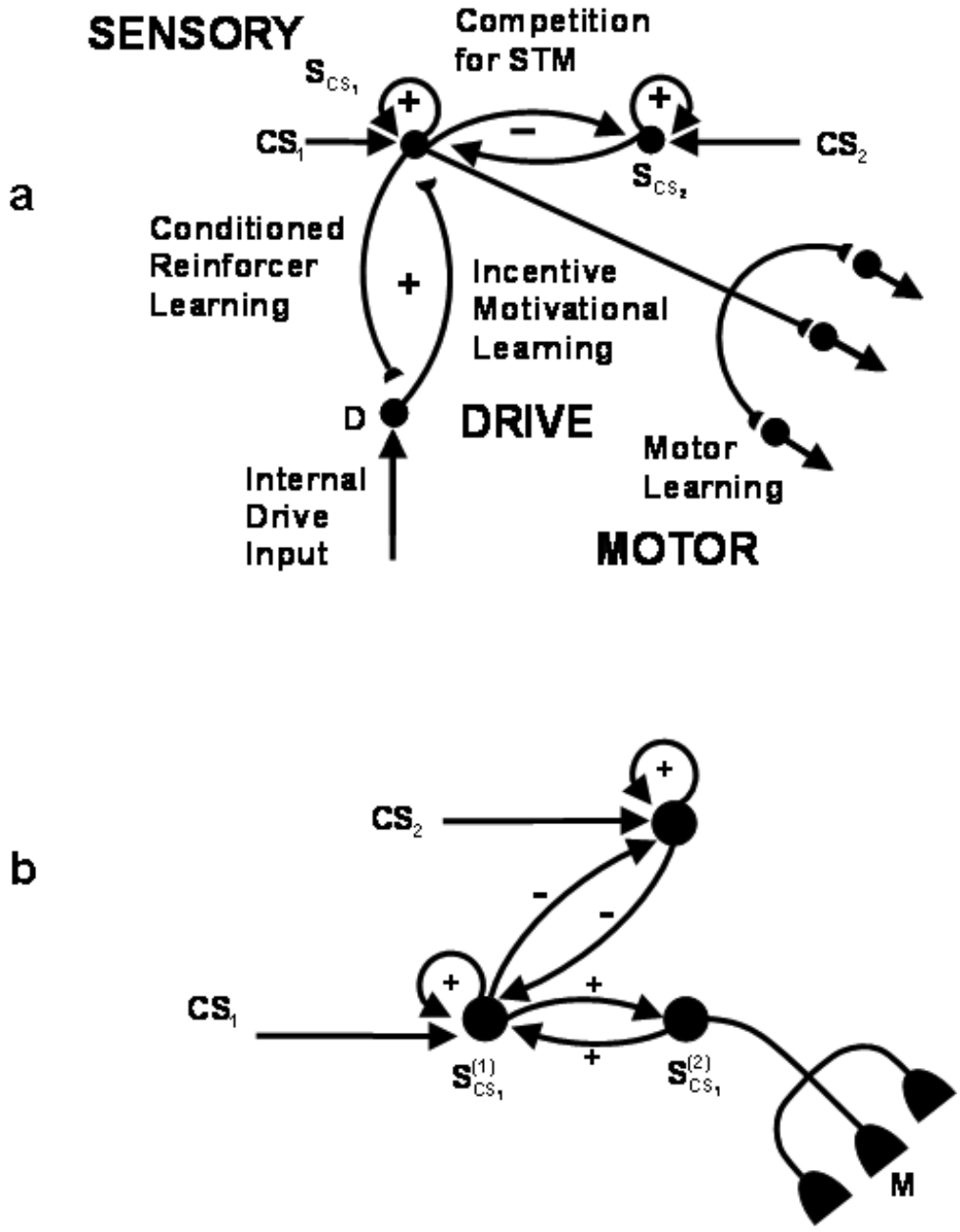


Figure 3. Macrocircuit of the Cognitive-Emotional-Motor model. (a) The simplest CogEM model: Three types of interacting representations (sensory, drive, and motor) that control three types of learning (conditioned reinforcer, incentive motivational, and motor) help to explain many reinforcement learning data. Sensory representations *S* temporarily store internal representations of sensory events in working memory. Drive

representations *D* are sites where reinforcing and homeostatic, or drive, cues converge to activate emotional responses. Motor representations *M* control the read-out of actions. Conditioned reinforcer learning enables sensory events to activate emotional reactions at drive representations. Incentive motivational learning enables emotions to generate a motivational set that biases the system to process information consistent with that emotion. Motor learning allows sensory and cognitive representations to generate actions. (b) In order to work well, a sensory representation *S* must have (at least) two successive stages, $S^{(1)}$ and $S^{(2)}$, so that sensory events cannot release actions that are motivationally inappropriate. See text for details. [Reprinted with permission from Grossberg and Seidman (2006).]

that are involved in sensory-motor control to be adaptively calibrated, thereby enabling a CS to read-out correctly calibrated movements.

Taken together, these processes control the learning and recognition of sensory and cognitive memories, which are often classified as part of a "declarative memory" system; and the performance of learned motor skills, which are often classified as part of a "procedural memory" system.

4.2. Multiple Sensory Stages Control Motivated Behaviors

To generate only motivationally appropriate behaviors, the circuit in Figure 3a needs to have two successive sensory processing stages, one in a sensory cortex and the other in a prefrontal cortex to which it projects (Figure 3b). These stages then release motivated behaviors only if both sensory and motivational support for those behaviors is present. In particular, each drive representation D obeys a *polyvalent* constraint whereby it can generate large incentive motivational output signals to sensory representations S only if it gets a sufficiently large primary or conditioned reinforcer input at the same time that it gets a sufficiently large internal drive input. The internal drive input designates whether an internal drive, such as hunger, thirst, or sex, is high and in need of satisfaction. Different drive representations exist to represent these distinct internal homeostatic states. The polyvalent constraint means that a drive representation cannot fire vigorously unless it simultaneously receives a sufficiently large external sensory input and internal drive input. Due to this polyvalent constraint, an external reinforcing cue does not activate strong incentive motivation, and with it action, to satisfy a drive that is already satisfied, because the drive input is too small.

A polyvalent constraint on prefrontal cortical cell firing prevents these cells from triggering an action unless they simultaneously receive sensory input from the corresponding sensory cortex and incentive motivational input from a drive representation. For example, presentation of a given cue, or CS, activates the first stage of its sensory representation (in sensory cortex); see $S_{CS}^{(1)}$ in Figure 3b. This activation is stored in short-term memory using positive feedback pathways from the sensory representation to itself. The stored activity generates output signals to all the drive representations with which the sensory representation is linked, as well as to the second stage $S_{CS}^{(2)}$ of the sensory representation (in prefrontal cortex; see Figure 3b). The second stage of the sensory representation obeys a polyvalent constraint: It cannot fire while the CS is stored in short-term memory unless it receives converging signals from the first

sensory stage and from a drive representation, thereby preventing motivationally-inappropriate release of actions.

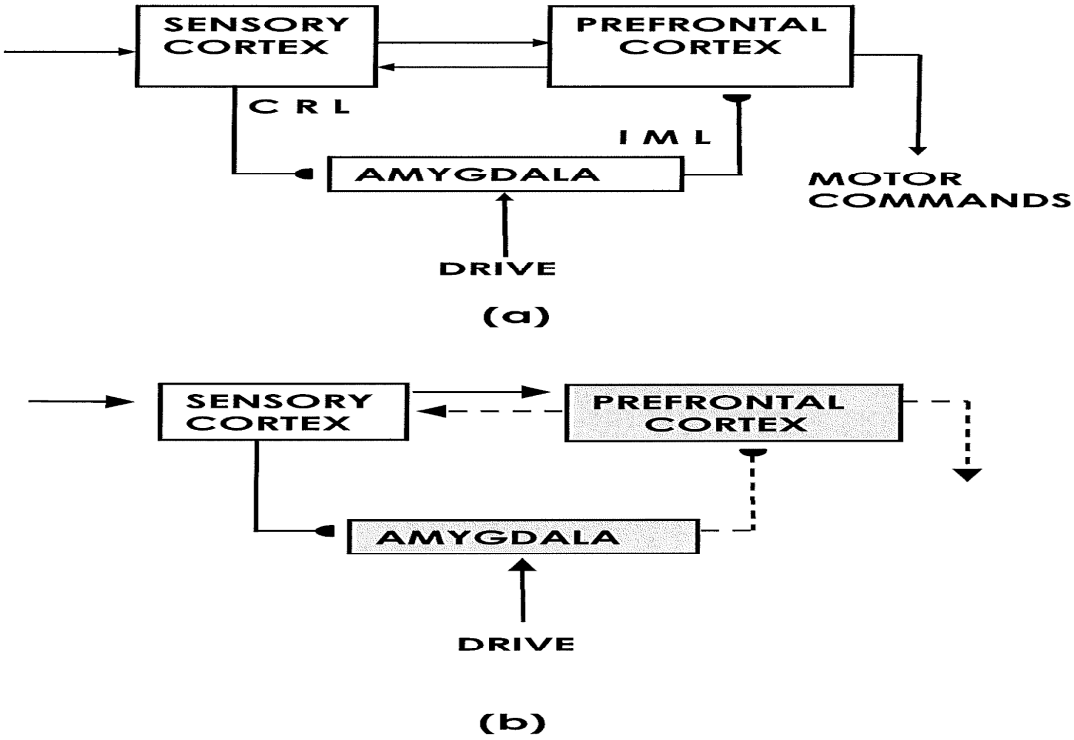


Figure 4. Anatomical interpretation of CogEM model. The two successive stages of a sensory representation S are interpreted to be in the appropriate sensory cortex (corresponds to $S^{(1)}$ in Figure 3b) and the prefrontal cortex, notably the orbitofrontal cortex (corresponds to $S^{(2)}$ in Figure 3b). The prefrontal stage requires motivational support from a drive representation D to be fully effective, in the form of feedback from the incentive motivational learning (IML) pathway. The amygdala is interpreted as one important part of a drive representation. Amydgala inputs to prefrontal cortex cause feedback to sensory cortex that selectively amplifies and focuses attention upon motivationally relevant sensory events. (b) When a drive representation like the amygdala gets depressed (gray box), diminished activation of its outputs in response to sensory events depresses motivational inputs to the prefrontal cortex in response to emotionally important events, and hereby attenuates motivationally-appropriate signals to and from the prefrontal cortex (dashed lines). As a result, motivationally irrelevant events are not attentionally suppressed, and prefrontally-mediated plans and actions are insufficently activated. [Reprinted with permission from Grossberg and Seidman (2006).]

4.3. Motivated Attention and Blocking

Positive feedback from the prefrontal cortex to its sensory cortex also exists; see Figures 3b and 4a. Such positive feedback can stabilize memories (Figure 2), direct attention to motivationally salient sensory events, and select motivationally appropriate responses. Attentional blocking can hereby occur, whereby sensory representations of unattended

and irrelevant sensory cues can be suppressed and thereby do not learn (Grossberg and Levine, 1987; Pavlov, 1927).

How does such cortical feedback enable attentional blocking to occur? As noted above, a model prefrontal cortical cell can fire only if a drive representation with which it is associated receives strong incentive motivational inputs from a drive representation. This positive feedback from the prefrontal cortex to the sensory cortex amplifies only active sensory representations that are motivationally consistent. This amplification enables these sensory representations to attentionally block less salient representations via recurrent lateral inhibitory connections among the sensory representations that are part of recurrent on-center off-surround networks which compete for stored activation without a loss of contrast sensitivity (Grossberg, 1973, 1980, 1982a).

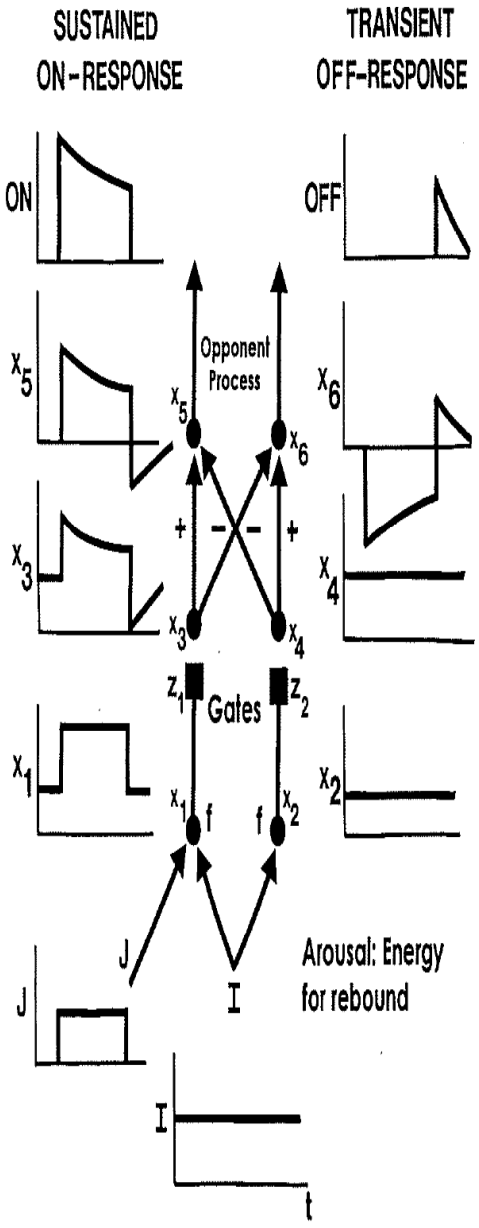
4.4. Sensory-Amygdala-Orbitofrontal Interactions during Motivated Behaviors

The circuit in Figure 4a is consistent with data from Barbas (1995), who noted that several types of sensory cortex, including visual, somatosensory, auditory, gustatory, and olfactory cortex, are connected to both the amygdala and prefrontal cortex, and that the amygdala also sends a strong projection to the prefrontal cortex. Cells in the orbitofrontal cortex hereby become sensitive to the reward associations of sensory cues, as well as to the level of drive satiation (Mishkin and Aggleton, 1981; Rolls, 1998). Both orbitofrontal cortex and amygdala help to select responses based on their emotional valence and success in achieving rewards (Damasio *et al.*, 1991; Passingham, 1997).

When a drive representation like the amygdala cannot fire at normal levels, as in Figure 4b (gray box), its diminished outputs depress motivational inputs to the prefrontal cortex in response to emotionally important events. The polyvalent constraint at the prefrontal cortex then prevents prefrontal cortex from firing adequately. As a result, prefrontally-mediated plans and actions are insufficiently activated, and motivationally irrelevant events are not attentionally suppressed. In short, Theory of Mind fails; see Section 4.7.

How can outputs from a drive representation become depressed? This can occur because emotional centers are often organized into *opponent* affective processes, such as fear and relief. These opponent-processing emotional circuits are called *gated dipoles* (Figure 5; Grossberg, 1984b, 2000b). Their response amplitude and sensitivity to external and internal inputs are calibrated by an arousal level and chemical transmitters that slowly inactivate, or habituate, in an activity-dependent way. The name "gated dipole" summarizes that habituative transmitters "gate," or multiply, signal processing in the opponent "dipole."

An Inverted-U occurs whereby dipole outputs become depressed if the arousal level is chosen too large or too small. Underaroused and overaroused depression can be distinguished clinically by their parametric properties; see below. The iSTART model proposes that some symptoms of autism are due to underaroused depression and the way in which this condition interacts with other circuits, notably cognitive and motor circuits. In particular, if the amygdala experiences underaroused depression, then this deficiency can ramify throughout the brain in the manner summarized in Figure 4b.


SUSTAINED
ON-RESPONSE
TRANSIENT
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Opponent
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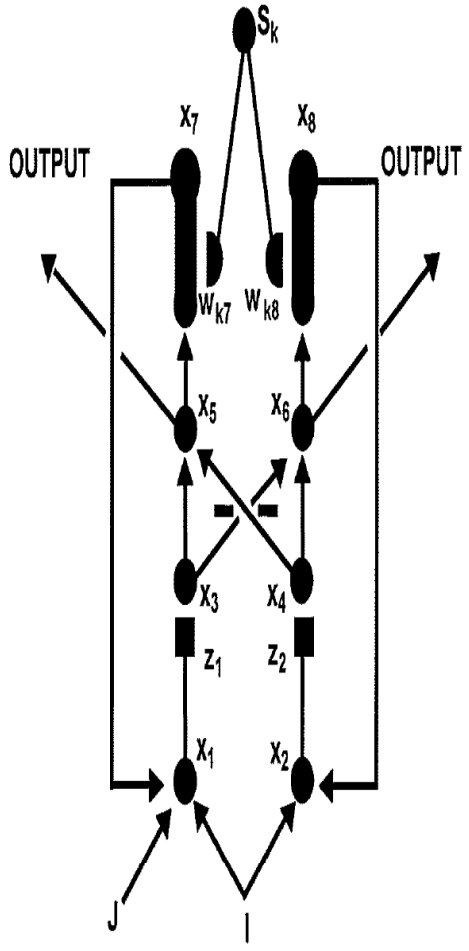
(a)


OUTPUT
OUTPUT

(b)

Figure 5. Gated dipole opponent process. (a) A gated dipole opponent process can generate habituative ON responses and transient OFF rebounds in response to the phasic onset and offset, respectively, of the input J to its ON channel. Term I delivers tonically active nonspecific arousal that energizes antagonistic rebounds when the phasic input J shuts off. Terms z_1 and z_2 are the habituative transmitter gates, or depressing synapses, in the ON and OFF channels; see the square synaptic symbols. They multiply, or gate, signals $f(x_1)$ and $f(x_2)$ that are derived from the ON and OFF activities x_1 and x_2, respectively, before the gated signals $f(x_1)z_1$ and $f(x_2)z_2$ excite the activities x_3 and x_4, respectively. The habituative gates convert the step-on-baseline activity pattern x_1 into the overshoot-habituation-undershoot-habituation pattern at activity x_3. Next, the opponent interaction works; namely, the baseline activity x_4 in the OFF channel due to the arousal I is subtracted from the habituative ON activity x_3 to yield x_5. When activity x_5 is thresholded to generate an ON output signal, it has an initial overshoot of activation, followed by habituation. When the signs of excitation and inhibition are reversed in the OFF channel, the activity x_6 is caused. The antagonistic rebound in the OFF output is generated by thresholding x_6. The antagonistic rebound is thus derived from the mirror-image of excitation and inhibition of the undershoot-habituation part of the ON channel activity at x_5. [Reprinted with permission from Grossberg (2000b).]; (b) A READ (Recurrent Associative Dipole) circuit. The READ circuit is a gated dipole with excitatory feedback pathways between activities x_7 and x_1, and activities x_8 and x_2. Feedback enables the READ circuit to maintain a stable motivational baseline to support an ongoing motivated behavior. A sensory representation S_k sends conditionable signals to the READ circuit that are gated by conditioned reinforcer adaptive weights, or long term memory traces, w_{k7} and w_{k8} to the ON and OFF channels, respectively. Read-out of previously learned adaptive weights is dissociated from read-in of new values of the learned weights. New weight learning is generated by teaching signals from the ON or OFF channel that wins the opponent competition. The combination of recurrent feedback and associative dissociation enables the adaptive weights to avoid learning baseline noise, maintain sensitivity to the relative balance of ON and OFF channel conditioning through time, and preserve their learned memories until they are disconfirmed by new learning contingencies. [Reprinted with permission from Grossberg and Schmajuk (1987).]

4.5. Opponent Processing: Inverted-U, Rebound, and Attentional Perseveration

Opponent emotions are a special case of a more general brain design for opponent processing, including opponent perceptual features like red and green colors, or downward and upward motions, or horizontal and vertical orientations. All of these processes can generate *antagonistic rebounds* whereby, say, offset of a sustained fearful cue can elicit a wave of relief, or removal of a desired food can elicit a wave of frustration, or offset of a sustained red image can yield a green aftereffect, or offset of a sustained downward motion of water can yield an upward motion aftereffect, and so on. In all of these cases, an OFF channels can experience an antagonistic rebound when the ON channel shuts off. Opponent rebounds control ART reset and search, as discussed above, as they rebalance sensory, cognitive, emotional, and motoric representations in response to rapidly changing environmental inputs.

Gated dipoles exhibit a Golden Mean of optimal behavior at an intermediate arousal level: For larger or smaller levels of arousal, behavior deteriorates in different

ways, thereby giving rise to an Inverted-*U* in network performance as a function of the arousal level. In different parts of the brain, the arousal source may differ. In general, arousal is an input that is *tonically* on (or internally generated and active during an interval of fast phasic inputs) and received equally by dipole ON and OFF channels.

Both the Inverted-*U* and the antagonistic rebound result from *habituative transmitter gates*, or chemical transmitters that are released in an activity-dependent way faster than they can recover. These transmitters exist in the opponent channels, where they multiply, or gate, the signals on their way to the opponent, or competitive, processing stage. Due to the factors that are summarized in Figure 5a, when arousal is too small, an elevated response threshold occurs in the ON channel, since there is not enough tonic arousal to generate outputs in response to normal phasic input levels. Paradoxically, such an underaroused circuit also gives rise to *hyper*excitable, or larger than normal, responses to increments in the ON input that exceed this elevated threshold. This is true because the arousal input, when it is gated by the habituative transmitter, acts like a gain that divides cell activation. Division by a smaller arousal level causes larger suprathreshold cell activations. When arousal is too large, the opponent process experiences a low behavioral threshold, since there is plenty of tonic arousal to boost the effect of phasic inputs. Paradoxically, an overaroused dipole gives rise to *hypo*excitable, or smaller than normal, outputs, in response to increments in the ON input that exceed the reduced threshold, because an abnormally large arousal divides the cell activation. Due to the Inverted-*U*, a suitable pharmacological "up" can reduce the supra-threshold hypersensitivity of patients with underaroused dipole circuits, as may occur in some autistic individuals.

Figure 5a defines the simplest gated dipole using a feedforward circuit. In general, gated dipoles include feedback pathways in order to store their operating levels in short-term memory and maintain a steady motivational baseline while an action is carried out. Figure 5b illustrates a feedback gated dipole that is called a READ (REcurrent Associative Dipole) circuit (Grossberg and Schmajuk, 1987). A READ circuit dissociates the read-out of previously learned emotional memories from the read-in of new emotional memories. This property enables learning to remain sensitive to changing reward contingencies, and emotional memories to remain stable until they are actively erased by disconfirmed sensory or cognitive expectations.

Sensory and cognitive representations, as well as emotional representations, can be organized into opponent channels with habituative ON and OFF cells. ART illustrates how an unexpected event can trigger a burst, or sudden increment, of nonspecific arousal (see Figure 2c). When such an arousal burst is received on top of the baseline tonic arousal input of a normal dipole, it can cause an antagonistic rebound of activity in its OFF channel. In this way, sensory, cognitive, and emotional hypotheses that are represented by dipoles across the brain can be disconfirmed by an unexpected event, thereby resetting ongoing processing and causing a shift of attention.

In an underaroused dipole, by contrast, an unexpected event can cause a paradoxical amplification of activity in the ON channel of the dipole, instead of an antagonistic rebound in the OFF channel. If, however, an arousal burst is sufficiently strong, then an unusually intense antagonistic rebound can be caused.

4.6. Autistic Hypervigilant Cognitive Learning and Underaroused Emotion

Consider what happens when underaroused emotional and sensory opponent processing are combined with hypervigilant and hyperspecific category learning. That is, consider how an underaroused CogEM model interacts with a hypervigilant ART model. This combined system exhibits formal symptoms that are familiar in autism.

For example, suppose that positive affect usually motivates a particular learned action. An underaroused emotional dipole can exhibit paradoxical enhancement of its motivational ON channel when a nonspecific arousal burst is triggered by an unexpected event. How can such paradoxical enhancements be caused in an individual with autism? Imagine that such an individual inspects an object closely in slightly different ways. Hyperspecific top-down expectations may cause mismatches with the different views of the object. These mismatches can cause nonspecific arousal bursts, which can cause enhanced ON channel responses. In particular, enhanced positive affect in underaroused emotional dipoles can lead to enhanced storage of the object representation in sensory and cognitive dipoles. A persistent and self-reinforcing perseverative behavior can result, which might manifest itself in persistently inspecting the same object from slightly different perspectives.

Suppose, however, that an arousal burst is caused which is significantly larger, say due to a larger mismatch of a presently active hyperspecific category prototype with a very different and unexpected event. Then an unusually intense, negative antagonistic rebound can be caused. Thus, novel experiences can be highly aversive when hyperspecific categories mismatch them and generate a burst of arousal to underaroused emotional dipoles. These negative rebounds may be one reason why individuals with autism are prone to experiencing severe negative reactions to unanticipated events.

One coping strategy is to avoid the type of novelty that will cause unbearable negative rebounds and to perseverate on small details of the environment, leading to the autistic "need for sameness".

4.7. Impoverishment of Motivated Goals, Intentions, and Theory of Mind

A depressed response in the outputs of emotion-representing areas can cause flat affect, which may cause an inability to represent others' beliefs and intentions, since mental states that depend upon interpreting one's own emotional state, or the emotional states of others, can be diminished. Such a deficiency can cause major difficulties in social communication. In the CogEM model, emotionally charged sensory inputs, such as the expressions on other people's faces or their tone of voice, may be able to activate the sensory representations in temporal cortex, but not the emotional responses from amygdala and related emotion-representing circuits via conditioned reinforcer pathways.

A problem with the setting of motivationally directed goals and intentions can arise because the depressed response of emotional representations, in brain areas like the amygdala, depresses the incentive motivational signals that would normally activate the prefrontal cortex in response to motivationally salient events (Figure 4b). As a result, the prefrontal cortex will not be adequately activated, and a hypofrontal condition can emerge. Due to hypofrontality, the working memory representations and plans within the prefrontal cortex will be degraded, so social goals and plans will not form in a normal

fashion. Depressed affective responses to environmental and internally generated cues, combined with insufficient motivational support for emotionally-appropriate plans and actions, helps to explain why individuals with autism are said to be without a Theory of Mind.

5. Adaptive Timing Model

5.1. Adaptively Timed Learning, Motivation, Attention, and Action

The above discussion illustrates the dichotomy between declarative memory and procedural memory, knowing that and knowing how, memory and habit, or memory with record and memory without record (Bruner, 1969; Mishkin, 1982, 1993; Ryle, 1949; Squire and Cohen, 1984). The amnesic patient HM exemplified this distinction by learning and remembering motor skills like assembly of the Tower of Hanoi without being able to recall having done so (Bruner, 1969; Cohen and Squire, 1980; Mishkin, 1982; Ryle, 1949; Scoville and Milner, 1957; Squire and Cohen, 1984). HM's surgical lesion included extensive parts of the hippocampal formation and amygdala. Subsequent animal studies have shown that damage to the hippocampal formation (Ammon's horn, dentate gyrus, subiculum, fornix) and the parahippocampal region (entorhinal, perirhinal, and parahippocampal cortices) can reproduce analogous amnesic symptoms (Mishkin, 1978; Squire and Zola-Morgan, 1991). These results implicate this aggregate hippocampal system in the processes that regulate declarative memory, or "knowing that". Such processes support a competence for learning recognition categories and being able to flexibly access them in a task-specific way (Eichenbaum, Otto, and Cohen, 1994). The discussion of ART above is about declarative memory, particularly about the learning of recognition categories, and involves predicted interactions between cortical and hippocampal representations. As noted in Section 3.2, ART exhibits a constellation of formal symptoms that strikingly resemble symptoms of medial temporal amnesia when the hippocampal part of its orienting system is lesioned (Carpenter and Grossberg, 1993).

A parallel line of research implicates the cerebellum in the processing of procedural memory, or "knowing how". The cerebellum is an essential circuit for conditioning discrete adaptive responses during eye movements, arm movements, nictitating membrane movements, and jaw movements (Ebner and Bloedel, 1981; Gilbert and Thach, 1977; Ito, 1984; Lisberger, 1988; Optican and Robinson, 1980; Thompson, 1988; Thompson *et al*., 1984, 1987).

A key property of cerebellar learning is that it is *adaptively timed*, so that learned responses are emitted at times that are appropriate within the environmental constraints of the learning paradigm. Cognitive-emotional learning is also adaptively timed, so that motivated attention can be maintained on salient goal objects for the necessary amount of time to carry out goal-directed actions.

Many goal objects may be delayed subsequent to the actions that elicit them, or the environmental events that signal their subsequent arrival. Humans and many animal species can learn to wait for the anticipated arrival of a delayed goal object, even though its time of occurrence can vary from situation to situation. Such learned behavioral timing is important in the lives of animals which explore their environments for novel sources of gratification. On the one hand, if an animal or human could not inhibit its exploratory behavior, then it could starve to death by restlessly moving from place to place, unable to remain in one place long enough to obtain delayed rewards there, such as food. On the

other hand, if an animal inhibited its exploratory behavior for too long while waiting for an expected source of rewards, such as food, to materialize, then it could starve to death if food is not, after all, forthcoming.

Thus, the survival of a human or animal may depend on its ability to accurately time the delay of a goal object based upon its previous experiences in a given situation. It needs to balance between exploratory behavior, which may discover novel sources of reward, and consummatory behavior, which may acquire expected sources of reward. To effectively control this balance, the human or animal needs to suppress its exploratory behavior and focus its attention upon an expected source of reward at around the time that the expected delay transpires for acquiring this reward.

5.2. Adaptively Timed Gating of Mismatch-activated Hippocampal Cells

To illustrate this sort of timing, suppose that an animal typically receives food from a food magazine ten seconds after pushing a lever, and that the animal orients to the food magazine right after pushing the lever. When the animal inspects the food magazine, it perceives the non-occurrence of food during the subsequent ten seconds. These non-occurrences disconfirm the animal's sensory expectation of seeing food in the magazine. What spares the animal from erroneously reacting to these *expected non-occurrences* of food during the first two seconds as predictive failures? Why does the animal not immediately become so frustrated by the non-occurrence of food that it shifts its attentional focus and releases exploratory behavior aimed at finding food somewhere else? Alternatively, if the animal does wait, but food does not appear after ten seconds have elapsed, then why does the animal then react to the *unexpected non-occurrence* of food by becoming frustrated, resetting its working memory, shifting its attention, and releasing exploratory behavior?

The processing of registering sensory mismatches or matches is not itself inhibited: If the food appears earlier than expected, the animal can perceive and eat it. Instead, the effects of these sensory mismatches upon reinforcement, attention, and exploration are somehow inhibited, or gated off. This adaptively timed gating process enables attention and action to be appropriately timed to generate adaptive behavior in each environment.

For example, during classical conditioning, a conditioned stimulus (CS) such as a tone or light, when paired with an unconditioned stimulus (US) such as a shock, can learn to generate conditioned responses (CR), such as fear or limb withdrawal, that were originally elicited only by the US. Such learning is optimal at a range of positive interstimulus intervals (ISI) between the CS and US, and is attenuated at zero ISI and long ISIs (Smith, 1968). The hippocampal formation has been implicated in adaptively timed processing of such cognitive-emotional interactions. For example, Thompson *et al*. (1987) distinguished two types of learning that go on during conditioning of the rabbit Nictitating Membrane Response: Adaptively timed "conditioned fear" learning that is linked to the hippocampus, and adaptively timed "learning of the discrete adaptive response" that is linked to the cerebellum.

The START model synthesis of the ART and CogEM models proposes a unified explanation of why both the hippocampal system and the cerebellum may need adaptive timing circuits for their normal functioning (Figures 6 and 7). The START model predicts how motivational mechanisms within the amygdala can rapidly draw motivated attention

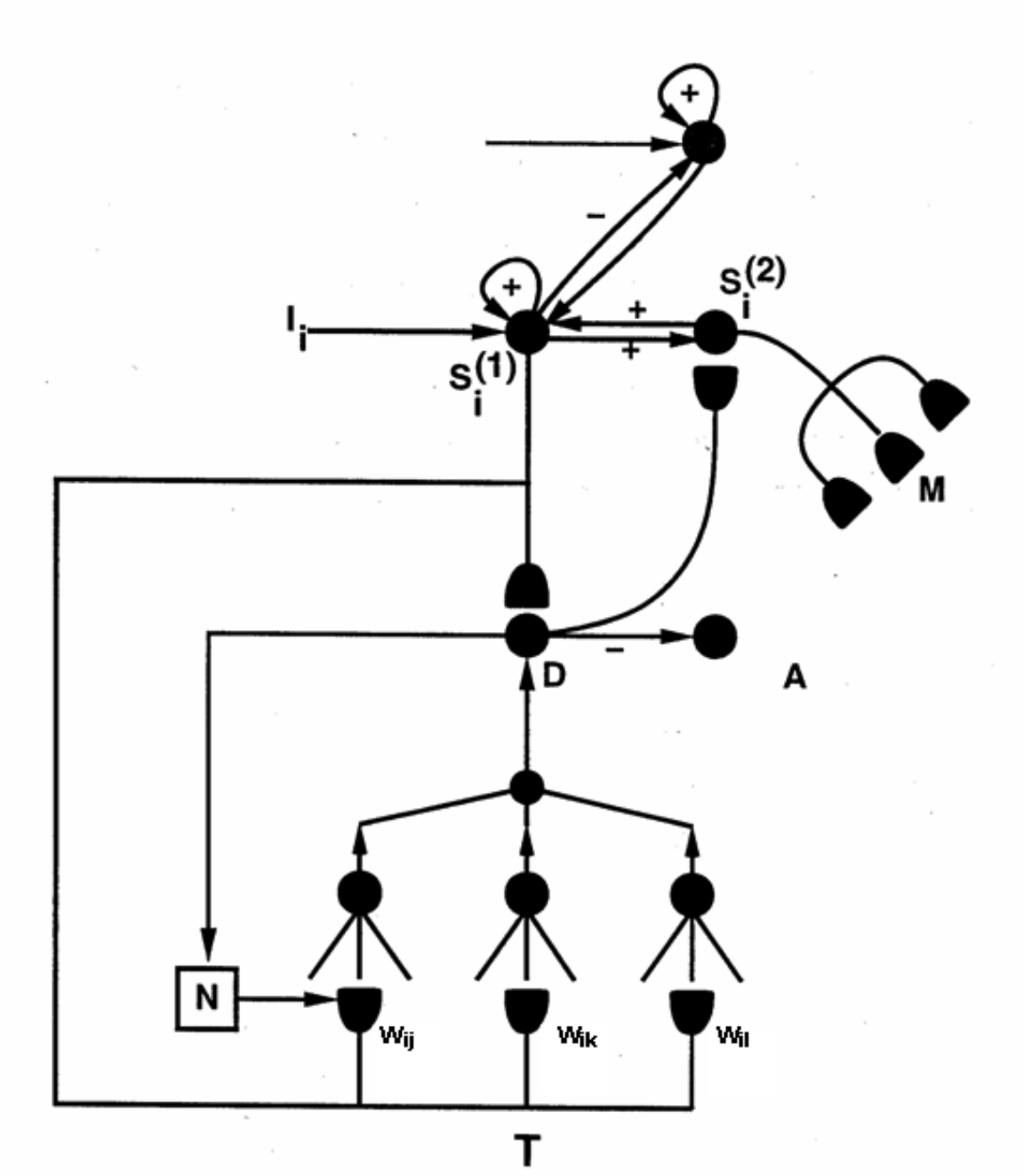


Figure 6. START model. Adaptively timed learning maintains motivated attention at the same time that it inhibits activation of the orienting system. See text for details. [Reprinted with permission from Grossberg and Merrill (1992).]

to salient cues via a cognitive-emotional resonance within CogEM feedback circuits between sensory representations *S* and drive representations *D* (see Figure 4). Once these sensory cue representations are selected and activated, what prevents the actions that they control from being prematurely released via the CogEM circuit?

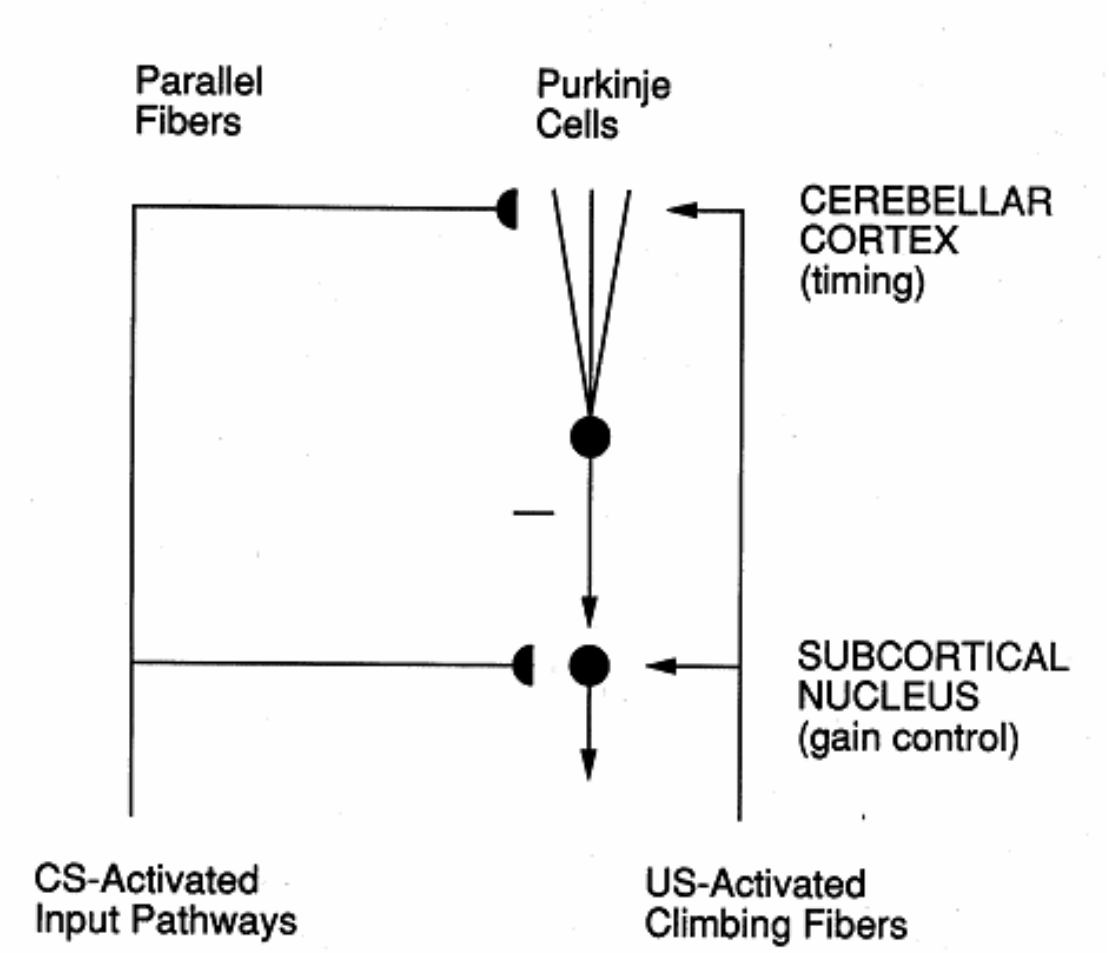


Figure 7. Adaptively timed cerebellar learning circuit. The cerebellum plays a role in the adaptively-timed control of learned actions. In particular, adaptively timed Long Term Depression at Purkinje cells depresses the level of tonic inhibitory firing of these cells to cerebellar nuclei, thereby disinhibiting nuclear cells and allowing them to expressed their learned gains to control actions in an adaptively timed way. See text for details. [Reprinted with permission from Grossberg and Merrill (1996).]

5.3. Spectral Timing in Cerebellum and Hippocampus and Metabotropic Substrate

Figure 7 summarizes a model of how the cerebellum adaptively times the release of motor commands by using a "spectrum" of learning sites that are each sensitive to a different range of delays between CS and US. A process of "spectrally timed learning"

selects that subset of sites whose reaction rates match the expected delays, or interstimulus intervals (ISIs), between the CS and the US. Learning at parallel fiber/Purkinje cell synapses depresses the tonically active output from cerebellar Purkinje cells to cerebellar nuclei. This Long Term Depression (LTD) occurs in an adaptively timed way. LTD hereby disinhibits the target cerebellar nucleus sites and allows the adaptively timed expression of learned gains from these sites when the US is expected.

In particular, suppose that a conditioned stimulus (CS), say via the motor output pathway *M* in Figure 4, activates pathways to both a subcortical cerebellar nucleus and to cerebellar cortex parallel fibers that synapse on Purkinje cells with a spectrum of differently timed intracellular processes. Unconditioned stimulus (US)-activated climbing fibers provide a teaching signal that also converges upon the parallel fiber/Purkinje cell synapses. This teaching signal causes the active synapses within the parallel fiber spectrum to become weaker (Long Term Depression) if they are activated by the CS when the US teaching signal becomes active. Synapses whose spectral activity does not overlap the climbing fiber signals become stronger (Long Term Potentiation, or LTP). Because the Purkinje cells tonically inhibit their subcortical target cells, their adaptively timed inhibition by the CS disinhibits the effect of tonic Purkinje cell outputs on cerebellar nuclear cells. In other words, a timed gate opens and allows the subcortical cells to fire. The model proposes that climbing fibers also control learning of adaptive gains along subcortical pathways through the nuclear cells. Thus, when the adaptively timed Purkinje cell gate opens, the learned gains can be expressed at the correct time and with the correct amplitude to cause a correctly calibrated motor response.

Fiala, Grossberg, and Bullock (1996) have modeled how cerebellar adaptive timing may use the metabotropic glutamate (mGluR) receptor system in adaptively timed learning. Hippocampus and basal ganglia may also use this biochemical mechanism.

Individuals with autism are known to have cerebellar deficiencies and also perform short-latency responses in the eye-blink paradigm. The Spectral Timing model prediction of a role for mGluR in adaptively timed learning raises the question of whether the mGluR system of some autistic individuals is dysfunctional.

Motivated attention needs to be maintained long enough for the adaptively timed action to be executed. Adaptively timed motivated attention can prevent irrelevant novel events from prematurely resetting thalamocortical sensory and cognitive representations as they actively read-out adaptively timed responses. The START model accomplishes this by showing how circuits within the hippocampus that are capable of adaptively timed learning can modulate the responses of ART and CogEM circuits.

In particular, as summarized in Section 3, the ART model proposes how attentional and orienting systems interact to categorize information and to develop resonant states if an active top-down prototype and a bottom-up sensory input form a sufficiently good match. If the mismatch is too big for resonance to occur, then other things being equal, the orienting system can trigger a search for a better category with which to categorize the information. The hippocampal system is proposed to be part of the orienting system that is activated by these mismatches and relays them as novelty-sensitive reset bursts to the thalamocortical system. Such an ART-mediated activation of the orienting system is not, however, sensitive to whether the novel event that caused the mismatch is relevant to the task. The START model clarifies how mismatches may be modulated by task-relevance in an adaptively timed way. In particular, the START model

suggests how motivationally salient cognitive representations may be enhanced, while orienting responses are inhibited, by an adaptively timed hippocampal dentate-CA3 circuit, during the same time intervals when conditioned responses are disinhibited by an adaptively timed cerebellar circuit.

In summary, as shown in Figure 6, the START model enables three key properties to simultaneously obtain:

1. *Fast Motivated Attention*. Rapid focusing of attention on motivationally salient cues occurs from regions like the amygdala to prefrontal cortex (the $D \rightarrow S^{(2)}$ pathway in Figure 6). Without further processing, fast activation of the CS-activated $S^{(2)}$ sensory representations could prematurely release motor behaviors.
2. *Adaptively Timed Responding.* Adaptively timed read-out of responses via cerebellar circuits, as in Figure 7, enables learned responses to be released at task-appropriate times, despite the fact that CS cortical representations can be quickly activated by fast motivated attention.
3. *Adaptively Timed Duration of Motivated Attention and Inhibition of Orienting Responses.* Adaptively timed inhibition of mismatch-sensitive cells in the orienting system of the hippocampus (pathway $T \rightarrow D \rightarrow A$ in Figure 6) prevents the premature reset of active CS representations by potentially distracting irrelevant cues during variable task-specific delays. This inhibition is part of the competition that exists between consummatory and orienting behaviors. Even while this inhibitory mechanism prevents CS representations from being prematurely reset, adaptively timed incentive motivational feedback ($D \rightarrow S^{(2)} \rightarrow S^{(1)}$ in Figure 6) helps to maintain the activation of these representations in short-term memory. As a result, the CS representations can continue to read-out the sensory signals that will elicit adaptively-timed responding. A neural marker of adaptively timed motivational feedback is the Contingent Negative Variation, or CNV, event-related potential.

6. iSTART Model of Autism

6.1. Combining Three Imbalances in the iSTART Model

Constellations of autistic symptoms arise in the iSTART model when START mechanisms become imbalanced: underaroused depression within drive circuits like the amygdala, hypervigilant learning in recognition learning circuits within temporal and prefrontal cortices, and a failure of adaptive timing within hippocampal and cerebellar circuits.

6.2. Interactions between Hypervigilant Learning and Underaroused Depression

When underaroused depression in CogEM is combined with hypervigilant and hyperspecific learning in ART, underaroused emotional and sensory dipoles can exhibit a paradoxical enhancement of their ON channels when a nonspecific arousal burst is triggered by an unexpected event. Perseverature bursts of positive affect can thus be expected when the same object, viewed in slightly different ways, mismatches the hyperspecific top-down expectations of an individual with autism. In contrast, if a significantly larger arousal burst is caused due to a larger mismatch of a presently active hyperspecific prototype with a different and unexpected event in the world, then a large negative antagonistic rebound can be caused. As noted above, one behavioral strategy for coping with these system properties is to avoid novelty that will cause unbearable negative rebounds by perseverating on small details of the environment.

6.3. Interactions between Underaroused Depression and Adaptive Timing

Failures of adaptive timing can cause additional problems. Early in development, emotional needs may be met by responding with simple motor patterns in response to basic sensory stimuli. However, continued successful development in a social environment requires learning to adaptively time new actions to receive the rewarding consequences of these actions.

If the cerebellar cortex is damaged, then responses can be prematurely released. If the dentate-CA3 hippocampal circuit is damaged, then adaptively-timed motivated attention and inhibition of orienting responses may not occur, so spurious resets of attention may more readily occur. Social skills and language development are rewarded, attended, and learned through adaptively timed behaviors in a process of shared attention and imitation. Removing adaptive timing reinforces the tendency to focus attention on lower-order sensory representations and tasks.

Flexible shifts of attention can be impaired because, if the timing circuit *T* in the hippocampus is damaged (see Figure 6), attention may more easily be distracted from goal objects during task-related delays. Such a lack of timed control over variable delays can harm behavior more when it is necessary to shift attention among different sets of cues. On the other hand, if the orienting system *A* is also damaged (see Figure 2), then flexible reset of attention in response to novel events may be impaired because mismatch-based novelty-mediated attention shifts are no longer operational. If the attentional system in the neocortex remains intact, then direct activation of a recognition code in response to a familiar event is still possible, and the matching process can partially update short-term memory. However, without an intact adaptive timing or orienting system, the network can no longer flexibly search for the proper configuration of targets to attend, especially in the presence of complex spatial layouts that include distracting cues.

A failure of adaptive timing can also be due to insufficient teaching signals from depressed emotional centers to adaptive timing circuits. Thus, underaroused emotional depression, in addition to its negative effects on the development of a Theory of Mind, can lead to a reduction of normal reinforcing signals to hippocampal and cerebellar circuits, and with it abnormalities of adaptively-timed, motivated attention and action. If any of these mechanisms fail, then adaptively timed behavior may fail, and the future rewards that would normally be received, contingent upon making these adaptively timed behaviors, may not be forthcoming.

Perhaps the breakdown of the normal cycle of behavior and reward, with a dramatic reduction in the normal frequency of behaviorally appropriate rewards, may contribute to reduction in the arousal of emotional centers, as in Figure 4b, thereby helping to cause the symptoms that occur when drive representations *D* are underaroused. Thus, a tendency towards emotional underarousal can lead to reduced learning signals for adaptively timed learning, which can lead to a reduction of reward frequency, which can reinforce the underarousal that prevents adequate reward-based learning signals from occurring. Such a feedback cycle would involve amygdala, cerebellum, and hippocampus, among other brain regions.

6.4. Interactions between Adaptive Timing and Hyperspecific Learning

Another example of how system-wide feedback can maintain, and even worsen,

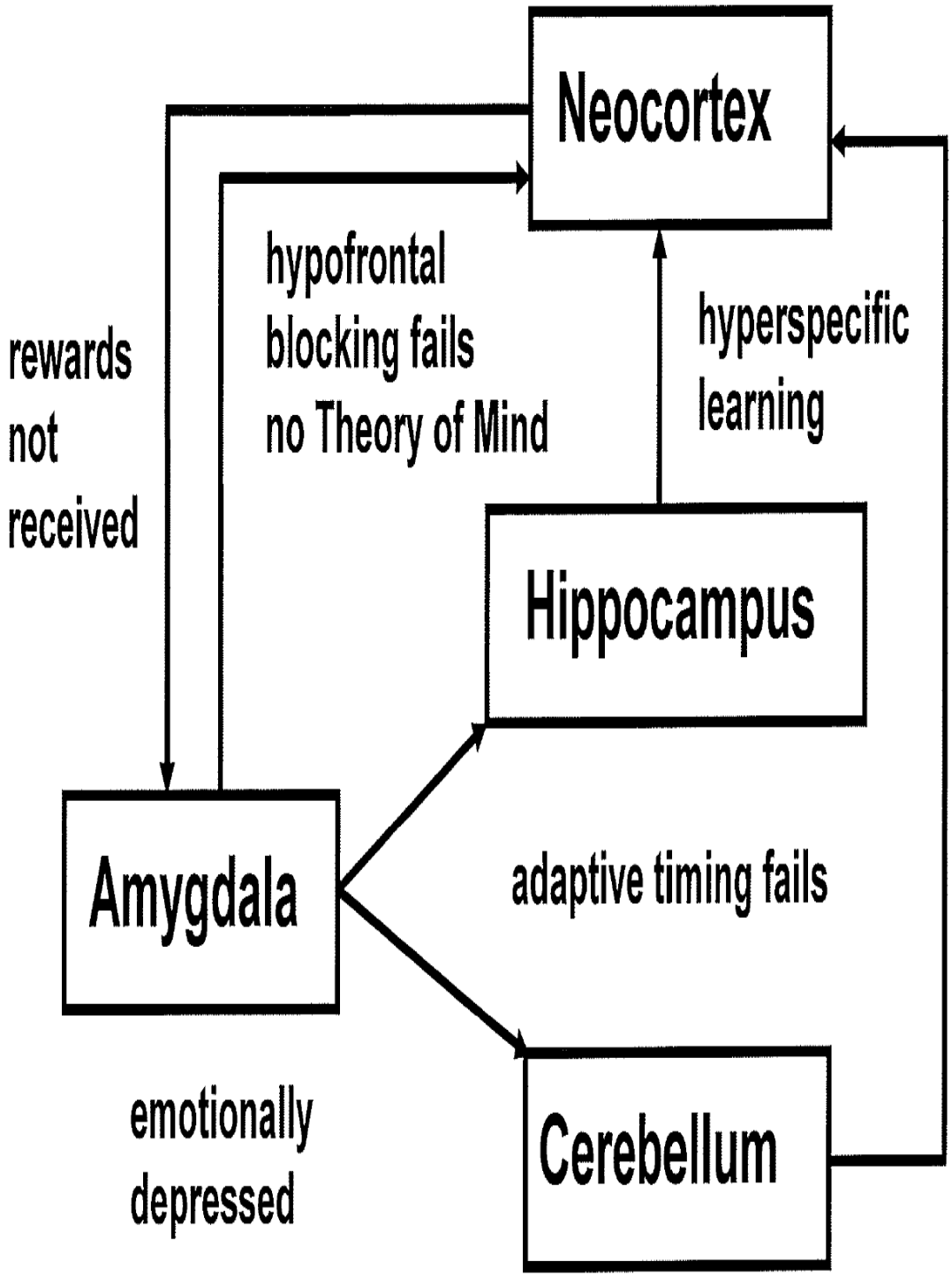

Neocortex
hypofrontal
blocking fails
no Theory of Mind
hyperspecific
learning
rewards
not
received
Hippocampus
Amygdala
adaptive timing fails
emotionally
depressed
Cerebellum

Figure 8. Schematic of processes that may contribute to autism. This figure summarizes how imbalances in particular brain processes may contribute to autistic symptoms, as well as feedback relationships whereby their effects may interact across the brain to sustain and reinforce these imbalances, and also create related symptoms. [Reprinted with permission from Grossberg and Seidman (2006).]

behavioral symptoms, concerns the fact that the frequent spurious orienting resets that can occur due to dysfunctional adaptive timing may also contribute to hyperspecific learning. If sensory inputs are prematurely reset, then this can interfere with the normal cycle of adaptively timed shifting of attention to the expected consequences of motor actions. Such a learner could not easily test whether variations on a sensory event predict similar consequences, so abstract prototype formation may not occur. This hypothesis predicts that there should be a correlation between the specificity of learned categories in inferotemporal cortex (Spitzer *et al.*, 1988) and the number of novelty-triggered N200 potentials (Deadwyler *et al.,* 1979, Deadwyler *et al.*, 1981) or even novelty-sensitive cells (Otto and Eichenbaum, 1992) in the hippocampus (Carpenter and Grossberg, 1993).

Drive satisfaction is often contingent on behaviors that use learned abstract prototypes to recognize variations of previously rewarded events. When this is prevented by hyperspecific learning, then the drive representations may become depressed. Thus, either hyperspecific learning in neocortical circuits, or failures in adaptive timing in cerebellar or hippocampal circuits, may contribute indirectly to underaroused emotional depression.

A "vicious circle" of environmentally mediated feedback can result in which depressed drives, say as measured by a hypoactive amygdala, fail to trigger the learning of adaptively timed behaviors (in hippocampus and cerebellum), whose absence enables the orienting system (in nonspecific thalamus, hippocampus, or cingulate cortex) to spuriously reset cognitive representations in the neocortex during times when attention should be given to a particular task, which then leads to hyperspecific learning of neocortical recognition categories, which then makes it easier to generate mismatch events with sensory cues, which then prevents the normal frequency of behaviorally-appropriate rewards from being received from the amygdala and other reward centers, which then contributes to maintaining depressed drives at these centers. Figure 8 summarizes some of these environmentally mediated feedback relationships. Table 2 summarizes model predictions to test these proposed mechanisms.

Depressed drive representations may cause a hypofrontal syndrome; see Figure 4b. As a result, the normal motivationally-selective top-down attentional priming signals to sensory cortices will be weakened, attentional blocking will be deficient, and motivationally irrelevant information can flood the sensory system, thereby making it even harder to process motivationally-relevant sensory cues, so that drives continue to be unmet, rewards unreceived, and the cycle perpetuates itself through this route as well.

The motivationally irrelevant information that can flood the brain in this way includes signals from lower-order sensory representations that have built-in pathways to emotional centers. Such a flood of signals can overcome the elevated thresholds due to underarousal of these centers. Excessive emotional responses can result. This property may also clarify the hypersensitivity of individuals with autism to a variety of lower-

Table 2
Model Predictions

Hyperspecific learning of recognition categories

Vary recognition exemplars gradually to test for narrow category boundaries

Test ERPs that indicate category reset, memory search, and attention shifts; e.g.,correlated series of P120, N200, and P300 potentials

Test fMRI measures of abnormal novelty detection in hippocampal area

Test the predicted relationship between increased specificity of learned categories, say in the temporal and prefrontal cortices, and the number of novelty-triggered N200 ERPs

Merge the Spitzer et al (1988) and Otto and Eichenbaum (1992) neurophysiological paradigms to test in monkeys the predicted relationship between difficult discrimination of matching from non-matching stimuli and the control of hippocampal novelty potentials

Study how baseline vigilance level is determined in normal individuals and clinical patients; cf. a possible role for Acetylcholine

Test if many individuals with autism have abnormally high baseline vigilance while individuals with medial temporal amnesia may have abnormally low baseline vigilance

Study how baseline vigilance may change during memory search for a correct recognition category; correlate vigilance changes with N200 and P300 ERPs

Test if the mismatch of a more general category causes vigilance to become momentarily greater than mismatch of a more specific category ("match tracking")

Test if hyperspecific learned categories prevent unrecognized variations of an event from becoming conditioned reinforcers due to their inability to trigger the actions that elicit the normal number of rewards

Underaroused Emotion Depression

Test if certain behavioral thresholds are elevated but responses are hypersensitive when the elevated threshold is exceeded by carefully controlled increments in stimulus intensity

Study possible brain basis for underaroused depression

Use classical conditioning blocking paradigm to test if attentional blocking is deficient

Correlate blocking properties with measures fMRI measures of amygdala unresponsiveness; does poor blocking correlate with amygdala unresponsiveness?

Test if orbitofrontal cortex is less responsive during intervals of amygdala unresponsiveness

Contrast possibility of low amygdala activation during inadequate attentional blocking with high amygdala activation during events that cause extreme negative emotional reactivity; e.g., variations in routines and responses to some sensory stimuli

Test if small mismatches of a hyperspecific recognition category do not generate a P300 ERP but large mismatches trigger both a P300 and strong amygdala activation corresponding to negative affect

Study ERPS and fMRI measures during perseverative attention to small details of an object for evidence that enhanced positive motivational activations, including ON motivational bursts, may occur during slight shifts of attention, rather than the extreme negative emotional activations that larger variations in routine and some sensory stimuli may cause

Failures of Adaptively Timed Learning

Test if adaptive timing deficits occur in certain autistic individuals, whether due to premature release of actions as a result of a cerebellar problem, or inability to maintain attention upon motivationally valued goal objects due to a problem in the dentate-CA3 hippocampal area

Study possible cerebellar deficiencies using the eye blink paradigm, including possible problems with metabotropic glutamate receptor functioning, for evidence of problems with adaptive timing of actions.

Study possible hippocampal deficiencies by testing inability to maintain attention upon a valued goal object for a predetermined time interval

Test if some failures of adaptive timing may be due to inadequate Now Print teaching signals in hippocampal and cerebellar circuits due to inadequate responses in areas like the amygdala to rewarding events

Use adaptive timing deficits as one marker for future autism in some infants

Functional imaging studies of brain regions associated with drives, such as the amygdala, will show abnormally strong activation to lower-order stimuli such as noxious smell, taste, and touch

Functional imaging studies of the same brain areas will show negligible responses to socially salient stimuli in most cases but extreme responses in the rare cases that the stimulus is able to overcome the threshold for response

order stimuli, such as noise and touch.

Deficient development of language is clarified by these model mechanisms. Language development requires shared, or joint, attention with a teacher and splitting attention between the objects of that shared attention, analysis of the sounds being produced, of the sounds just heard, and of the motor actions required to make those sounds. Language development also requires adaptively timed learning to enable children to learn culturally important sequential motor actions through imitation. For example, cerebellar adaptive timing mechanisms, together with neocortical working memory mechanisms, model how children may learn handwriting through imitation of script (Grossberg and Paine, 2000; Paine, Grossberg, and Van Gemmert, 2005). In the adult, conversation requires sustaining attention and the ability to flexibly disengage it. It requires delaying a motor response appropriately so that reciprocal communication may occur. It requires recognition of representations of social value. All of these processes are impaired within the iSTART model herein described.

6.5. Circular Reactions for Imitative Behaviors

How is joint attention learned? The CRIB model (Grossberg and Vladusich, 2010) proposes how imitation learning can occur between a student and a teacher who experience the world from different perspectives. The development of joint attention, whereby a student learns how to orient and pay attention to an object that the teacher is attending, is a key step in imitation learning and social cognition. Piaget (1952) showed how infants discover and learn sensory-motor mappings through self-initiated cycles of action and perception, called (*intra*-personal) circular reactions. CRIB shows how *inter*-personal circular reactions between a student and teacher enable joint attention to be learned, but only if attention is properly coordinated within the student. Inter-personal circular reactions clarify how a glance at a particular pose of a teacher's face can activate an eye movement command to look at the location in space to which the caregiver is attending, and to thereby achieve joint attention with the goal object at this location.

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