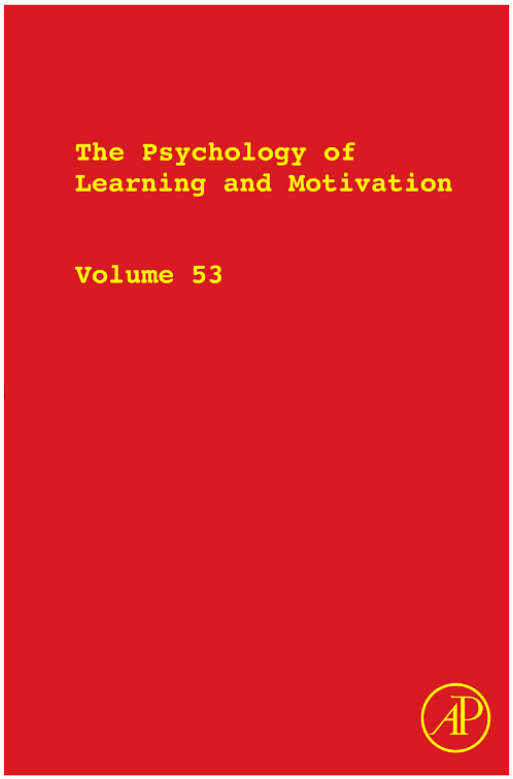

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CHAPTER SEVEN

Event Perception: A Theory and Its Application to Clinical Neuroscience

Jeffrey M. Zacks *and* Jesse Q. Sargent

Contents

Abstract

The chunking of continuous ongoing activity into discrete events is a central component of perception and cognition. It plays important roles in attention, cognitive control, and memory. Here, we review a theory of how the mind/brain

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segments ongoing activity into meaningful events. The theory proposes that event segmentation arises because perceptual systems make predictions about the near future. These predictions are guided by working memory representations, and when predictions fail memory representations are updated. We apply the theory to six conditions in clinical neuroscience: schizophrenia, obsessive-compulsive disorder, Parkinson's disease, lesions of the prefrontal cortex, aging, and Alzheimer's disease. This analysis makes novel suggestions for interventions to address these conditions, and points the way to new avenues of research.

1. Introduction

For humans to experience the world as structured, the brain must organize the cacophonous wash of information that comes in through the senses. One powerful organizational principle is *chunking*: grouping a contiguous region of the input space under one representation. Chunking occurs at many stages in the central nervous system. Close to the sensory surfaces, a single neuron in the primary visual cortex may code for information gathered from many individual photoreceptor cells in the retina and thus represent an extended, coherent feature in the visual environment, such as a line at a certain orientation (Hubel & Wiesel, 1968; Marr & Ullman, 1981). At later stages, information in memory appears to be organized so that some specific items are associated or grouped with other specific items. For example, items that are learned in close temporal proximity are more likely to be recognized later if they are again presented in close temporal proximity (e.g., Faust, Balota, & Spieler, 2001; Underwood, 1957).

In this chapter, we present a theory of how the human brain chunks the continuous stream of experience associated with everyday life into discrete episodes, or events. If asked to recall yesterday's activities, one might organize the description into separate chunks such as going to the grocery store, doing the laundry and calling a friend. We propose that this organization is not just the result of conscious efforts to present an orderly description. Rather, as a function of perceiving and experiencing those episodes, a network of specific brain regions automatically inserts boundaries between discrete events as they occur. As a fundamental element of normal perceptual processing, this type of segmentation is suggested to be at the center of attention, action control, online memory updating, and episodic memory encoding. Because these functions are so important for everyday functioning, conditions that affect event segmentation may produce significant changes in cognition. Accordingly, this chapter investigates event segmentation in relation to cognitive disorders in which everyday event understanding is disturbed. We hope this will prove useful in organizing the facts about

deficits of higher level cognition, providing testable hypotheses, and suggesting specific interventions that might not emerge from other theoretical perspectives. We begin with an overview of the theory and then consider how it may be useful in efforts to understand several neuropsychological disorders and the cognitive changes associated with healthy aging.

2. Event Segmentation Theory

Event Segmentation Theory (EST) describes how and why our nervous systems segment ongoing experience into discrete episodes (Zacks, Speer, Swallow, Braver, & Reynolds, 2007; see also Kurby & Zacks, 2008; Swallow & Zacks, 2008). For example, consider what might happen during a typical visit to a coffee shop: you wait in line, you give your order, you pay, you put cream in your coffee, you leave. Different people will generate somewhat different lists of activities, but all are able to describe experience across time as organized into distinct units and overall there will be considerable agreement across individuals regarding what those units are. EST proposes this happens because, as part of normal perceptual processing, humans automatically segment episodes into units. In fact, EST suggests that the ongoing segmentation of experience is at the center of cognitive control, working memory (WM) updating, and storage and retrieval from episodic memory.

The core components of EST, corresponding hypothesized neurophysiological structures, and the basic flow of information are illustrated in Figure 1. Reference to Figure 1 may be helpful as we describe the components of EST and review some of the relevant empirical evidence below. For a more detailed presentation of the neurocognitive account, see Zacks et al. (2007). For a more detailed computational presentation and computer simulation results, see Reynolds, Zacks, and Braver (2007).

EST starts from the supposition that some of the most important products of perception and comprehension are predictions about what will happen in the near future. Prediction is front and center in many contemporary accounts of perceptual processing (Enns & Lleras, 2008), learning (Schultz & Dickinson, 2000), and language (Elman, 2009). Good predictions are adaptive because they allow one to plan actions more successfully (e.g., avoiding hazards or intercepting desired objects). Also, good predictions can facilitate efficient perceptual processing. For example, if a pitcher winds up and completes a throwing motion, the perceptual system anticipates that the ball will fly out of the pitcher's hand toward home plate. In the absence of such anticipation, perceiving the ball whizzing through the air would be much more difficult—in fact, one might miss it altogether!

According to EST, prediction is abetted by WM representations called *event models*. Event models may be thought of as representations of

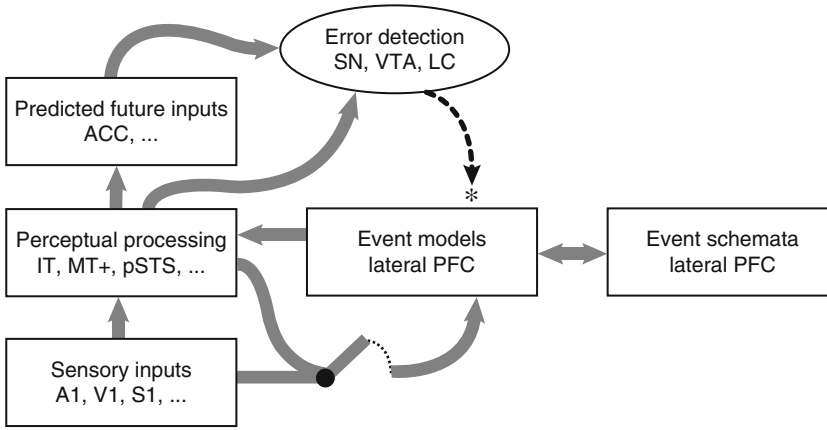

Figure 1 Schematic depiction of the model, with hypotheses about the neurophysiological structures corresponding to the different components of the model. Thin gray arrows indicate the flow of information between processing areas, which are proposed to be due to long-range excitatory projections. Dashed lines indicate projections that lead to the resetting of event models. PFC, prefrontal cortex; IT, inferotemporal cortex; MT+, human MT complex; pSTS, posterior superior temporal sulcus; ACC, anterior cingulate cortex; SN, substantia nigra; VTA, ventral tegmental area; LC, locus coeruleus; A1, primary auditory cortex; S1, primary somatosensory cortex; V1, primary visual cortex. (Adapted with permission from Zacks et al., 2007.)

what-is-happening-now. EST suggests that all perceptual input is processed in the context of a currently activated conception of what-is-happening-now. Our conceptualization of event models borrows heavily from work on *situation models* in discourse comprehension (e.g., Zwaan & Radvansky, 1998). Event models represent those aspects of a situation that are consistent within an event, while ignoring those aspects that vary haphazardly from moment to moment. Such representations are helpful not only for prediction but also because they allow the disambiguation of ambiguous sensory information and the filling-in of missing information. For example, at a baseball game an event model would represent the location of the baseball while it is hidden in the pitcher's glove. We have proposed that event models are maintained in lateral prefrontal cortex (PFC). Event models combine current perceptual information with information acquired very recently in the present context, and with patterns of information learned over a lifetime of experience. For example, if you have never seen a baseball game, the first time the pitcher sets up to throw, you may have very little idea where the ball will go. As the pitch count goes up, your expectation that each upcoming pitch will go to home plate increases. However, if you are an experienced baseball fan, each pitch in an at-bat is perceived in the context of an event model informed by relatively stable long-term semantic memory about what happens at ball games. In EST, these long-term weight

based representations are referred to as *event schemata*. In contrast, event *models* are activation-based WM representations. So, the content of an event model may overlap at any given time with a particular event schema, but when an event model ceases to have predictive value, it can be rapidly and completely updated to reflect the changing situation. We propose that event schemata as well as event models are implemented by the lateral PFC. A number of studies suggest that representations of events are maintained in the anterior, lateral PFC (e.g., Grafman, 1995; Schwartz et al., 1995; Wood & Grafman, 2003). We review some of this evidence in more detail in Section 6. The exact nature of the interaction between event models and event schemata is currently a topic of active research.

So, while event models may be informed by current perceptual information, they can also influence how the perceptual system processes that incoming information (see Figure 1). For example, as described above, information provided by event models allows the visual system to anticipate the flight of a baseball before it is released by the pitcher. However, event models may facilitate processing of all types of sensory information across numerous, distributed brain regions. Perceptual analysis is accomplished by hierarchically organized neural systems specialized for vision, hearing, touch, and the other sensory modalities. For example, in the visual system (Felleman & Van Essen, 1991), information is initially represented in terms of simple local visual features in the early visual areas (V1 and V2, in the posterior occipital cortex). Successive processing stages form representations that are increasingly extended in space and time. Two broad streams process information important for object identification and for motor control relatively separately (Goodale, 1993). Features relevant to object identity and category are differentially represented in inferior temporal cortex (IT), whereas features related to motion and grasping are differentially represented in dorsal regions including the human MT complex (MT+) and the posterior superior temporal sulcus (pSTS). Although there is communication between the streams and massive feedback throughout the system, these systems can be described as hierarchically organized, following a rough posterior-to-anterior spatial organization. Many of the classical studies characterizing these perceptual systems were conducted in nonhuman primates and relied on radically simplified stimuli. However, recent neuroimaging studies have shown similar responses in these areas across individuals during movie viewing (Bartels & Zeki, 2004; Hasson, Nir, Levy, Fuhrmann, & Malach, 2004; Hasson, Yang, Vallines, Heeger, & Rubin, 2008). EST proposes that event models bias processing in these streams. As we shall see shortly, EST also proposes that the updating of event models regulates processing over time in these streams.

A critical feature of event models is that they need to be protected from moment-to-moment changes in sensory and perceptual information. Updating one's event model to delete the baseball when it disappeared

from sight would clearly be counterproductive. However, event models have to be updated eventually in order to be useful—the baseball game model will not be helpful at a gas station! The question is, when and how can event models be updated adaptively? EST's answer is that event models are updated in response to transient increases in prediction error, mediated by systems in the anterior cingulate cortex (ACC) and midbrain neuromodulatory systems. The ACC maintains predictions and constantly compares them to actual inputs, producing an online error signal. Studies have shown this region to be sensitive to the commission of overt errors and to covertly measured cognitive conflict (e.g., Botvinick, Braver, Barch, Carter, & Cohen, 2001) and to the learning of sequential behaviors (Koechlin, Danek, Burnod, & Grafman, 2002; Procyk, Tanaka, & Joseph, 2000). When prediction error increases suddenly, this is detected by monitoring systems in the midbrain, which broadcast a global reset signal to the cortex. This system may include dopamine-based signaling subserved by the substantia nigra (SN) and ventral tegmental area (VTA) and norepinephrine-based signaling subserved by the locus coeruleus (LC). Neurons in the SN and VTA are sensitive to errors in reward prediction (e.g., Schultz, 1998). Dopamine cells in the SN and VTA project broadly to the frontal cortex, both directly and through the striatum, providing a mechanism for a reset signal such as is posited by EST. The LC has been implicated in regulating the sensitivity of an organism to external stimuli (e.g., Usher, Cohen, Servan-Schreiber, Rajkowski, & Aston-Jones, 1999). It also has broad connections to the cortex, these based on norepinephrine rather than dopamine. The reset signal transiently opens an input gate on the event models, exposing them to the early stages of sensory and perceptual processing (see Figure 1). This produces a short burst of increased activity in the perceptual processing stream and the event models settle into new states. As the event models are updated, predictions become more adaptive and errors decrease. The system returns to a stable configuration. A schematic representation of the temporal dynamics of the error-based updating process is shown in Figure 2.

According to this account, event segmentation is an ongoing concomitant of everyday experience, which happens without intent or necessarily awareness. The processing that occurs at event boundaries can be viewed both as focal attention and as memory updating. An appropriate (stable) event model is a WM buffer whose outputs bias processing in that stream. The opening up of event models' inputs is a form of focal attention, and the settling into a new state is a form of memory updating. Event segmentation in and of itself is not the goal of the system; instead, it is a by-product of mechanisms evolved in support of a more efficient, predictive perceptual system.

Important for thinking about how EST applies to daily experience, it is suggested that event segmentation occurs simultaneously at multiple time scales. Consider the coffee shop example given above. If one's event model for going to a coffee shop generates predictions consistent with all the distinct units of activity typically involved (e.g., waiting, ordering, paying),

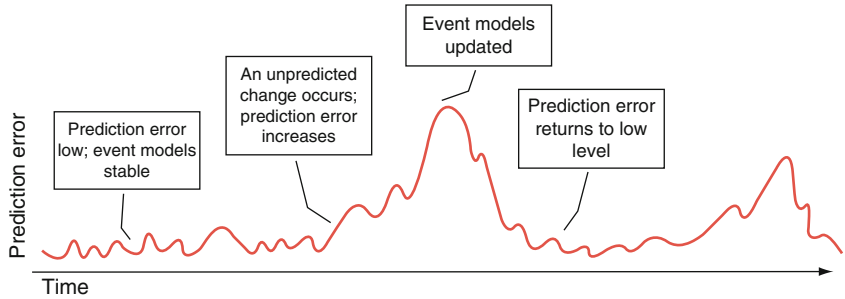

Figure 2 Temporal dynamics of event segmentation. Most of the time prediction error is relatively low and event models are stable. As a model becomes less adaptive, prediction error increases. In response, information form sensory and perceptual processing is gated into the model, updating its contents. After updating, error declines and the model settles into new state.

then no error signal would be generated, the model would be stable throughout the episode, and no event boundaries would occur. So, how does EST explain the segmentation of going to a coffee shop into distinct units of activity? We may consider events as hierarchical representations. The event "going to a coffee shop" is at a higher level in the hierarchy than the events "waiting in line" and "ordering". Lower level aspects of an event representation are sensitive to prediction error signals integrated over shorter time scales. So, when it comes time to place an order, the "waiting in line" level of the hierarchical event representation generates some degree of prediction error. That hierarchical level becomes unstable until the "ordering" model is instantiated at which point the error signal decreases. Meanwhile, at a higher level of the event representation, "going to a coffee shop" is insensitive to such short lived error signals. Higher levels are sensitive to error signals integrated over longer time scales. When one leaves the coffee shop, it is likely that there will be a more prolonged increase in error. The resulting prolonged error signal causes instability at a higher level of the hierarchical representation and "going to a coffee shop" is abandoned for a more adaptive model. In accordance with this explanation, we would expect models at higher hierarchical levels to make less specific predictions. Also, we would expect boundaries between events at higher hierarchical levels to align with boundaries between events at lower levels.

2.1. Prior Evidence

EST makes a number of claims about behavior and brain function, some of which are consistent with previous research and some of which have been tested directly. First, EST predicts that event segmentation is an ongoing part of normal perceptual processing. Evidence for this proposal comes from

behavioral and functional magnetic resonance imaging (fMRI) studies. In a typical event segmentation paradigm, participants watch movies of actors engaged in everyday activities (e.g., doing laundry) and are instructed to press a button whenever they believe one meaningful unit of activity has ended and another has begun (Newtson, 1973). When instructions direct attention to larger (coarse grain) or smaller (fine grain) units of activity, the behavioral data are thought to reflect ongoing event segmentation at higher or lower levels of hierarchical event representation. Studies have demonstrated that segmentation of videos using this method shows both stable intersubject agreement, and stable individual differences over a period of more than a year (Newtson, 1976; Speer, Swallow, & Zacks, 2003; Zacks et al., 2007). Furthermore, observers spontaneously group fine-grained event boundaries into hierarchically organized coarse-grained events (Newtson, 1976; Zacks, Tversky, & Iyer, 2001). That is, coarse grain boundaries tend to correspond to a subset of fine grain boundaries, which supports the view that event segmentation occurs simultaneously at multiple time scales. The reliability and structure of the data from the segmentation task support the suggestion that this paradigm is capturing an ongoing feature of normal perception. Ultimately, however, these results prove only that individuals *can* segment ongoing experience into units. Evidence that individuals *do* segment experience in the course of normal day-to-day perception comes from neurophysiological studies. Using fMRI, Zacks et al. (2001) first monitored participants' brain activity during passive viewing of simple movies. Afterward, participants segmented the movies by indicating whenever, in their view, one meaningful unit of activity had ended and another had begun. During passive viewing, a collection of regions transiently increased in activity at those moments that viewers later identified as event boundaries. These regions included areas in lateral posterior cortex (including the inferior and superior temporal sulci and ventral temporal cortex), medial posterior cortex (including the cuneus and precuneus), and lateral frontal cortex. Similar results have been generated using several variations of this general paradigm (Speer, Zacks, & Reynolds, 2007; Speer et al., 2003; Zacks, Swallow, Vettel, & McAvoy, 2006).

Second, EST predicts that perceptual processing increases at event boundaries. The fact that brain activity transiently increases at event boundaries is consistent with this prediction—particularly suggestive are the increases in posterior regions associated with perceptual processing. It has been shown that memory for perceptual details at or around event boundaries is better than that for details associated with event middles (Newtson & Engquist, 1976; Schwan, Garsoffky, & Hesse, 2000). Also, EST suggests that if the surface structure of events is consistent with the underlying event structure, then event segmentation mechanisms should operate more efficiently, and again, memory for the episode should improve. This too has

been borne out in the laboratory (e.g., Schwan & Garsoffky, 2004). For example, Boltz (1992) showed participants a feature film with no commercial breaks, breaks that corresponded to event boundaries, or with breaks placed at nonboundaries. Recall of activity and memory for the temporal order of events in the movie was improved by the breaks at event boundaries and reduced by the breaks at nonboundaries. Further support for the suggestion that segmenting events in a manner that corresponds to their intrinsic structure improves memory for those events comes from a study of individual differences. Zacks, Speer, Vettel, and Jacoby (2006) found that group-typical segmentation of movies, which may be assumed to reflect intrinsic structure, predicted better performance on subsequent memory tests after controlling for overall cognitive level.

Another prediction of EST is that information associated with the current event model, and thus active in WM, should be more accessible than information associated with a previously active model. When using text material, event boundaries can be induced by imposing a change such as a temporal break (e.g., "...a day later...") or a shift of spatial location (e.g., "the detective burst into the room"). Such shifts result in the perception of an event boundary for films as well (Zacks, Speer, & Reynolds, 2009). Numerous studies using text comprehension have shown results consistent with this prediction (e.g., Bower & Rinck, 2001; Zwaan & Radvansky, 1998). For example, Speer and Zacks (2005) required participants to read narratives and showed that memory for items in the narrative was lower when a temporal break intervened between the mention of the item and the test. Similar results have recently been obtained with movies (Swallow, Zacks, & Abrams, 2009).

In sum, EST proposes that predictions about the near future are guided by WM representations of the current event, which are updated in response to transient increases in prediction error. This updating includes upregulation of the perceptual processing pathways feeding into event models. The experience of an error spike and consequent updating is perceived as a boundary between meaningful events. Thus, event segmentation is an ongoing perceptual mechanism standing at the center of attention, cognitive control, and memory. It is subserved by a distributed set of brain mechanisms described above (see Figure 1). If one or more of these is selectively affected by a disorder or age-related process, it may have substantial consequences for cognition.

In the following sections, we apply EST to the analysis of six conditions in clinical neuroscience. We have selected six conditions based on the overlap between the neurocognitive mechanisms implicated in each and the mechanisms of event segmentation as proposed by EST. The six are: schizophrenia, obsessive-compulsive disorder (OCD), Parkinson's disease (PD), lesions of the PFC, aging, and AD. Our selections are necessarily heuristic and surely incomplete. However, we think the analysis shows the

potential for EST to provide new insights regarding major cognitive deficits associated with these disorders.

3. Schizophrenia

Schizophrenia is a developmental neurocognitive disorder that affects approximately 1% of adults (Bresnahan et al., 2000). In most cases, it is diagnosed in early adulthood and has consequences throughout adult life. Schizophrenia has classically been characterized by positive symptoms, which include hallucinations, delusions, and paranoia, and by negative symptoms, which include flattened affect, reduced volition, and anhedonia. However, it has become increasingly clear that cognitive impairments are a prominent part of the disease, and that these have profound effects on people's lives. In a review and meta-analysis, Green, Kern, Braff, and Mintz (2000) examined the relations between cognitive deficits and functional outcomes. The cognitive variables studied included secondary (long-term) verbal memory, immediate verbal memory, executive control, and vigilance. The functional outcome measures included success in psychosocial skill acquisition, social problem-solving, and daily functioning such as occupational functioning and independent living. All of the cognitive variables were related to functional outcomes, accounting for 20–40% of the variance across individuals. Thus, cognitive performance is a major predictor of the ability of persons with schizophrenia to maintain employment, build social ties, and live independently.

The etiology and pathophysiology of schizophrenia are complex and not fully understood. Schizophrenia selectively affects the PFC, as well as the hippocampus and thalamus (Harrison, 1999). The neurotransmitter dopamine has been shown to play a major role in the disorder, though its functions are still not fully known. Dopamine's role in schizophrenia has been reviewed by Guillin, Abi-Dargham, and Laruelle (2007). Early research focused on the D2 dopamine receptor, which is widely expressed in the midbrain where large numbers of dopamine neurons are found. In a classic set of studies, Creese, Burt, and Snyder (1976) discovered that the effectiveness of antipsychotic medications to treat the positive symptoms of schizophrenia was correlated with their ability to occupy D2 receptor sites. Current theory holds that abnormally high activity of D2 receptors in the midbrain reduces the effectiveness of glutamate, an excitatory neurotransmitter. More recently, attention has focused on D1 receptors in the PFC. D1 receptors have been found to have reduced activity in the dorsolateral PFC, possibly as a compensatory response to chronic

overstimulation. Reduced D1 responsiveness interferes with inhibitory local signaling based on the neurotransmitter GABA.

3.1. Cognitive Deficits

The cognitive deficits in schizophrenia are specialized rather than global. For example, implicit memory appears to be relatively well preserved (Clare, McKenna, Mortimer, & Baddeley, 1993). However, WM—the ability to store and manipulate information over short durations—is impaired. Barch (2005) reviewed the data on WM impairments in schizophrenia and proposed that a specific component of WM is affected by schizophrenia. Baddeley's (1986) WM theory proposes that WM is implemented by a set of passive storage systems and a central executive that manages the updating and transformation of information in the storage systems. Barch argued that the data suggest no impairment in the maintenance of auditory information, possible impairment of visuospatial information, and substantial impairment of the central executive. This central executive impairment is associated with functional differences in the PFC. In particular, Barch (2006) and colleagues have proposed that the ability to maintain cognitive representations of task set and use them to guide behavior is impaired in schizophrenia. This conception of the central executive is consistent with Baddeley's (2000) recent proposal of an *episodic buffer*, a component of the central executive that maintains integrated multimodal representations of the current behavioral episode.

3.2. Schizophrenia and Event Segmentation

In terms of EST, the neurochemical and cognitive disturbances identified in schizophrenia could produce two different effects on event understanding. If D2 hyperactivity impairs the effectiveness of long-range excitatory projections from the midbrain, this would be expected to impair event model updating. If D1 hypoactivity in the PFC affects the maintenance and use of information, this should be reflected as an impaired ability to maintain information in event models. This proposal fits with the behavioral findings that the central executive may be impaired in schizophrenia. In particular, it is consistent with the proposal that task set representations are affected by the disease. Both of these possibilities—impaired event model updating and impaired maintenance—would be expected to lead to deficits in event segmentation.

There is very little direct evidence on event perception in schizophrenia, but the existing data support the existence of an event segmentation deficit. Zalla, Verlut, Franck, Puzenat, and Sirigu (2004) asked outpatients with schizophrenia and healthy controls to view movies of everyday activities and segment them into fine and coarse events. Patients and controls

identified similar numbers of events, and their fine-grained event boundaries were located in similar locations. However, the patients tended to identify coarse-grained boundaries in normatively incorrect locations. This tendency was correlated with schizophrenic symptomatology. In a pilot study in our laboratory (Zacks & Barch, unpublished data), we replicated the finding that persons with schizophrenia segmented in a less normative fashion than healthy controls. Persons with schizophrenia also showed impaired memory for the temporal order of events, and impaired recognition memory for pictures taken from the events.

In the future, it will be important to follow up these results to determine if schizophrenia produces a selective deficit in event model updating or maintenance. If updating is selectively impaired, it may be possible to remediate this by teaching explicit strategies for identifying event boundaries, or by explicitly highlighting event boundaries in texts or films. If event model maintenance is selectively impaired, it may be possible to intervene by teaching explicit strategies to rehearse key information such as characters and task goals, or by providing external memory aids to support maintenance. Finally, in the meta-analysis described above, Green et al. (2000) note that the mechanism by which the cognitive deficits associated with schizophrenia lead to lower functional outcome scores remains unclear. Here, it is interesting to consider that event segmentation mechanisms may be closely related to performance on functional outcome measures. For example, in one functional outcome measure, patients are required to interpret vignettes depicting interpersonal interactions. The ability to form and maintain appropriate event models would seem to be central to this task. This suggests that measures of event segmentation ability might be particularly informative regarding the ability of schizophrenics to function independently in society.

In sum, cognitive dysfunction is a salient component of schizophrenia and a major predictor of the disease's impact on a person. Neurophysiological studies implicate the dopaminergic system, including midbrain D2 receptors and prefrontal D1 receptors. Disruption of either system could produce disorders of event segmentation and memory. The limited available evidence supports the existence of such disorders and suggests point of intervention to remediate them.

4. Obsessive-Compulsive Disorder

OCD is a psychiatric condition characterized by persistent intrusive thoughts and compulsive behaviors. The obsessive thoughts often have to do with threats to safety and threats of contamination. Compulsive behaviors often relate to alleviating these threats (e.g., compulsive washing associated with obsessive thoughts about dirt or disease), but in some cases

the behaviors appear to be unrelated to the obsessive concerns (Boyer & Lienard, 2008).

For a time, a dominant view of the neurochemical mechanism of OCD was that it was caused by hypoactivity of the neurotransmitter serotonin. This was motivated largely by the finding that the symptoms of OCD were ameliorated by serotonin reuptake inhibitors (SRIs), antipsychotic drugs that strengthen the effects of serotonin in the synapse. However, the effects of SRIs are widespread and complex, and some studies that have directly intervened in the action of serotonin have cast doubt on its being the primary causal mechanism. As a result, some attention recently has focused on a possible role of dopamine in OCD (Fornaro et al., 2009). Another possibility that has been proposed is that a circuit involving the orbitofrontal cortex (OFC), the SN, and the basal ganglia is dysregulated, leading innate motor programs to be triggered inappropriately (Rapoport, 1990), or to hypersensitivity of attentional systems to environmental threats (Saxena & Rauch, 2000). Both serotonin and dopamine play important roles in this circuit. Huey et al. (2008) have presented a psychological and neuroanatomical model of OCD that is particularly relevant to the current discussion because of the central role played by event representations. This model suggests that the PFC supports goal-oriented, structured sequences of events (*structured event complexes*, or SECs). Once this type of event representation is activated, a network of neural systems involved in reward and emotional processing (e.g., OFC, limbic system), support a motivational signal, experienced as anxiety, that abates upon completion of the SEC. The authors suggest that OCD patients do not experience the relief from anxiety normally associated with completion of an SEC. Obsession is the behavioral manifestation of the neural signal that an SEC has been "left hanging."

4.1. Cognitive Disturbances

Evans and Leckman (2006) have recently reviewed the epidemiology, symptomatology, and neurophysiology of OCD. They note that the intrusive thoughts associated with OCD are similar to those experienced by healthy controls—they are just more frequent and more difficult to dismiss. Obsessive behaviors vary over the lifespan in healthy persons, being most prominent in early childhood (2–6), at puberty, and after becoming a new parent. Early childhood and puberty are also the peak times of onset of clinical OCD. Together, these patterns suggest that persons with OCD do not have disordered representations of events, objects, or persons; rather they have a disruption in the ability to control the unwanted influence of some of these representations. Evans and Leckman propose that OCD arises from the dysregulation of evolutionarily adaptive systems for threat monitoring and avoidance.

What is the nature of this dysregulation? Current accounts propose hyperactive monitoring of overt behavioral errors or of covert conflict between information processing streams (van Veen & Carter, 2002). Degree of obsessive thought is correlated with errors on the Wisconsin Card Sort Test (WCST). The WCST requires one to sort cards according to the number, shape, or color of objects on the card. One must discover a rule for sorting cards based on feedback, and then adapt one's performance when the experimenter covertly changes the rule. This task would seem to be quite sensitive to dysfunction in error detection mechanisms. However, there is no evidence that increased WCST errors among OCD patients are related to error or conflict processing, and the relationship could reflect broader cognitive impairments. There are stronger data linking OCD to selective deficits in motor inhibition and response suppression (Evans & Leckman, 2006). However, the strongest data come from studies that have measured neurophysiological responses to situations that produce errors or high conflict. These studies have focused on the ACC. As we have described in Section 2, the ACC is associated with monitoring conflict between information streams, and in EST it is proposed to support the evaluation of prediction error.

In one study, Gehring, Himle, and Nisenson (2000) asked persons with OCD and healthy controls to perform the Stroop task. In this task, participants are shown color words printed in various ink colors (either congruent or incongruent with the color named by the word) and asked to name the ink color, rather than to read the word. This requires suppressing a prepotent response to read the word and produces slow responses or errors, depending on the task constraints. Gehring and colleagues used electroencephalography to measure a correlate of error processing, the error-related negativity (ERN), during task performance. The ERN is a negative-going wave that is found just after people commit errors in simple cognitive tasks. It is strongest over frontocentral electrodes, and is thought to originate in the ACC. The Stroop task requires a high degree of cognitive control and leads to frequent errors, accompanied by ERNs. In persons with OCD, these responses were exaggerated. In a functional MRI experiment from this group, persons with OCD and controls performed a *flanker task*, in which they had to respond to the identity of the central character in an array while ignoring the characters on either side. Like the Stroop task, the flanker task produces a conflict between response tendencies driven by the target stimulus information and those driven by the to-be-ignored information. Also, like the Stroop task, it produces many errors. Both the control and patient groups showed increased activity in the dorsal portion of the ACC on trials in which they made errors. However, the OCD patients also showed significant increases in the rostral ACC. Overt errors do not appear to be necessary to produce activation in the ACC, nor to dissociate the neurophysiological response of control and OCD participants to task performance (van Veen & Carter, 2002). In one study, Ursu, Stenger, Shear,

Jones, and Carter (2003) asked participants with OCD and healthy controls to perform a version of the *continuous performance task*. In this task, participants view a sequence of alphabetic characters and are asked to respond only when a particular two-step subsequence is presented (e.g., an A followed by an X). The stimulus set was constructed such that when the first character (A) appeared, the second (X) was quite likely. This establishes a strong prepotent tendency to respond to the following character. Thus, if the following character is a nontarget (e.g., Y), there is a conflict between the prepotent response and the correct nonresponse. On such trials, persons with OCD showed larger responses in the ACC than controls—even when they successfully withheld their responses.

A striking feature of all three of these studies—using three different tasks—is that the behavioral performance of persons with OCD did not differ substantively from the controls. Thus, the neurophysiological markers of exaggerated error or conflict processing were present even when there was no evidence of "compulsive" behavioral performance.

4.2. Obsessive-Compulsive Disorder and Event Segmentation

In terms of EST, we consider three possible pathways by which OCD could be related to event segmentation. One possibility is that compulsive behavior results from attending to or integrating prediction error signals at an abnormally short time scale, which should cause one to experience events as segmented at an abnormally fine grain. Boyer and Lienard (2008) propose that this is the case and that it accounts for the ritualized character of compulsive behaviors. If one attends to events on a very fine time scale, one should neglect their relation to larger events and the larger goals of one's actions (Vallacher & Wegner, 1987). Boyer and Lienard propose that this shift to a fine grain of event segmentation is adaptive because it occupies WM, which reduces the intrusion of obsessive thoughts. A recent study by Zor et al. (2009) provides support for this possibility. In this study, participants with OCD were videotaped performing activities that formed the basis for their compulsive behavior, for example, filling a pet's bowl, lighting a cigarette, or blowing one's nose. For each patient participant, a control participant was videotaped performing the same activities. The low-level actions (e.g., checking the bowl's position, waving hands) were coded from each videotape. The patient group performed many more actions than the controls and repeated actions more often. Importantly, these "extra" actions tended to be idiosyncratic and apparently nonfunctional, such as waving one's hands when filling a pet's bowl. This result suggests that the patients were attending to the activity at a low level that neglected the goal relevance of the individual actions. A straightforward prediction from this proposal is that patients with OCD should segment activity into finer-grained events than control participants. This could be tested using the

behavioral tasks described previously. (One caveat is worth mentioning: Grain of segmentation in explicit event-marking tasks is quite sensitive to instructions and participants' interpretations of those instructions. These effects presumably affect the output processes involved in performing the explicit task, such as deciding when to press a response key and executing the response, rather than affecting the ongoing segmentation process. So, if comparing patients to controls, one would want to minimize task demands that could affect segmentation grain and use converging measures to help distinguish between differences in the mechanisms of ongoing segmentation and differences in task-specific output processes.)

A second possibility is that obsessions and compulsive behavior result from a chronically high prediction error signal or a too-low threshold for error-based gating. (In EST, these two components are not uniquely identifiable, because raising or lowering the mean error signal can be compensated for by lowering or raising the gating threshold.) This accords with OCD patients' frequent report that things "just don't feel right." It is also consistent with the findings of exaggerated error and conflict signals in the ACC, described above. Exaggerated prediction error responses could result in more frequent activation of the error-based gating mechanism and therefore more frequent event boundaries. At the same time, if prediction error signals are chronically elevated, this would reduce the ability of the error-based updating system to distinguish between intervals of low and high prediction error. This in turn should produce unreliable, idiosyncratic event segmentation. Thus, this proposal predicts that the segmentation of patients with OCD should be more idiosyncratic than that of controls as well as finer grained. Failing to segment activity into the proper event units would be expected to reduce the effectiveness of actions and could produce the sorts of interruptions and perseverations reported by Zor et al. (2009).

A final possibility is that persons with OCD have disordered event schemata. Schemata are long-term memory representations implemented by synaptic weight changes. These representations reflect commonly activated event models. For example, the event schema representing making toast is built up over a lifetime of experience making toast. Now, suppose that one began performing some nonfunctional perseverative behavior, such as tapping the toaster three times, whenever one made toast. Eventually, the schema for making toast would include this nonfunctional behavior. In this case, the presence of the toaster-tapping in the event schema would represent a source of compulsive behavior, in addition to whatever other sources may exist. Although this does not explain the initial appearance of the compulsive behavior, it may be a mechanism by which such behavior becomes difficult to expunge.

In sum, the clinical profile and pathophysiology of OCD suggest it may involve a dysregulation of mechanisms for monitoring error or conflict. Such dysregulation could affect event segmentation directly or indirectly,

through its long-term impacts on event schemata. If so, event segmentation measures may prove valuable for better understanding the mechanisms of OCD or for diagnosing it. We believe this is a fertile area for future research.

5. Parkinson's Disease

PD is a neurological condition characterized by a disorder of movement (Binder, Hirokawa, & Windhorst, 2009). Patients with PD have tremor, rigidity, postural instability, and bradykinesia—difficulty initiating movements combined with slow movement execution. Bradykinesia is often the most debilitating motor symptom. PD is diagnosed by a cluster of these motor symptoms combined with a finding that the individual responds to medications that increase the effectiveness of dopamine. PD is also associated with nonmotor symptoms including loss of smell, depression, anxiety, autonomic dysfunction, sleep disturbance, and cognitive deficits.

The etiology of PD is not fully understood; most likely, PD can arise through multiple pathways (Olanow & Tatton, 1999). It occurs occasionally in middle age, but becomes more prevalent after age 60. The motor symptoms are the result of a dramatic reduction in the projection of dopamine cells from the SN to the striatum. These pathways form part of a set of *thalamocortical loops*, which are thought to be important for the online control of movement and cognition. However, PD is also associated with more diffuse lesions to subcortical and cortical structures as well. PD often is accompanied by frank dementia, particularly in the later stage of the disease (Aarsland et al., 2001). Dementia in PD is characterized by deficits in executive control, visuospatial processing, and personality disorder (particularly depression). The mechanisms of PD dementia are not well understood. The fact that it is not well controlled by dopamine agonist medications suggests that PD dementia may be caused by lesions other than those to the dopamine cells in the SN described above. Because this dementia is relatively global and its mechanisms are not currently well understood, its relevance to event perception is limited. In earlier or milder cases, the cognitive deficits are more focal and therefore may be more informative. Thus, we focus here on cognitive deficits in PD patients without dementia.

5.1. Cognitive Deficits

In a comprehensive review, Taylor and Saint-Cyr (1995) described the primary cognitive deficit in PD as a selective impairment of the selection of action plans when the environment provides cues for multiple potential action plans. For example, patients with PD typically are impaired on the WCST. A key characteristic of this task is that the cues provided by the card

underdetermine the correct response. PD patients are also impaired on a version of the Tower of Hanoi task. The Tower of Hanoi is a puzzle in which participants must move a stack of discs of various sizes from one of three pegs to another, subject to two rules: One can only move one disc at a time, and a larger disc can never be placed on a smaller disc. In this case, several moves are possible on each turn and the participant must hold multiple evaluations in mind to select a better move. Taylor and Saint-Cyr propose that the cognitive deficits can be understood neurobiologically in terms of two thalamocortical loops projecting from the SN. Both loops project through the basal ganglia to the cortex, primarily the PFC. Whereas the motor dysfunction in PD may be due to damage to projections from the SN to the caudate nucleus of the basal ganglia, the cognitive deficit may be due to projections from the SN to the putamen, as well as direct projections to the supplementary motor are and the dorsolateral PFC.

A recent study focused, in particular, on cognitive deficits in PD patients without dementia (Green et al., 2002). Patients and controls were administered a battery of cognitive tests. Patients had relatively preserved short-term memory span and long-term recognition memory. However, impairments were frequently observed in the WCST and in fluency tasks (e.g., naming as many animals as possible within a 1-min interval). These deficits were interpreted as reflecting damage to the "cognitive" thalamocortical loops. However, patients also were frequently impaired on judgments of line orientation and the acquisition of new verbal memories; these deficits do not fit as well with this interpretation.

Persons with PD are impaired at learning new sequential behaviors (Seger, 1994). For example, in the serial reaction time task, participants are cued to press one of several keys by the onset of a light above the key. Trials follow each other rapidly, and a repeating sequence of keys can be embedded in the string of trials. Performance improves over time for two reasons: Participants become faster at responding to the light, and they learn to anticipate the sequence of keypresses. This can be seen by contrasting the condition with the repeating sequence to a condition in which each light follows the previous one randomly. Performance in this control condition improves somewhat with practice, but not as much as in the sequential condition. Sequence learning in the serial reaction time task often occurs without participants becoming aware of the repeating sequence, particularly if the sequence is relatively long. Patients with PD show substantially reduced sequence learning in this and related tasks.

In sum, in the early stages of PD, there may be a relatively selective deficit due to selective damage to the thalamocortical loops. This may result in impairments of action selection when multiple potential actions are possible, and in learning associations among actions in these conditions.

5.2. Parkinson's Disease and Event Segmentation

In terms of EST, a primary lesion to the dopaminergic projections from the SN would be expected to produce a deficit in updating event models. The deficits of PD patients in the WCST accord well with this possibility. However, we will see that similar deficits can be produced by lesions to frontal cortex, which we interpret as selectively affecting event model maintenance or event schemata (see Section 2). This task is not well suited to teasing apart event model updating from maintenance. Similarly, impairments in action selection and sequential learning are consistent with a deficit in event model updating, but do not discriminate this possibility from numerous others.

A pair of studies by Zalla and colleagues (Zalla et al., 1998, 2000) strongly suggest that event schemata are intact in patients with PD, dissociating their performance from those of patients with prefrontal lesions. In one study (Zalla et al., 1998), patients with PD were given cards describing steps in everyday activities such as toasting bread and going to the movies. On each trial, 20 cards were given, 5 for each of 4 activities. Participants were asked to sort the cards such that the steps for each activity were segregated and ordered. Whereas frontal lesion patients frequently mixed steps from the different activities, PD patients were able to segregate the activities and order the cards. However, their performance was quite slow, and when distractor steps were included (which did not belong to any of the activities), PD patients were less able to set these aside. Zalla et al. concluded that whereas the frontal lobe patients had deficient event representations, the PD patients had intact event knowledge but had difficulty shifting their cognitive set in order to deploy that knowledge efficiently in the task.

In the second study (Zalla et al., 2000), PD patients and patients with frontal lobe lesions were asked to generate lists of the steps involved in a similar set of everyday activities. Again, the frontal lobe group showed evidence of impaired event knowledge, producing fewer correct steps and failing to place them in the correct order. The PD group showed neither impairment. However, they were less able to identify which steps were important for completing the activity. Zalla et al. interpret this as an impairment in action selection.

Little is known about event segmentation in PD. EST predicts that if cortical updating due to dopamine signaling is impaired, then patients with PD should show disorganized event segmentation. This should be evident in segmentation behavior: Patients with PD should show reduced segmentation agreement. Moreover, patients with PD should show reduced evoked brain responses at event boundaries, reflecting reduced updating. This should hold whether normative event boundaries or those identified by the patient are used to estimate the evoked responses.

Thus, the deficits in event understanding observed in PD are consistent with the hypothesis that dopamine-based updating is impaired in this disorder. However, the tasks that have been used thus far do not differentiate this possibility from the possibility that event model maintenance may be impaired. This is an important question for future research.

6. Lesions of the Prefrontal Cortex

Lesions to the PFC produce cognitive disturbances that are at once subtle and profound. On the one hand, prefrontal lesions rarely produce dramatic deficits in sensation, perception, or movement control (though lesions to the immediately posterior parts of frontal cortex produce profound motor deficits). On the other hand, prefrontal lesions often produce disorders of intentional action that interfere greatly with everyday functioning. There is a large literature on the cognitive deficits associated with prefrontal lesions (for reviews, see Fuster, 1997; Grafman, 1995). Here, we focus on those aspects of cognition that are most relevant for event understanding.

6.1. Cognitive Deficits

Persons with prefrontal lesions frequently suffer from particular forms of *apraxia*, or disorder of action. Whereas persons with posterior lesions are more likely to experience apraxias in which they are unable to pick up objects or perform body movements on command, persons with prefrontal lesions often have intact ability to perform simple actions but deficits in the ability to organize these actions effectively. Schwartz and her colleagues have described this as *action disorganization syndrome* (Schwartz, 2006; Schwartz et al., 1995).

One potential cause of action disorganization syndrome is damage to the long-term memory representations supporting structured action. Evidence that such knowledge depends critically on the PFC comes from several sources. Grafman and colleagues have suggested the PFC stores representations of typical actions called *structured event complexes* (SECs; e.g., Grafman, 1995, 1999; Sirigu et al., 1998). SECs correspond closely to the event schemata described above (see Section 2). They are structured representations that capture information about the actions that make up an activity, their relations, the social structure of the activity, and the activity's characteristic physical setting and objects. It is posited not only that SECs are stored in PFC, but that they are stored with category-specific localization. Using fMRI, Wood and Grafman (2003) showed that when participants made classification judgments about whether single words belonged to particular semantic categories, PFC activation patterns differed from those observed when judgments were made about whether action phrases

belonged to particular SECs (e.g., going out to dinner). Furthermore, patterns of PFC activation differed depending on whether the classified items were social in nature or not. Similar results have been reported by Zanini and colleagues (Zanini, 2008; Zanini, Rumiati, & Shallice, 2002). One of the factors that distinguish SECs from other types of memory representations is the inclusion of information regarding the sequencing of behaviors over time. For example, Sirigu et al. (1996) examined the selection and temporal organization of actions among normal controls and patients with lesions to either the PFC or to more posterior regions. This study used the same paradigm that Zalla et al. (1998, 2000) used to measure event knowledge in patients with PD (see Section 5). Participants were given cards printed with the steps in a set of four everyday activities, and asked to sort the cards to separate the activities and place the steps in order. Patients with PFC lesions were more likely to place steps out of order, and more likely to intrude steps from one activity into another. Research by Humphreys and colleagues has directly compared action observation and action performance, suggesting that a common deficit in event knowledge can impair both (Humphreys & Forde, 1998; Humphreys, Forde, & Riddoch, 2001).

A second potential cause of action disorganization syndrome is disruption to the ability to maintain representations of one's current actions and goals online. It has been proposed that the PFC maintains representations of one's current goals and task (e.g., Miller & Cohen, 2001; Mushiake et al., 2009). This proposal is based in part on the finding that PFC neurons exhibit sustained firing during memory and other tasks (e.g., Fuster & Alexander, 1971; Levy & Goldman-Rakic, 2000). In human fMRI studies, sustained activity is found in PFC when participants attempt to maintain information over a delay (Wager & Smith, 2003). Some individual cells in monkey PFC are sensitive to which task the monkey is to perform, independent of the sensory input (e.g., Muhammed, Wallis, & Miller, 2006), and in human fMRI experiments PFC is sensitive to the complexity and timescale of task instructions (Koechlin & Summerfield, 2007).

Norman and Shallice (1986) proposed a model in which the posterior cortex stores representations of low-level actions, and the PFC is selectively involved when multiple low-level actions compete for activation. In these cases, competition has to be resolved using event knowledge and maintenance of current goals. This theory has been implemented recently as a computational model, which can reproduce the qualitative features of action disorganization syndrome (Cooper & Shallice, 2000, 2006). Another very different computational model proposes that goal maintenance and competition resolution are combined in a single processing framework that uses similarity structure learned from previous experiences to resolve competition (Botvinick & Plaut, 2004, 2006). Although they differ dramatically in their computational architecture, both models propose that knowledge

about event structure and maintenance of current task information is subserved by the PFC.

More generally, the available data support the view that PFC is important both for long-term knowledge about events and for the online maintenance of task and goal information. An important open question is whether these two functions are neurophysiologically dissociated. In terms of EST, event knowledge, or SECs, corresponds to event schemata, and current task and goal representations correspond to event models.

6.2. Prefrontal Lesions and Event Segmentation

The data on cognitive deficits associated with prefrontal lesions have two straightforward implications for event segmentation. First, according to EST, impairments to event schemata should reduce one's ability to use previous experience to form adaptive event models. Thus, disordered event schemata should reduce one's ability to use knowledge to support WM and long-term memory encoding. This is not a terribly original conclusion; it is one shared with many current theories of WM and long-term memory. More specific to EST, impaired event schemata should negatively affect one's ability to identify normative event boundaries—particularly for activities that are familiar and thus should have strong support from schemata in control participants. Second, EST proposes that impairments to event models should affect event segmentation and memory because impaired event models should be less effective in biasing predictions. Memory for recently encountered information should be particularly impaired—specifically information encountered within the current event. Segmentation should be broadly impaired. Again, the conclusion that memory should be impaired is not original, but the conclusion that event segmentation should be affected is.

Importantly, disruption of event schemata and disruption of event models should produce two qualitatively different event segmentation deficits. Disordered event schemata should produce stronger impairments for more familiar activities—those for which one has a schema. Disordered event models should produce global impairments in event segmentation. More speculatively, one might guess that disordered event schemata would selectively impair segmentation at coarser temporal grains, because coarse-grained segmentation may be more sensitive to top-down influence (Zacks & Tversky, 2001). If both event schemata and event models were impaired, one would expect to see both types of impairment.

To our knowledge, there has been only one study of event segmentation in patients with frontal lobe lesions (Zalla, Pradat-Diehl, & Sirigu, 2003). In this experiment, participants with PFC lesions and healthy controls segmented two short movies of everyday activities at coarse and fine temporal grains. The patient group did not differ significantly from the controls in their fine segmentation, but their coarse segmentation was less

well ordered and delayed relative to the controls. The fact that coarse segmentation was selectively affected suggests, albeit weakly, that these patients had impaired event schemata. The fact that fine segmentation did not show obvious impairment suggests—again, weakly—that the patient group's event models may have been intact.

Clearly, there is a need for more data on the effect of PFC lesions on event segmentation. It would be particularly valuable to vary the familiarity of the activities to be segmented, and to directly compare event knowledge with segmentation. If impairments in segmentation track impairments in event knowledge and both are caused by PFC lesions, this would support EST's proposal that event schemata subserved by the PFC contribute to forming adaptive event models. It also would be valuable to combine event segmentation measures with measures of the memory functions of event models. The available data strongly suggest that memory for within-event information is impaired by PFC lesions (e.g., Müller & Knight, 2006). If the degree of this impairment tracks impairment in event segmentation, this would support EST's proposal that event models bias perceptual prediction. More specifically, memory impairments should predict segmentation impairments above and beyond impairments attributable to deficits in event knowledge.

In sum, lesions to the PFC are likely to be of profound consequence for event segmentation. Although there are few data that bear directly on this possibility, those that exist are consistent with it. This is important in its own right, but also is important for thinking about other conditions that affect the PFC. We turn now to two such circumstances—adult aging and AD.

7. Aging

While clearly neither a neuropsychological nor a cognitive disorder, normal aging has been associated with a host of changes in brain and behavior. Perhaps, the most concrete age-related change is reduction in brain weight and volume. Postmortem studies show that total brain weight declines by about 2% per decade over progression from early to late adulthood (Kemper, 1994) and *in vivo* volumetry MRI studies show median correlations between brain volume and age to be about −0.5 (Raz, 1996). However, reduced volume, and age-related changes in general, occur differentially across different brain regions. Here, we will focus on changes in two brain systems that are relevant to event understanding: the PFC and neuromodulatory systems in the midbrain.

7.1. Prefrontal Cortex

Although some regions (e.g., primary sensory cortices) show very little age-associated shrinkage, reduction in PFC volume is severe (e.g., Raz et al., 1997). Perhaps more meaningful, age-associated reductions in synaptic density and dendritic arborization (e.g., Liu, Erikson, & Brun, 1996) and in resting cerebral blood flow (e.g., Shaw et al., 1984) are greatest in the PFC. Evidence that physiological changes are most pronounced in PFC accords with findings showing age-related cognitive deficits specifically in tasks that are thought to depend on the PFC. For example, WM tasks measure ability to maintain information in a readily available state while simultaneously performing other cognitive operations of varying complexity. Numerous studies have shown age-related deficits in WM tasks (e.g., Belleville, Rouleau, & Caza, 1998; Hartman, Dumas, & Nielsen, 2001; Verhaeghen & Salthouse, 1997; see Hasher & Zacks, 1988, for review). Damage to lateral PFC regions impairs performance on a range of WM tasks (e.g., Baldo & Shimamura, 2000; D'Esposito & Postle, 1999; Goldman-Rakic, 1987; Hartley et al., 1998). Also, neuroimaging studies show that, during the retention interval of WM tasks, dorsolateral PFC increases in activity as the degree of concurrent information processing increases (see Cabeza & Nyberg, 2000; D'Esposito & Postle, 1999; D'Esposito et al., 1998, for reviews).

Attentional control, which also shows age-related decline, is another specific cognitive function thought to depend on PFC (see Posner & Peterson, 1990, for a review). There are a number of different tasks that are used to measure attentional control. Selective attention tasks require deployment of attention to a particular channel (e.g., left or right ear). Focused attention tasks might require maintenance of attention on a particular target or region of space, while divided attention tasks might tap the ability to monitor several stimuli at once, or to rapidly switch attention between multiple targets. Performance on tasks that assess attentional control declines with age. For example, divided attention costs have been shown to be greater in older than in younger adults (e.g., Hartley, 1992, 1993). Hasher, Zacks, and colleagues have presented an inhibition-deficit view of cognitive aging. According to this view, many age-related cognitive deficits are due to a decreased ability to limit access to WM and delete unwanted information from WM (e.g., Hasher & Zacks, 1988; Hasher, Zacks, & May, 1999; Zacks & Hasher, 1994). For example, participants were presented with italicized passages containing distracting text (in regular font) and instructed to read the italicized and ignore the regular font text (Connelly, Hasher, & Zacks, 1991). Older adults showed slower reading times and poorer comprehension, indicating reduced ability to focus attention on only the relevant portions of the text. Providing support for this interpretation, recent results show that older adults actually retain more of the to-be-ignored material as evidenced by

implicit memory tests (Thomas & Hasher, 2009, submitted for publication). Recent work by Hasher and colleagues suggests that declines in prefrontal mediated inhibition of distracting information are responsible for age-related declines in episodic memory (Healey, Campbell, & Hasher, 2008; Stevens, Hasher, Chiew, & Grady, 2008).

In sum, the volume and structural integrity of the PFC decline with age. These declines are associated with reduced WM capacity and attentional control.

7.2. Midbrain Neuromodulatory Systems

Neuromodulatory systems whose neurons have cell bodies in the midbrain may undergo significant age-related changes. As described in Section 2, neurons in the anterior LC signal with norepinephrine, project broadly to the forebrain, and may code error signals. These cells show attrition with age (e.g., Chan-Palay & Asan, 1989a,b; McGeer & McGeer, 1989). Evidence of age-related decreases in the dopamine system comes from several findings. First, postmortem studies have shown an age-related decrease in the number of dopamine neurons (Fearnley & Lees, 1991). Also, D2 receptor binding in the striatum has been shown to decline with age (Sakata, Farooqui, & Prasad, 1992). In a particularly relevant study (Volkow et al., 1998), striatal D2 receptor binding in adults ranging from 24 to 86 years of age was assessed using positron emission tomography (PET). A cognitive battery including the WCST was also administered. Consistent with previous findings, D2 receptor binding in caudate and putamen decreased with age. In addition, a significant relationship between receptor binding and cognitive performance remained even after controlling for the effects of age. This strengthens the observed relationship between decreased dopaminergic system activity and cognitive deficits. In sum, midbrain neuromodulatory systems involved in signaling errors show age-related declines, and these may be related to changes in cognitive function.

7.3. Episodic Memory and Situation Model Construction

We have established that age-related differences in WM and attentional control are substantial and have been associated with differences in specific brain structures. Age-related differences in episodic memory are also substantial. However, the medial temporal lobes, which are critical to episodic memory formation (Squire & Zola-Morgan, 1991), undergo minimal change with healthy aging (Head, Snyder, Girton, Morris, & Buckner, 2005; Raz, 2000). One possibility is that age-related declines in episodic memory are due to changes in controlled processing during encoding and retrieval, which may be mediated by the PFC (Healey et al., 2008; Stevens et al., 2008).

Older adults have particular difficulty remembering contextual aspects of studied material. For example, memory is poorer for perceptual details such as the color, case, or font in which target material appeared (e.g., Kausler & Puckett, 1981; Naveh-Benjamin & Craik, 1995), location of target material (e.g., Chalfonte & Johnson, 1996; Uttl & Graf, 1993), temporal order of target material (Dumas & Hartman, 2003; Kausler, Salthouse, & Saults, 1988), and even whether the target material was presented visually or auditorially (Light, La Voie, Valencia-Laver, Albertson-Owens, & Mead, 1992). Accordingly, older adults are also less likely to correctly identify the source of a memory, for example, was the stimulus, seen or imagined (e.g., Norman & Schacter, 1997). This age-related deficit in source memory has been tied to differences in activity in the PFC (e.g., Swick, Senkfor, & Van Petten, 2006).

Although aging is associated with significant deficits in memory for events, particularly for their contextual details, some aspects of event memory show striking preservation. There is evidence that reading and comprehending prose is facilitated by the construction of *situation models*, and that older adults rely on situation models during comprehension as much as younger adults. Situation models are higher level representations that describe the gist of the situation described in the text (e.g., Zwaan & Radvansky, 1998). For example, reading the sentence "She entered the hotel lobby" might result in the formation of a situation model wherein there is a hotel lobby with a reception desk and elevators, even though these contextual details were not in the text. Rather, they were supplied by semantic memory for what makes up a hotel lobby. Zwaan and Radvansky (1998) distinguish between a *current* model, which represents the current state of affairs and is updated at boundaries between events, and an *integrated* model of the current event together with all the previous ones. The final integrated model (or *complete* model) determines later episodic memory. In the terms of EST, current models correspond to event models, and semantic memory for events is provided by event schemata.

Studies have shown that these situation models are maintained and updated to similar extents by younger and older adults. For example, in a study by Morrow, Leirer, Altieri, and Fitzsimmons (1994), younger and older participants read narratives that described a protagonist moving from room to room. When reading was interrupted by probe questions about certain objects mentioned in the texts, answers were faster and more accurate for objects that were closer to the protagonist's current location, for both younger and older adults. This suggests that readers in both age groups maintained spatial situation models that were updated to reflect the protagonist's current location. While a number of studies on discourse processing show older adults are able to construct and maintain situation models (e.g., Radvansky & Curiel, 1998; Radvansky, Zacks, & Hasher, 1996), some suggest that the use of such models may be more demanding

for older adults (Morrow et al., 1994; Morrow, Stine-Morrow, Leirer, Andrassy, & Kahn, 1997).

It is important to distinguish between the proposal that older adults rely heavily on situation models and the proposal that situation model processing is unaffected by aging. The data seem clear that older adults rely at least as heavily on situation models as younger adults. One possibility is that older adults' construction and use of situation models is relatively intact, and reliance on them is an adaptive response to compensate for deficits in other processing domains (Radvansky & Dijkstra, 2007). However, it is also possible that older adults' situation models are impaired but still exert a heavy influence on comprehension. This could come about because older adults prioritize global gist in comprehension over the processing of fine details (Stine-Morrow, Gagne, Morrow, & DeWall, 2004). It could also come about because it is difficult to implement comprehension strategies that do not rely heavily on situation models, even if they would be adaptive. In our view, the currently available data provide strong evidence that older adults rely heavily on situation models, but are less convincing in showing that those situation models are not negatively impacted by aging.

7.4. Aging and Event Segmentation

The neurocognitive changes associated with aging make contact with the mechanisms of event segmentation at multiple points. The data reviewed above suggest three ways in which event segmentation may change with aging. The first two possibilities follow directly from the preceding discussion of the effects of PFC lesions on event understanding (see "Frontal Lobe Lesions," above). First, PFC dysfunction may indicate that event model maintenance is impaired in aging. As illustrated previously, both WM and attentional control are associated with PFC function. Moreover, current theories suggest that attentional control plays a central role in determining WM capacity (Baddeley, 1986; Kane et al., 2004; McCabe, Roediger, McDaniel, Balota, & Hambrick, 2006). But what is attentional control? One view is that attentional control is the ability to maintain task-relevant information in the face of distracting sensory stimulation (Darowski, Helder, Zacks, Hasher, & Hambrick, 2008). Another view is that attentional control is the ability to maintain a representation of one's current task and goals (Braver & Cohen, 2001; Miller & Cohen, 2001). These proposals lead to the suggestion that changes in the ability to maintain appropriate event models and update them adaptively could be at the core of age-related differences in attentional control, accounting for some of the age differences in cognitive performance.

Second, PFC dysfunction may indicate that event schemata are impaired with aging. This is possible, but seems less likely than the possibility that event models are impaired. One reason to doubt that event schemata are

impaired in older adults is that other domains of semantic knowledge, such as those measured by vocabulary tests, show no impairments—rather, older adults often show better performance than younger adults (Verhaeghen, 2003). Further, older adults' scripts for everyday events do not differ systematically in their structure or content from those of younger adults (Rosen, Caplan, Sheesley, Rodriguez, & Grafman, 2003). Finally, as we have shown above, older adults appear to make as heavy use of situational knowledge as do younger adults in text comprehension and memory.

Third, reductions in the efficacy of the D2 or norepinephrine systems could produce deficits in the ability to update event models in response to spikes in prediction error. Deficits in either prediction error calculation or in error-based updating would be expected to introduce noise into the timecourse of event model updating. Although a simple change to the system, such a deficit would have cascading effects: If event models are updated at inappropriate times they will form less adaptive representations of the current situation. These representations should be reflected in poorer comprehension and performance online, and in poorer later memory.

We believe that the available data suggest most strongly the possibility of age-related declines in the maintenance of event models, in their updating in response to prediction error spikes, or both. Either possibility predicts that event segmentation should become less reliable and less adaptive with age. Support for this proposal comes from a study using the event segmentation paradigm described above (Zacks, Speer, et al., 2006). Older and younger participants watched movies of actors engaged in everyday activities (e.g., making a bed) and indicated when they believed one natural meaningful unit of activity had ended and another had begun. Then participants performed an order memory task in which they were given 12 cards with still pictures taken from each movie, randomly ordered, and asked to sort them into the order in which they had occurred in the movie. Participants also performed a recognition memory task for each movie. On each trial, participants were shown one picture from the movie they had viewed and one picture from a similar movie, and asked to choose the picture from the movie they had seen. Finally, participants also completed a psychometric battery, including a measure of semantic memory for event order, the Picture Arrangement subtest of the WAIS (Wechsler, 1997). In the Picture Arrangement test, participants are given a set of cartoon drawings for a common activity (e.g., going fishing) and asked to sort them into the order in which they typically occur. Thus, whereas the order memory test is a measure of one's *episodic* memory for the order of events in a particular experienced activity, the Picture Arrangement test is a measure of semantic knowledge about how events typically unfold. This may be said to measure the accuracy and depth of participants' event schemata.

There were no systematic differences in boundary location between older and younger adults, which allowed calculation of *segmentation*

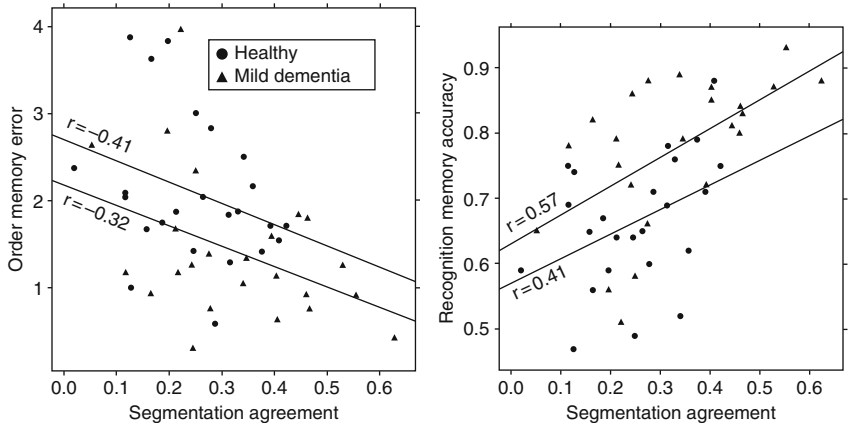

Figure 3 Event segmentation in older adults correlates with memory for event order (left) and recognition memory (right). For recognition memory, this relationship remained statistically significant after controlling for clinical dementia status and psychometric performance. (Data from Zacks, Speer, et al., 2006; Zacks, Swallow, et al., 2006.)

agreement scores by comparing each individual's segmentation to that of the sample as a whole. Segmentation agreement was lower for older adults than younger adults; in other words, older adults' segmentation was more variable. Older adults also had poorer order memory and recognition memory. Most importantly, for older adults, after controlling for global psychometric performance, segmentation agreement was significantly correlated with memory scores. Thus, those older adults who identified boundaries in a more normative fashion showed better memory for the movies (see Figure 3). Although further work is needed, it appears that age-related dysfunction of event segmentation mechanisms may be a causal factor in age-related episodic memory problems.

Picture Arrangement scores were significantly lower for the older than for the younger adults, and among the older adults these scores correlated with memory scores. One possibility is that in addition to maintenance problems, semantic event schemata information available to inform event models is reduced in older adults. However, as reviewed previously there is some evidence that semantic information about events is preserved with aging. Another possibility is that the variance shared between Picture Arrangement scores and episodic memory scores reflects not knowledge about events, but shared cognitive operations between the Picture Arrangement task and the memory tasks. In particular, both the order memory test and the Picture Arrangement test require participants to sort cards with pictures on them in temporal order. This may depend heavily on WM and attentional control.

Current work in our laboratory is further exploring the relations between age, event segmentation, and memory (Kurby & Zacks, under review). As noted previously, observers spontaneously group fine-grained events hierarchically into coarse events (Zacks et al. 2001; see Section 2). This hierarchical organization is weakened in older adults compared to younger adults. Moreover, like segmentation agreement, hierarchical organization predicts subsequent memory within the older adult group.

In sum, these experiments show that older adults do not segment activity in as reliable or as organized a fashion as younger adults. Across individuals, the ability to segment well predicts later memory performance. This is consistent with EST's proposal that event models partly determine episodic memory encoding. However, the available data do not do much to tell us which of the systems affected by aging is responsible for the changes in event segmentation and memory performance. We noted previously that event model maintenance and error-based updating are good candidates for mechanisms that undergo changes due to the aging process. In future research, it will be important to test directly which of these mechanisms is affected. One possibility is to measure evoked brain responses at event boundaries during passive viewing. We know that a substantial subset of healthy older adults, when asked to perform an explicit segmentation task, segment events at less normative and less effective points in time. We have proposed that the posterior cortical responses at event boundaries may reflect the consequences of error-based updating (Speer et al., 2003). If older adults have impaired updating, this would predict that older adults with poor event segmentation would show reduced responses in these areas at event boundaries during passive viewing. Alternatively, if event model maintenance is impaired, this would predict intact responses at event boundaries during passive viewing. Another possibility is to directly measure the activity of the error signaling system. Current research in our laboratory is characterizing the response of this system in younger adults using fMRI (Kurby, Zacks, & Haroutunian, 2009); in the future we plan to extend these studies to explore age differences.

8. Alzheimer's Disease

AD is a progressive neurodegenerative disease associated with old age. The earliest neuropsychological symptoms typically cited are deficits in episodic memory (e.g., Huff et al., 1987; Welsh, Butters, Hughes, Mohs, & Heyman, 1992). However, more recently it has been suggested that attentional control deficits may be observed even earlier in the disease course (e.g., Balota & Faust, 2001; Tse, Balota, Moynan, Duchek, & Jacoby, in press). As the disease progresses, memory is affected more globally

and eventually all higher order cognitive processes break down resulting in symptoms such as disorientation and loss of speech. In some respects, the changes in behavior associated with early AD resemble accelerations in the changes associated with normal aging (Storandt & Beaudreau, 2004). For example, episodic memory problems are associated with both normal aging and AD, and it is primarily the more severe memory loss that distinguishes AD. In fact, a recent study showed that 20–40% of a sample of healthy older adults had the neuropathological markers of AD and that even in this sample, the degree to which these markers were present at autopsy was correlated with premorbid cognitive function (Price et al., 2009). This raises interesting questions about the relationship between the cognitive and brain changes associated with normal aging and those associated with early-stage AD. However, cognitive deficits that are qualitatively unique to AD have also been identified (e.g., Johnson, Storandt, & Balota, 2003). In any case, research has shown a clear pattern of brain changes and cognitive deficits associated with AD.

8.1. Brain Changes and Cognitive Deficits

Definitive diagnosis of AD requires postmortem identification of characteristic intraneuronal neurofibrillary changes (tangles) and extracellular amyloid deposits (plaques) in the brain. Through postmortem examination of healthy and diseased brains, Braak and Braak (1991) identified six stages of AD development on the basis of the distribution pattern of neurofibrillary tangles (NFTs) and neuropil threads (NTs). Stage I is associated with the appearance of NFTs and NTs in the entorhinal cortex in the medial temporal lobe. In stage II, the hippocampus is also affected. Stages III and IV are marked by denser accumulation of markers in these areas and some spreading to other limbic structures. In stage V, neocortical association areas are affected and by stage VI primary cortical areas are affected as well.

The potential causal relationship between the appearance of these neuropathological markers and the clinical course of AD is complex and not fully understood. For example, although amyloid plaques are commonly thought to be causally related to AD (e.g., Hardy & Higgins, 1992), they are found in significant percentages of cognitively normal older adults (e.g., Arriagada, Marzloff, & Hyman, 1992; Mintun et al. 2006; Price et al., 2009; Sperling et al., 2009). However, there is little doubt that the progression of AD is marked by the accumulation of these markers in specific areas (e.g., Berg et al., 1998; Martin et al., 1987; Price & Morris, 2004), and that the presence of these markers in a particular region is associated with neuronal dysfunction in that region (e.g., Berg et al.; Hardy, 2002). For example, Kanne, Balota, McKeel, Storandt, and Morris (1998) showed evidence that accumulation of cored senile plaques (late-stage amyloid deposits) in specific brain areas was associated with deficits on specific cognitive tasks believed to

involve those areas. A large sample of participants with mild and very mild AD completed a cognitive test battery. A factor analysis identified three factors: a mental control/frontal factor, a memory-verbal/temporal factor, and a visuospatial/parietal factor. Forty-one of these participants came to autopsy an average of 5.1 years after testing. The relative density of senile plaques in each region was correlated with performance on that region's putative corresponding psychometric factor. This study provides some support for the idea that the cognitive changes associated with AD provide indicators of which structures are accumulating neuropathological markers and failing in their functional duties.

Further support comes from imaging techniques that allow antemortem examination of AD-related brain changes. *In vivo* amyloid deposition can be examined using a radiological contrast compound (C-PIB) that binds specifically to amyloid plaques and can be imaged using PET. For example, Klunk et al. (2004) showed that AD is associated with C-PIB uptake in the frontal cortex, particularly the medial portion, in temporal and occipital cortices, and in the striatum as well. Using fluorodeoxyglucose PET (FDG-PET) to examine patterns of glucose metabolism in the brain, the authors also showed that these regions were associated with reduced glucose metabolism. Subsequently, Buckner et al. (2005) presented converging measures showing AD pathology in a similar network of brain regions. In addition to atrophy in the MTL, early AD was associated with atrophy (as identified by structural MRI), amyloid deposition, and reduced metabolism in precuneus, posterior cingulate, and lateral temporal and parietal regions. It is noteworthy that atrophy in the MTL and precuneus was observed in very early stages, and even in healthy converters who were not diagnosed until later.

Work involving radiological contrast compounds that bind to NFTs is in very early stages of development. Already, there is some evidence that binding of compounds with an affinity for both plaques *and* tangles across temporal, parietal, posterior cingulate, and frontal regions differentiates between normal controls and AD patients better than FDG-PET or brain volume as measured by MRI (Small et al., 2006). As the *in vivo* imaging of amyloid plaques and NFTs improves, a clearer picture of the relationship between the accumulation of these markers in specific areas and the clinical course of AD will emerge (for more see Hardy & Higgins, 1992; Price & Morris, 2004).

The general progression of AD neuropathology identified by Braak and Braak (1991), from medial temporal structures, throughout the limbic system, cortical association areas, and eventually to the entire neocortex is supported by imaging studies of brain volume (Devanand et al., 2007; Henneman et al., 2009) and metabolism (Dickerson & Sperling, 2008; Li et al., 2008). This is in keeping with the observation of episodic memory deficits in early AD (e.g., Huff et al., 1987; Welsh et al., 1992). However,

there is also evidence suggesting that the precuneus shows atrophy, and the medial frontal cortex accumulates amyloid very early in the disease course (e.g., Buckner et al., 2005). Both of these regions have been associated with attention (e.g., Mao, Zhou, Zhou, & Han, 2007; Nagahama et al., 1999; Thienel et al., 2009). Accordingly, deficits in attentional control are observed in very early-stage AD (Perry & Hodges, 1999; Rizzo, Anderson, Dawson, Myers, & Ball, 2000; Tse et al., in press) and even identify healthy older adults who will subsequently convert to AD (e.g., Balota et al., in press; Twamley, Ropacki, & Bondi, 2006). Although the neurophysiological correlates of changes in attention in AD are not currently well understood (Hirao et al., 2005; Johnson et al., 1998), the literature does indicate that changes in attention and the precuneus, as well changes in memory and the MTL, may characterize early and even preclinical AD.

Recently, researchers have been particularly interested in a network of regions that show greater activity during rest or in passive control conditions than during focused cognitive tasks. These include a set of midline regions in the anterior and posterior cortex and regions in lateral parietal cortex. Dubbed the "default mode network" (DMN; Raichle et al., 2001), this network has been proposed to subserve a set of tasks performed on an ongoing basis to sustain normal functioning. Interestingly, the brain regions identified above as particularly vulnerable to early amyloid deposition (i.e., MTL, medial parietal and prefrontal areas) show considerable overlap with the DMN. The DMN appears to increase in activity during episodic and autobiographical memory retrieval, and decrease in activity when attention to external stimuli is required (e.g., Shulman et al., 1997; Svoboda, McKinnon, & Levine, 2006; Wagner, Shannon, Kahn, & Buckner, 2005). Within the DMN, AD patients show increased amyloid accumulation and disrupted neural activity, for example, decreased connectivity (e.g., Bai et al., 2008; Buckner et al., 2005; Greicius, Srivastava, Reiss, & Menon, 2004). Even in older adults without dementia, high levels of amyloid deposition in the DMN have been associated with abnormal neural activity in this network during memory tasks as measured by fMRI (Sperling et al., 2009). While work relating the DMN to AD is in early stages of development, results to date support the connection between biomarker deposition in the DMN and cognitive dysfunction observed in AD.

In sum, evidence suggests that the MTL and the precuneus are affected earliest in the course of AD, followed by other cortical regions such as the posterior cingulate, temporoparietal region and the medial frontal cortex (e.g., Buckner et al., 2005). These brain changes correspond, at least partially, to the cognitive changes in the disease: Episodic memory and attention are selectively affected early on; further deterioration in these areas is observed in the middle stages, and in the late stages cognition is globally impaired.

8.2. Alzheimer's Disease and Event Segmentation

This progression suggests that the effects of early-stage AD on event segmentation should resemble exaggerated versions of the effects of aging. Event segmentation itself may be little affected by selective lesions to the MTL memory system. However, such lesions predict that event segmentation has an exaggerated effect on memory accessibility. Among healthy adults, the ability to remember details from a narrative is reduced if the narrative includes a change likely to trigger an event boundary (e.g., temporal or spatial shift) since the mention of such details (e.g., Speer & Zacks, 2005). Given the importance of the MTL for retrieval of items no longer maintained in WM, or no longer in the current event model, we would expect even poorer memory for details requiring retrieval across event boundaries among early AD patients.

There is also reason to believe that AD-related neuropathology in medial posterior regions, particularly the precuneus and the posterior cingulate, would have negative consequences for event segmentation mechanisms. As described previously, research in our laboratory suggests that these regions are part of a network involved in event segmentation, which shows transient increases when perceivers experience event boundaries during comprehension (see Section 2 above). We suggest that these posterior regions may be important either for detecting changes in the various dimensions that define events (e.g., time, space, actors, goals, etc.), or in providing inputs to event models when error-based gating mechanisms update a current event model. Either way, AD-related dysfunction in the posterior cingulate and precuneus might be expected to interfere with the updating of event models that no longer provide accurate predictions. Given that event models serve to guide attention, this could manifest as the type of attention problems observed in very early AD.

Although we have focused on how AD-related brain changes might affect event segmentation mechanisms, it is also possible that such mechanisms might be preserved, particularly earlier in the disease course. This possibility is supported by the fact that there is relatively little overlap between the brain regions associated with EST (see Figure 1) and those affected by early-stage AD pathology described above. Previous work in our laboratory with older adults, both healthy and with very mild AD, suggests that individual differences in event segmentation predict event memory independently of clinical dementia status (Zacks, Speer, et al., 2006). Work is currently underway, using larger sample sizes, which will enable us to ask whether the strength of the relationship between event segmentation and event memory varies across levels of clinical dementia status. If this relationship is as strong among early-stage AD patients as among healthy older adults, this would suggest that some mechanisms of event segmentation are independent of those degraded in the early stages of the disease. The finding that mechanisms of event segmentation

are robust against the moderate neural lesions of early-stage AD would have an important clinical application: Event segmentation would be an attractive target for training to remediate memory deficits. One possibility is that deliberate attention to event segmentation itself will improve memory encoding. In addition, imaging data will afford the opportunity to ask whether structural integrity in certain brain regions mediates the relation between event segmentation and memory. According to EST, effects in PFC would suggest that early dementia affects either the formation of event models or the use of event knowledge. Effects in posterior cortex would suggest early dementia affects either the processes of detecting an event boundary or of updating an event model.

In the later stages of AD, damage to neural integrity is widespread, and deficits in cognition are comparably broad. Early in the disease progression, the encoding of new memories is affected but the retrieval of previously learned material is preserved (e.g., Huff et al., 1987; Welsh et al., 1992). As the disease progresses, access to autobiographical memories declines. In the later stages, even the most overlearned semantic associations are lost. At this point, in addition to the frontal maintenance problems discussed above, it is likely that reliable event schemata are no longer available or accessible. Accordingly, the perceptual guidance provided by event models is likely to be severely limited. This represents a fundamental breakdown of the event segmentation system and would have wide ranging deleterious consequences such as those observed in advanced AD, for example, disorientation. However, at this stage in the disease, global cognitive function has deteriorated to the point where drawing connections to EST may be of limited value.

In sum, the brain changes associated with early AD may lead to attention and memory problems by way of disruption of event segmentation mechanisms. Alternatively, it may be that event segmentation abilities, or certain aspects thereof, are relatively well preserved in AD. In the latter case, clinical efforts to maximize the cognitive burden carried by particularly well-preserved event segmentation mechanisms may reduce attention and memory problems. Work is currently underway that will begin to address these possibilities.

9. Conclusions

We have reviewed a complex and diverse set of clinical neuroscientific circumstances—and there are many more we have had to leave to the side for lack of space. A heuristic overview of the pattern of deficits we have observed is provided in Table 1. We would like to emphasize that the set of mechanisms we have examined, as well as the set of conditions, is selective. For example, we have not discussed the role of the medial temporal episodic memory system (Cohen & Eichenbaum, 1995) in event understanding and

Table 1 Overview of Potential Event Segmentation Mechanism Impairments.

	Sensory-perceptual processing	Prediction monitoring	Error-based updating	Event models	Event schemata
Schizophrenia	0	0	+	++	0
Obsessive-compulsive disorder	0	+	+	0	+
Parkinson's disease	0	0	+	0	0
Frontal lobe lesions	0	0	0	+	++
Aging	0	0	+	+	0
Alzheimer's disease	0	+	+	+	+

+: Suggestive evidence for impairment; ++: strong evidence for impairment; 0: not yet tested.

memory, nor have we considered persons who experience anterograde amnesia after damage to this system. For none of the conditions we have examined do we find evidence for deficits in sensory-perceptual processing. However, in other conditions—for example, visual form agnosia or motion blindness—deficits in sensory-perceptual processing are clearly evident and likely have important consequences for event segmentation.

We believe the picture that emerges from this review underwrites a strong message: The mechanisms of event segmentation provide a valuable framework for understanding cognitive dysfunction. This provides an exciting leverage point for clinical diagnosis and treatment. People, including those of us who are aging or coping with a neurological or neuropsychiatric condition, tend to care about their ability to comprehend the everyday events around them, to remember those events later, and to plan adaptive actions. Theory-driven interventions that may improve event comprehension and memory have the potential to substantially improve quality of life. As we have described throughout the chapter, researchers coming from a range of theoretical perspectives are applying such interventions to a range of clinical problems. We are hopeful that the current chapter illustrates how EST may contribute to this effort.

However, the basic science base underlying such interventions needs extending on at least two fronts. First, there is an urgent need for many more data on event understanding in clinical populations and in healthy aging. One can draw inferences about the mechanisms of event segmentation from the available data concerning attention, memory, and performance. However, such inferences are necessarily weak and invite direct verification. Second, there is a need for formal models that make fine-grained predictions about the consequences of specific neurological changes for specific aspects of event segmentation and memory. An initial step in this direction was taken with the computational model of Reynolds et al. (2007). This model was a connectionist implementation of the core architecture of EST. It would be valuable to extend this model to produce moment-by-moment predictions for event perception and memory. Virtual lesions could then be applied to the model, and the model's performance could be directly compared with that of patients from the groups discussed here. Such comparisons would provide powerful means to constrain theories of event understanding and to characterize the cognitive dysfunction in these conditions.

Clearly, there is much work to be done. We believe this is an exciting time for researchers studying deficits in higher cognition. New landscapes of theory and methods are opening up—the lens of event segmentation that we have applied here can encompass only a small field of view over this terrain. Basic scientists who wish to better understand how people comprehend and remember the everyday events that make up their lives have a lot to gain by taking up this exploration. Those with disorders of event perception also stand to benefit from this endeavor.

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