Explicit Memory and Brain-Electrical Activity in 10-month-old Infants

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(ABSTRACT)

One of the most intriguing and enduring issues in contemporary developmental cognitive neuroscience centers on the development of the ability to remember past experiences and the neural systems which support this capacity. Over the past 25 years, through methodological advancements and direct challenges to established assumptions, the focus of this developmental question has shifted to highlight the second half of the first year of life as the time when true explicit memory functionally emerges and begins to rapidly develop. The purpose of the following study was to test specific hypotheses regarding the biobehavioral development of explicit memory during infancy and present a new approach to studying the behavioral and physiological expression of this system. This study, which was guided by hypothesized neural substrates of this memory system, is the first direct investigation of continuous brain electrical activity during both the encoding and retrieval phases of explicit memory processing in infants. Memory-related differences in behavior and task-related brain activity in individual cortical areas were of particular interest.

The results of this study provided some support for the hypothesis that baseline-to-task changes in EEG power can distinguish between successful and unsuccessful ordered-recall memory. Specifically, decreases in brain-electrical activity relative to a baseline period were found at both frontal and temporal locations during stimulus encoding and retrieval for infants who failed the recall tests. However, either no change, or increases in EEG power at frontal and temporal sites was related to successful performance on this task. In addition, different patterns of brain-electrical activity were present for correct and incorrect responses from the same child.

This study contributes to our understanding of the biobehavioral expression of infant explicit memory in three main ways. First, changes in both frontal and temporal lobe activity are directly involved in explicit memory processing both during event encoding as well as retrieval. Second, this work provides evidence of a developmentally appropriate and valid pattern of electrophysiology specific to explicit memory processing. Finally, this study bridges the gap between a classic behavioral task of infant memory (which has been conceptually linked to neuropsychological data) and current developmental cognitive neuroscience.

Dedication

I would like to dedicate this work to my husband, Mike. Without your strength, gentle encouragement, and humor, this dream would not have been realized. Thank you for the life we've built together, the love you give so endlessly, and for making me a Mommy.

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Introduction and Background

Over the past several decades, an increasing number of researchers have argued that memory is not a unitary process, but rather is comprised of two or more biobehavioral systems that serve separate functions and operate according to different principles (Eichenbaum, 1997). Explicit memory involves the capacity for recall and recognition of names, places, dates, events, and other kinds of episodic knowledge (Tulving & Donaldson, 1972; Tulving, 1993; Graf & Schacter, 1985; Bauer, Wenner, Dropik, & Wewerka, 2000). It is characterized as rapid (supporting one-trial learning), fallible (memory traces degrade, retrieval failure occurs), and flexible (not associated with a specific modality or context). Importantly, explicit memory is characterized by conscious processing, and the awareness that current behaviors are enhanced or changed as a result of a previous experience (Tulving, 1993; Bauer, 2004). Explicit memories can be consciously brought to mind as representations in the absence of ongoing perceptual support (Nelson, 1995). Tasks assessing explicit memory share a required recollection of a specific episode.

In contrast, implicit (or *non-declarative*) memory comprises a variety of non-conscious abilities, including the capacity for learning habits and skills, priming, and some forms of classical conditioning (Parkin, 1997). Implicit learning is characterized as slow, resulting from gradual or incremental learning usually as a result of repeated motor practice (Sherry & Schacter, 1987). It is both reliable (memory traces are less likely to degrade once the skill has been acquired), and inflexible (associated with a specific modality or context). Unlike explicit memory, implicit memory functions outside of consciousness (Graf & Schacter, 1985), without

the individual's awareness that the current behavior is affected by previous experiences. Tasks assessing implicit memory do not require the recollection of a specific episode (Nelson, 1995).

The adult cognitive neuroscience literature supports the distinction between explicit and implicit forms of memory with evidence of individual differences in these memory abilities in healthy adults, from dissociated deficits in adults with specific brain damage and disease, and in lesion studies with animal models, as well as results from neuroimaging studies (Schacter, Wagner, & Buckner, 2000). However, this distinction has not been universally adopted in developmental science. The chief argument against the existence of unique explicit and implicit systems is that one of the required components of explicit functioning is "conscious access" to the memory trace, and it is difficult to determine whether infants are consciously recalling a memory if they cannot provide a verbal report (e.g. Rovee-Collier, 1997). Because these different forms of memory vary according to their function, context-sensitivity, rules of operation, and different neural substrates, one assumption of this study is that these systems are, indeed, distinct, and that explicit memory is a real and measurable construct in infancy.

In this document, I first describe the challenges unique to the study of explicit memory function during infancy and present an argument detailing why imitation-based tasks are an appropriate measure of infant recall abilities. Next, I review the current literature regarding infant recall abilities during the second half of the first year of life, a period marked by tremendous changes in infants' behavioral repertoires and neurological development. Then, I discuss evidence of the neural substrates underlying explicit memory function and how the development of these structures, and the integrative circuit linking them, may mirror concurrent behavioral developments during this period. Additionally, I highlight recent research directly examining the relations between evoked neural activity and recall function towards the end of the

first year of life. Finally, I describe the current study and present and interpret the results of this concurrent investigation of behavioral and electrophysiological indices of recall in 10-month-old infants.

Measuring Explicit Memory in Infancy

Because infants and very young children are pre-verbal, in order to study the development of the ability to recall the past, a nonverbal analogue to conscious verbal recall is required. Indeed, the expression of explicit memory must be evoked from a task that demands the same cognitive processes as those involved in verbal recall, yet does not require a verbal response (Bauer, DeBoer & Lukowski, 2007). When adults verbally report a past experience, it is clear that the memory has been made consciously available. However, this confirmation is unavailable when examining explicit memory development with infants and very young children.

Recently, imitation-based tests are becoming increasingly common and recognized as acceptable infant analogues to verbal report of recall processing. Imitation paradigms involve an observational learning procedure that exploits infants' natural tendency to reproduce the actions of other people in their environment (Hayne, 2004, 2007). Generally, in imitation paradigms, interesting props are used by the experimenter to model a specific action or action-sequence. Either immediately after the demonstration (elicited imitation), after some delay (in the case of deferred imitation), or both, infants are allowed to imitate the modeled actions (Bauer, 2004; Lukowski, Wiebe, Haight, DeBoer, Nelson, & Bauer, 2005). Dependent measures of interest during this test are the number of target actions reproduced and/or the number of pairs of correctly ordered actions (sequences) which the infant reproduces. The average quantity of target actions or sequences reproduced is then compared to either a between-subjects control group or a within-subjects measure of spontaneous production of the target events and

sequences. Recall of the event is inferred when the production of imitation behaviors is significantly higher after the modeled sequence than under appropriate control conditions. Bauer and colleagues (for a review see Bauer, 2006; Bauer, Wiebe, Carver, Lukowski, Haight, Waters, & Nelson, 2006; Bauer et al., 2007; Lukowski et al., 2005) set forth the following four-point argument that highlights some of the most compelling evidence that infant imitation-based tasks are appropriate tests of early recall abilities, an argument which has been developed in detail elsewhere (e.g. Bauer, Wiebe, Carver, Waters, & Nelson, 2003; Carver, Bauer, & Nelson., 2000; Lukowski et al., 2005; Mandler, 1990; Meltzoff, 1990; Nelson & Fivush, 2000).

First, deferred imitation of a display is one of the hallmarks of representational function (e.g., Piaget, 1952). Piaget was the first to note the importance of imitation studies for studies of memory development. According to his theory, imitation that occurred following a delay signaled the infant's ability to form a mental representation of the model's behavior and to retain that representation over the retention interval. Piaget believed that deferred imitation marks the final portion of the sensorimotor stage when the ability to form and utilize mental representations has emerged. He believed that the development of this explicit memory system was a necessary precursor to the onset of language. However, empirical studies have now shown that Piaget underestimated the developmental emergence of imitation, as he theorized facial imitation emerged between 8 and 12 months of age (Meltzoff & Moore, 1977, reported facial imitation by 6-week-old infants), and object imitation between 18 and 24 months (though constrained, object imitation has been observed after a 24 hour delay in infants as young as 6 months; Barr, Dowden & Hayne, 1996; Hayne, Boniface & Barr, 2000). Nevertheless, Piaget's views about the relation between imitation and memory continue to provide the theoretical cornerstone for research in this area.

Second, once children are linguistically capable, they describe past events experienced in the context of imitation (e.g. Bauer, Wenner & Kroupina, 2002; Myers, Clifton, & Clarkson, 1987). This is compelling evidence that the encoded representations are explicit or declarative memories, as opposed to implicit or non-declarative (as these memory processes are not conducive to verbal report). In addition, Hayne & Herbert (2004) demonstrated that verbal cues enhance memory retrieval by participants who are not yet fluent speakers themselves (18-month-olds). Interestingly, they found that general prompts to capture the infants' attention during the display or to encourage action during the memory test (i.e. "Look what this does!") did not increase recall performance as much as explicit object and action labeling did. This mnemonic malleability to the effects of specific verbal cues serves to further support the explicit nature of the memory traces.

Third, imitation-based tests pass the "amnesia test". Temporal lobe amnesiacs tend to perform normally on a variety of implicit tasks (Cohen & Squire, 1980), but they cannot perform tasks thought to require explicit memory, such as deferred imitation (Squire, 1987). McDonough, Mandler, McKee, & Squire (1995) tested neurologically normal controls and amnesic adults with confirmed damage to the hippocampal formation and using a paradigm similar to that used with infants. Patients were shown a series of eight different action sequences on one day and, on the next day, were tested under two conditions: either instructed to imitate the previously seen sequences or simply observed to see if they do spontaneously. McDonough et al. (1995) reported that only the control participants were able to reproduce the sequences and were able to do so under both instructed and spontaneous conditions. In contrast, the amnesic participants failed to reproduce any sequences in either condition. This pattern of findings suggests that performance on the sequencing task depends on intact hippocampus or medial

temporal region, an area well-studied for its role in explicit memory processing. Similar patterns have recently been shown for individuals who experienced temporal lobe damage as children, rather than adults (Adlam, Vargha-Khadem, Mishkin, & de Haan, 2005). These studies suggest that the demands of the imitation procedure exploit the memory system that gives rise to delayed recall performance.

Finally, the process of imitation, regardless of delay, taps recall rather than recognition. The recall process involves retrieving a mental representation, which was formed by past experience, in the absence of ongoing perceptual support for that experience (Mandler, 1986). Expression of a visual preference or detection of novelty or familiarity, assessed in tests of recognition, do not require that a mental representation be created or maintained. At some level, all recall is cued, either by an external prompt providing perceptual support for the target actions, or by an internal representation maintaining the memory of the event (Spear, 1978). In the imitation paradigm, the props used to demonstrate the event are available to the infant during the recall test (indeed, infants must use them to "report" their memories) and they may provide perceptual cues to reproduce the target actions. However, information about the temporal order in which the sequence of actions occurred is no longer perceptually available and is not inherent in the test stimuli (Bauer & Wewerka, 1995). Indeed, in order to reproduce an ordered sequence, the infant must have encoded information about the temporal order of events at the time the actions were modeled and must retrieve that information from a representation of the event, in the absence of ongoing perceptual support. In this, the elicited imitation task clearly requires recall processes. Thus, the parameters and requirements of the imitation paradigm are analogous to those of verbal recall tasks (Mandler, 1990).

Recall within the first year: Explicit memory development

Imitation paradigms offer an investigation of memory consistent with information processing theory and are compatible with the modal model of human memory function proposed by Atkinson & Schiffrin (1968). This model identifies the connectivity between memory formation, storage, and retrieval processes within the context of both short- and long-term memory. Manipulation of the various components of the imitation paradigms (encoding, storage, and retrieval) allows researchers to target memory function at specific stages of this model.

By testing infants of different ages and manipulating the components of the imitation paradigm, several consistent age-related trends have been revealed in the general expression of explicit memory. During the demonstration phase, older infants can tolerate more objects being displayed and more steps per event sequence, require less time and repetition in the demonstrations, and they perform better than younger infants with arbitrarily-related (rather than enabling) events (Hayne, 2004; Bauer Wiebe, Waters & Bangston, 2001). Older infants also can tolerate longer delays between encoding and retrieval (Carver & Bauer, 2001), and reminders during the delay facilitate recall more for older infants than for younger (Bauer, Hertsgaard, & Dow, 1994). During the retrieval phase, older infants are better at exploiting retrieval cues from their environment. For example, recall performance in older infants is robust to changes (from encoding conditions) in context (Hayne, et al., 2000), objects (Herbert & Hayne, 2000), medium (live demonstration vs. televised demonstration; Barr & Hayne, 1999), and experimenter (Learmonth, Lamberth, & Rovee-Collier, 2005).

Although many developments within the first two years of life undergo gradual and continuous change, the end of the first year is highlighted by rapid changes in recall capacities. At 9 months of age, approximately 50% of infants can express recall for individual actions

immediately (Meltzoff, 1988), after delay of 24 hours (Meltzoff, 1988; Collie & Hayne, 1999) and up to one month (Carver & Bauer, 1999, 2001; Bauer et al., 2001; Bauer, et al., 2003; Bauer et al., 2006; Lukowski et al., 2005) after modeling. In addition, older infants require fewer encounters of events in order to maintain a memory over a long delay. For example, whereas 11- to 13-month-olds only need one exposure of an event to show long-term recall (Mandler & McDonough, 1995; Bauer & Hertsgaard, 1993), 9-month-olds may require multiple opportunities to encode across several days to maintain the memory over a 1 month delay (Bauer, et al., 2001).

Although some evidence of deferred imitation has been observed in infants as young as 6 months old (Barr et al., 1996; Collie & Hayne, 1999; Hayne, et al., 2000), very few of these infants produce more than one target action, and thus reports of ordered recall for infants younger than 9 months old is unavailable. By 9 months of age, most infants are capable of recalling target actions, yet recalling the order of these events is a more challenging task as approximately 50% of these infants demonstrate ordered recall (at least one pair of correctly ordered actions) after a 1 month delay, a finding which has been replicated in several independent samples, (Bauer et al., 2001; Bauer, et al., 2003; Carver & Bauer, 1999; Lukowski, et al., 2005).

However, one month later, 10-month-old infants, who are able to accurately recall actions up to 3 months after modeling (Carver & Bauer, 2001; Mandler & McDonough, 1995), are also becoming more adept at recalling the temporal sequences between actions. Wiebe, De Boer, Waters, Rademacher, Yanda, Nelson, & Bauer (2001) reported that 60% of 10-month-old infants displayed ordered recall after a 1 month delay. This capacity to demonstrate ordered recall continues to develop: by 11 months of age, 86% of infants show immediate ordered recall (Bauer

& Mandler, 1992), by 12 months of age, 58% show ordered recall after a 24-hour delay (Morasch & Bell, *under review*) and by 13 months, 78 % recall the temporal sequence of events after a 1-month delay (Bauer et al., 2000).

Therefore, whereas some infants encode, store, and retrieve memory traces for temporal order over delays, others lack either these capabilities or the ability to express them. Thus, individual differences are likely to be highest during this transitional period at the end of the first year of life. The observed individual differences and age-related changes in the encoding, storage, and retrieval of remembered material have been theoretically linked to the development of the underlying neural substrates responsible for explicit memory processing, a system which, itself, undergoes significant development towards the end of the first year.

The Neurological Foundation of Explicit Memory and Its Development

Our understanding of the neurological components supporting explicit memory function in adulthood has made tremendous progress over the past 30 years. However, how these neurological components develop and communicate to support the emergence and development of explicit memory processing (and how this process coincides with behavioral expressions of recall) in infancy is not as well understood. The majority of the evidence for the neural basis of explicit memory has relied on data collected from imaging studies of intact human adults, clinical studies of patients with neurological disorders or surgical ablations, and lesions studies from the animal literature (see Table 1).

The medial temporal lobes (MTL), consisting of the hippocampus, amygdala, and the surrounding cortical areas (e.g. parahippocampal structures such as the entorhinal cortex), are thought to be involved in the process of encoding and consolidating explicit memories. Studies of patients with temporal lobe lesions have shown that the MTL is important for acquisition of

new explicit memories (Squire, 1992) but not new implicit memories (Cohen & Squire, 1980). Indeed, temporal lobe amnesiacs tend to perform poorly on tasks designed to assess explicit memory processes, such as deferred imitation (Zola & Squire, 2000). Therefore, the integrity of MTL function is an important component of the recall process.

In addition, damage to the frontal lobe also impacts expression of delayed recall in adults. Frontal lobe patients often fail tasks of imitating actions in a displayed sequential order; even when target behaviors are reproduced, the temporal sequence connecting these actions is disrupted (Zanini, Rumiati, & Shallice, 2002). Additionally, there is evidence in frontal lobe patients for a double dissociation in the neurological substrates of memory, indicating that the frontal lobes are specifically involved in sequential tasks, whereas the parietal lobes are involved in tasks requiring visuo-spatial memory processing (Villa, Gainotti, de Bonis & Marra, 1990). These studies indicate that the integrity of the frontal lobe is especially necessary for ordered recall in adults.

There is support from both behavioral and neuropsychological studies that a specific region of the frontal lobes, the prefrontal cortex (PFC), is a critical component in the retrieval of memories from long-term storage in neocortical association areas. In their review, Wheeler, Struss, and Tulving (1997) state that the prefrontal cortex is what permits individuals to consciously access representations of past experiences. This argument suggests the PFC serves a crucial role in explicit memory processing. In addition, one of the principle and also most general functions of the PFC is the temporal organization of actions towards biological or cognitive goals (Luria, 1966; Fuster, 2002). The PFC, its lateral region in particular, specializes in the temporal structuring of new and complex goal-directed series of actions, whether in the form of behavior, speech, or reasoning (Goldman-Rakic & Leung, 2002). Further, participation

of the PFC in choosing between alternatives, in decision making, and in executing temporally structured action are the reasons that this cortex has also been considered the "central executive" (Fuster, 2002).

At the root of the temporal ordering and timing of actions is the neural process of integrating information along the time axis. The temporal organization of novel and complex behavioral sequences is not possible without the integration of temporally separate stimuli, actions, and action plans into goal-directed sequences of behavior. According to Fuster (2002), this process of integration is the essential physiological role of the PFC. The execution of temporally structured behavior is then supported by the function of this "central executive".

The most contemporary theories indicate that the capacity to produce ordered-recall of a past event may depend not upon independent neural structures, but upon a specific integrated neural system, namely a temporal-cortical circuit, involving reciprocal connectivity between MTL structures (such as the hippocampal formation and surrounding cortex), as well as higher level cortical association areas including the PFC (Nyberg, McIntosh, Cabeza, Habib, Houle, & Tulving, 1996; Liston & Kagan, 2002). In this proposed circuit, temporal lobe structures are necessary for the encoding and consolidation of new memories. Specifically, the memory trace begins as inputs from multiple neocortical association areas that converge on parahippocampal areas. Encoded aspects of the event are temporarily stored in the entorhinal cortex. The hippocampus then integrates these mnemonic components into an organized memory. During the consolidation process, the memory trace becomes distributed and deposited into long-term neocortical association storage areas. This process of consolidation may last for days or months, and during this period, memory traces are vulnerable to loss if the process is interrupted (by interference, injury, etc.). The retrieval of the memory representation has been linked

behaviorally and neurologically to the structure and function of the PFC (Nyberg et al., 1996). Thus the establishment and maintenance and delayed recall of specific event memories are supported by the integrity of a circuit connecting medial temporal lobe structures, neocortical association areas, and the PFC, as well as the reciprocal connections between these areas (Bachevalier, 1992).

If explicit memory is supported by an integrated neural network comprised of both subcortical and cortical structures, what is changing in the neural architecture to support such dramatic and well-documented developments in the expression of explicit memory as are seen during the second half of the first year of life? There is increasing speculation and evidence that in human infants, the neural substrates necessary for explicit memory undergo significant development during the second half of the first year of life.

In terms of MTL development, however, the hippocampus and surrounding cortical areas are thought to be relatively early developing structures in postnatal development (Nelson, 1995). For example, the distribution of cholinergic receptors in the monkey hippocampus is adult like at birth, in contrast to those in the cortex, which mature later (O'Neil, Friedman, Bacehevalier, & Ungerleider, 1986). In addition, the structures of the human limbic system rapidly become adult-like by the second half of the first year of life (Kretschmann, Kammradt, Krauthausen, Sauer, & Wingert, 1986). Additionally, metabolic activity in the temporal lobes increases significantly the third month of life and precedes that of the prefrontal cortex (PFC) by several months (Chugani, 1994). The only area functionally related to the hippocampus that appears to have somewhat protracted postnatal development is the area of the dentate gyrus (Eckenhoff & Rakic, 1991), and its function within explicit memory processing has not been clearly defined (although some speculate that it serves as an indirect link between cortical association areas and the hippocampal

formation). Collectively, these data support the conclusion that human and non-human primate hippocampus and surrounding structures (save the dentate gyrus) mature early in life. Thus, the late development of explicit memory, characterized by decreased vulnerability of the memory trace, is unlikely to be supported exclusively by extended MTL development.

The development of the PFC is unique in that it is both delayed (the PFC is typically not associated with task performance until the second half of the first year of life), and protracted (it continues to structurally develop from infancy and into early adulthood) (Bell, 2001, 2002; Bell & Fox, 1994; Diamond, 2001; Goldman-Rakic & Leung, 2002). In addition, performance on tasks which are dependent on the PFC also displays a linear progression throughout development. Indeed, performance on other infant tasks of delayed recall, such as object retrieval (Diamond, 2001) and a looking version of the A-not-B task (Bell, 2001) have been linked to the development of the PFC, and reflect a pattern of better performance on increasingly challenging asks with age. In addition, superior performance on this A-not-B task has been associated with larger baseline-to-task activations in 6-9Hz EEG power at frontal sites during short-term encoding, storage, and retrieval (Bell, 2001).

If the development of the temporal-cortical circuit (Nyberg et al., 1996) is driven by stable MTL function and extended PFC development and is the anatomical correlate to the changing behavioral manifestations of explicit memory, this circuit is central to studies indexing biobehavioral explicit memory development. The following section describes efforts that have been made to date to explore physiological and behavioral indices of explicit memory development.

Concurrent Electrophysiological and Behavioral Indices of Explicit Memory

For a summary of the current studies reporting biobehavioral indices of infant recall, see Table 2. Carver, et al. (2000) were the first to demonstrate that brain activity at one age (event-related potentials at 9 months) was predictive of long-term explicit memory at a later age (delayed recall at 10 months). Following an infant directed control session which established a baseline for spontaneous production of actions and sequences, the experimenters demonstrated three 2-step novel events. To maximize the likelihood of correct recall, infants were re-exposed to these demonstrations twice more within the next three days after the initial demonstrations. One week after the third demonstration, infants were tested for long-term recognition of the steps of the demonstrated sequence as well as novel events.

To examine the integrity of the event memories during the delay phase of the elicited imitation procedure, Carver et al. (2000) collected event-related potentials (ERPs) in response to infants viewing digitized photographs of actions and end states from both familiar and novel event sequences. ERPs are neural responses which are time-locked to a specific stimulus event and, based on the amplitude and latency of the waveform of the evoked potential, are primarily used in developmental psychophysiology to indicate responses to novelty or familiarity (recognition memory). Carver et al. (2000) hypothesized that based on individual differences in ERP waveforms to the pictures of familiar events, delayed recall performance could be predicted. One month after the ERP collection, infants (now 10 months old) were given their first opportunity to imitate the modeled actions (and sequences). Based on their performance in the delayed recall task, infants were classified into two groups: those who produced at least 1 correctly ordered pair of sequential actions (50% of the sample) and those who produced no ordered pairs.

Analysis of interim recognition memory (via 1 week ERP) indicated that although all infants visually attended to the stimuli (the Nc component of the ERP was present in both groups, indicating obligatory attention), the form of this component differentiated between novel and familiar events only for infants who would later go on to reproduce at least one correctly ordered pair of actions. Specifically, infants who went on to show ordered recall at 10 months showed different slow wave ERP components one week after encoding than those infants who did not recall.

The positive slow wave (PSW) component of infant ERP is taken to represent familiarity detection, recognition, and as memory-updating responses to pictures of old events. The negative slow wave (NSW) component is indicative of novelty detection, or response to pictures of new events. Therefore, in this study, a PSW should be elicited for pictures of steps from the familiar sequence, and a NSW should occur in response to photos of novel events. This pattern held true only for infants who went on to recall at least one sequential pair of events 4 weeks later. Infants who did not reproduce any ordered pairs at test (thus did not show delayed ordered recall) also exhibited evoked responses which did not distinguish between familiar and novel events 1 week after encoding. For these infants, all events were perceived as novel.

By sampling recognition memory via ERP only once during the retention interval, Carver et al. (2000) were unable to pinpoint why some infants failed to imitate though others succeeded. It could be that they failed to encode the events (or the temporal sequences connecting the events), they failed to adequately store representations of events, or because they were unable to retrieve representations of events from long-term memory. Extending the design of Carver et al. (2000) , Bauer et al. (2003) collected ERP measures of recognition memory twice during the retention interval of the delayed recall paradigm (once immediately after encoding and again one

week later). By including ERP collection immediately after the final exposure session Bauer et al. (2003) were able to better specify the point of memory trace degradation for long-term recall in 9-month-olds. They found that immediate recognition memory (defined as ERP responses at a zero delay) did not differentiate between successful and unsuccessful recall. Regardless of eventual performance on the recall test, immediately after the final demonstration, all infants responded with the expected ERP patterns to novel stimuli (large NSW response) and familiar stimuli (large PSW response for memory updating), demonstrating immediate recognition memory and intact encoding. Just as in Carver et al. (2000), the ERP's collected during the delayed recognition test were predictive of success on the delayed recall test.

Interestingly, all reported ERP findings were localized to the frontal midline scalp site (Fz), yet the neurological support of this interim recognition was attributed to the protracted development of the dentate gyrus, a structure within the medial temporal lobe. Thus, although these two studies demonstrate the benefits of incorporating ERP measures into imitation paradigms (as they can illuminate the quality of a memory trace during different stages of information processing), they do not provide conclusive evidence as to which specific neural structures and circuits are and are not actively involved with explicit memory development.

Indeed, after reviewing the extant literature, there is still no ultimate agreement on exactly what developments are responsible for changes in the expression of explicit memory in infancy or exactly which brain areas drive these changes and how. Is it the ability of the infant to effectively encode actions and/or temporal relationships among those observed events? Is it a more efficient and sophisticated process of consolidation? Is it a decrease in the susceptibility to storage failure which maintains information across long delays? Or is it the infants' increasing ability to exploit cues from the environment at the time of retrieval?

Traditionally, prior work (Bauer et al., 2000; Bauer et al., 2003; Hayne, 2004) has pointed to the developmental change in the infant's ability to exploit retrieval cues from the environment in order to evidence delayed recall. However, as has been discussed previously, in the deferred imitation paradigm, although the objects used to display retention are powerful mnemonic cues themselves, there is no ongoing perceptual support for recall of specific temporal ordering of the actions, and therefore no available cues to exploit. What might inform some infants about sequential information in the absence of perceptual support? In addition, what causes some infants to be able to correctly reproduce all three target actions out of order and infants who correctly order the recalled actions? If the PFC supports the temporal sequencing of behavior, could it be that individual differences in retrieval are related to the development and function of this particular structure or its extended cortical/temporal network? Or could it be possible that the infants who are able to extract effective retrieval cues from their environment also encoded the original information in a more enriched manner?

According to Bauer et al. (2003), the individual differences in explicit memory performance were unrelated to the encoding phase because all infants, even those who went on to generalize familiar and novel photos after one week, distinguished between old and new events on tests of immediate recognition (with ERP) just after the last encoding session. Because of this, it was concluded that all infants encoded the old events. Therefore, encoding processes were eliminated as a contributing factor to individual differences in delayed recall performance. However, no speculation was made about the quality of initial encoding, specifically that infants who went on to display ordered recall may have encoded the initial events in a more sophisticated or enriched manner.

In a recent short-term longitudinal study, however, Bauer, et al. (2006) found that whereas at both 9 and 10 months of age, infants ostensibly encoded events (differentiated familiar and novel photos in an ERP test of immediate recognition), only memories created at 10 months of age were maintained over the long retention interval (as evidenced by ordered recall). The authors argued that age-related differences in encoding processes might be responsible for this pattern of results, as 10-month-olds showed more dramatic changes in amplitude of evoked responses than at 9 months. Therefore, individual differences in the quality of encoding may contribute to individual differences in recall performance. Interestingly, the authors suggest that at 9 months, the infants may have only encoded the features of the objects used in the events, whereas at 10 months, the infants encoded both the individual actions of the events as well as the temporal order of the event-sequence (Bauer, et al., 2006).

However, in all of these studies investigating behavioral and electrophysiological indices (ERP) of explicit memory, neither behavior nor electrophysiology was examined during the actual encoding of the modeled sequences. Perhaps changes in activation of the temporal-cortical circuit during event modeling may be related to individual differences in encoding and therefore explicit memory performance. This approach would be especially valuable as behavioral measures such as the quality of looking during the demonstration phase of imitation tasks have been shown to be poor predictors of later recall performance (Courage, Howe & Squire, 2004).

More recent evidence (Bauer, 2005) specifically implicates the storage process as the stage at which memory may degrade during a long delay. Although this study was conducted with older infants (13, 16, and 20-month-olds) the inclusion of a savings assessment allowed the author to examine infants' ability to relearn forgotten familiar information quicker than learning

completely novel sequences. In this case, if recall scores improved after a relearning treatment, savings in relearning was said to have occurred. Savings occur because the memory created during the relearning process has presumably integrated with an existing but inaccessible memory trace (the original encoding). Therefore, the impaired recall was likely due to retrieval failure. If, however, the relearning process does not improve recall performance (no evidence of savings) this indicates that sometime during the storage phase (between encoding and retrieval) the memory trace degraded. Bauer (2005) suggests that what drives the development of explicit memory may be an age-related decrease in the susceptibility for storage failure, therefore it can be inferred that storage vulnerability may lead to individual differences in explicit memory in infants.

Another approach to investigating individual differences in explicit memory processing would be to examine both behavior and physiology during the available phases of cognitive processing (the encoding and retrieval phases) as well as during a savings assessment to further identify changes in the integrity of memory traces. Electroencephalography (EEG) is psychophysiological tool used to record and measure electrical activity from the scalp which is related to cortical activity (Stern, Ray, & Quigley, 2001). Some advantages to EEG collection are that it is a non-invasive technique and is appropriate for assessing brain electrical activity across the lifespan. Recordings can be collected during both non-active (baseline) periods as well as during cognitive tasks. Like ERP, EEG affords optimal temporal resolution, but because it is continuous, it allows real-time psychophysiological collection during recall processing. Specifically for imitation-based tasks, recording continuous EEG does not require adjustment of the protocol. Studies using ERP to assess the quality of the memory trace with recognition tests just after encoding and/or during the retention interval are forced to rely on intermediate

neurological responses to pictorial representations of the events, and then can indirectly infer where the trace degrades based on this information. A study employing continuous EEG during the imitation task would allow physiological collection during real-time processing.

Based on the anatomical information reviewed above it is clear that both temporal lobe structures, as well as higher level cortical association areas (most likely the PFC) subserve explicit memory function. Although the extent to which one region plays a more prominent role in each stage of information processing is unclear, continuous EEG recording during cognitive processing may provide valuable insights. It may be that the MTL is involved in the initial encoding of the modeled events and sequences and that the PFC is involved in exploiting effective cues during the retrieval phase of the imitation paradigms. Another interesting possibility is that this temporal-cortical circuit (which incorporates both PFC and MTL lobe structures) may develop and function as a unit during each task. Performance-related patterns of baseline-to-task activation during cognitive processing may lend support to this theory.

Recently, Morasch & Bell (*under review*) conducted a between-groups investigation of the neural processes underlying delayed recall with a between-groups deferred imitation task. The behavioral methodology, as well as selection of the retention interval, was guided by the work of Hayne and colleagues (for a review, see Hayne, 2004, 2007). In this study, forty-eight 12-month-olds visited the laboratory on two consecutive days. Half were exposed to a novel, 3-step event sequence and the other half saw a control display. Continuous EEG was collected during event modeling. After a 24 hour delay, infants were allowed to imitate the event, however, during this phase, only behaviors were recorded. Of the 24 infants in the demonstration condition, 14 (58%) produced at least 1 ordered pair of actions and 10 did not. Only one control infant produced an ordered pair of events. Therefore, there was a good deal of

individual variability in recall performance. However, these differences could not be accounted for by the EEG collected during event modeling. This may have occurred for several reasons.

First, there were no overall or performance-related changes in baseline-to-task EEG power during the demonstration. However, within the same session, baseline-to-task activations were observed during two other memory tasks, one assessing spatial working memory, the other assessing recognition memory. It may be that although some infants encoded sufficiently to show delayed recall, the display may not have captured and held their attention enough to evidence changes in brain-electrical patterns. Another explanation may be that the display lasted between 40-50 seconds, and infants became bored with the event; however, looking times did not vary according to performance group (nor did analysis of the first 20 seconds of the EEG for the display). Also, only one event was used in this study, where other investigators use several props and determine recall group based on average memory performance.

It may be that using a between groups design, and essentially eliminating half of the subjects as they do not receive the demonstration exposure, limited statistical power so dramatically that real changes in EEG were not detected. I do not feel that this is likely as, again, baseline to task activations were detected during other memory tasks. It is important to note, that the other tasks within in those sessions involved narration by the experimenter, whereas the between groups imitation procedure did not. A more appropriate choice for investigating individual differences in explicit memory related to continuous EEG may be to employ a within-subjects design and include naturalistic narration (these two considerations are central to the work of Bauer and colleagues; for reviews see Bauer, 2004; Bauer et al., 2007).

Especially with this inclusion of narration in the imitation paradigm, it is important to be able to account for certain non-mnemonic individual differences which may affect recall

performance, such as language development and temperament. The MacArthur Communicative Development Inventory for Infants (MCI-I; Fenson, Dale, Reznick, Bates, Thal, & Pethick, 1994) is a standardized measure of language development that quantifies both infant language comprehension and production of words, phrases, and gestures for children aged 8 to 16 months. Additionally, the Infant Behavior Questionnaire (IBQ-r; Gartstein & Rothbart, 2003) is one of the most widely used measures of temperament for infants between 3 and 12 months of age. It is administered as a parent report of individual differences in 14 dimensions of temperamental factors including emotional, perceptual and motor reactivity, approach and avoidance, and self-regulation. Prior work has assessed the relations between temperament, language development, and memory performance in infancy (Carver & Bauer, 1999; Bauer et al., 2003; Lukowski et al., 2005), indicating that individual differences in these dimensions do not significantly contribute to delayed-recall performance. These two measures have been employed in several longitudinal and cross-sectional examinations in our laboratory, and should be included in an individual differences exploration of infant memory performance.

Finally, it may be that individual differences in explicit memory are simply not related to encoding processes, behaviorally or physiologically. In this case, it would be beneficial to collect physiology during the retrieval phase of the task, thus collecting electrophysiology during both phases of the task in order to identify sources of individual variability in recall. Addition of a savings assessment (and physiological collection during this process) may provide additional insight to understanding this biobehavioral process.

Synopsis of Current Study

This study was designed as a within-subjects test of explicit memory with both behavior and continuous EEG measured during each phase of the imitation tasks. Infants were exposed to

multiple 2-step memory events at 10 months of age, a time of neurocognitive transition, and a 24-hour retention interval was imposed between encoding and retrieval. This interval was selected for several reasons.

First, a 24-hour delay between encoding and retrieval has been commonly used to ensure ample time for representations to be processed into long-term memory storage (Meltzoff, 1988; Barr, et al., 1996; Collie & Hayne, 1999; Hayne, et al., 2000; Hayne, 2004). With this delay, 9 ½ month olds have been shown to show 50% recall of target actions (Meltzoff, 1988) and are able to produce significantly more target actions and ordered pairs of actions than control infants (Wiebe, 2003; Wiebe, Lukowski, & Bauer, 2002). In addition, when using a 1-month delay, both 9- and 10-month olds require multiple (usually 3) exposure sessions (separated by 1-2 days each) in order to accrue enough encoding experiences to show delayed ordered-recall 1-month later. However, this opportunity for expanded encoding using multiple modeling sessions may confound the procedure, in that the repeated encodings of the same display incorporates both new learning, as well as recall for the prior modeling sessions (thus both encoding and retrieval processes, and no longer only encoding). This would be simplified by using a shorter long-term delay (24-hours) in order to require only a single exposure session. Because equivalent variability in delayed recall performance (approximately 50 %) is achieved with the 24-hour delay (using one exposure session) and a 1-month delay (using 3 exposure sessions), the 24-hour interval presents a parsimonious choice for the delayed procedure.

The current study was designed to systematically investigate brain/behavior relations during the formation and retrieval of event memories. The additional assessment of savings from relearning was included to further pinpoint whether long-term memory failure was due to problems in retrieval or degradation of the memory trace during storage. Patterns of brain-

electrical activity at rest and during cognitive processing provided insight to the contributions of specific brain areas and neural circuits to explicit memory function. The targeted age group for this study, 10-month-old infants, was purposefully selected to capitalize on a period of behavioral and neurophysiological transition to ensure a wide range of individual variability in memory performance. In addition, recent reports indicate that the encoding abilities of 10-month-olds are more robust than at 9 months (Bauer et al., 2006) and memories created at 10 months of age are less susceptible to degradation than are memory traces created at 9 months. Also, for a majority of the work indexing evoked responses and explicit memory, the age at recall testing is 10 months (Bauer et al., 2003; Bauer et al., 2006; Carver et al., 2000; Lukowski et al., 2005). Finally, this research design affords a within-subjects comparison of immediate recall and delayed recall, which, when coupled with real-time electrophysiological recordings, will add to our understanding of infant short- and long-term memory processing.

Hypotheses

For ease of reading the hypotheses, the following is reiteration of several key terms. Recall performance in all of the following hypotheses was defined as a proportional score of correctly-ordered pairs of target actions. All delayed recall assessments occurred after the 24-hour retention interval. Immediate recall was assessed seconds after modeling. A relearning (second modeling) session was embedded into the delayed recall procedure to assess savings through relearning (residual memory traces despite poor performance). Continuous EEG was recorded throughout both the delayed recall procedures (encoding on day 1, retrieval on day 2) as well as immediate recall procedures (both encoding and retrieval on day 2).

Behavioral Hypotheses

1. Infants would produce a greater number of ordered pairs of actions during the post-modeling tests than during the pre-modeling baseline periods. In addition, consistent with previous research (Bauer et al., 2001; Bauer et al., 2003; Bauer et al., 2006; Carver & Bauer, 1999; Lukowski et al., 2005), approximately 50% of infants would show delayed ordered recall (one or more correctly order pairs of target actions) after 24-hours.
2. Infants would show better memory performance on the test of immediate recall rather than delayed recall.
3. Infants with difficulty in delayed recall performance would benefit from additional modeling, thus demonstrating savings in relearning.
4. Consistent with previous work with infant recall, non-mnemonic individual differences would not predict memory performance. Temperament characteristics (IBQ-r) and language development (MCDI-I) would be unrelated to recall performance.

Electrophysiological Hypothesis

5. Infants would show baseline-to-task 6-9Hz EEG power activation at frontal and/or temporal locations during each phase of the imitation assessments.

Biobehavioral Hypotheses

6. Infants who showed ordered recall (one or more pairs of correctly-ordered actions) would have different patterns of baseline-to-task EEG activation than those who did not.
    6a. Specifically, for delayed recall, infants who evidenced ordered-recall would have had greater temporal (T3/T4) activation during encoding and/or greater frontal (F3/F4) activation during retrieval.

6b. For both the immediate and delayed assessments, infants who did not evidence ordered recall would show no significant baseline-to-task activation during either the encoding or retrieval phases.

7. Within subjects, correct and incorrect performance (determined by the presence or absence of ordered recall) for each assessment would be related to different patterns of frontal (F3/F4) and or temporal (T3/T4) activity during encoding and retrieval.
8. Frontal and temporal changes in EEG power during recall tasks would account for a significant portion of variance in ordered-recall performance.

Method

Participants

Forty-eight 10-month-old infants (+/- 2 weeks) from the New River Valley section of Southwest Virginia were recruited for participation in this study from the Developmental Sciences Database at Virginia Tech. The sample consisted of 16 females and 32 males. Ethnic and racial distribution in the sample was as follows: 44 infants were reported to as non-Hispanic (92%) and the remaining 4 infants were reported as Hispanic (8%). Forty-three infants (89%) were reported as Caucasian, 2 (4%) were reported as American Indian/Alaska Native, 1 (2%) was reported as Asian, and for two (4%) of the infants no racial information was provided (both of these families reported ethnicity as Hispanic). Average maternal age was 29.6 years (SD= 4.8, range= 23-42) and average paternal age was 31.9 years (SD= 5.85, range= 24-48). All parents completed at least a high school degree, with the distribution of highest level of education completed as follows: for 7 parents (7%) high school was the highest education level completed, for 7 (7%) it was technical school, for 46 (49%) it was college, and 34 parents (36%) completed a graduate degree. One family did not provide educational information.

Initial contact with parents was via an information letter (see Appendix A), followed by phone calls. Infants were recruited if they were born within 2 weeks of their expected due dates and experienced no prenatal or birth complications. Recruited infants weighed at least 2,500 grams at birth, required no oxygen at birth, and had no neurological diagnoses.

Materials

Behavioral Stimuli. Stimuli for the imitation tasks were six novel two-step event sequences, with individual actions connected by enabling relations to lead to an interesting end state (see descriptions in Appendix B). All events were adapted from published stimuli used by Bauer and colleagues (Bauer & Mandler, 1992; Bauer, et al., 2001; Carver & Bauer, 1999, 2001; Carver et al., 2000). For example, in the event sequence "make a gong", the event consisted of a U-shaped wooden base with a thin dowel connecting 2 side panels, a small brass bell attached to an S-hook, and wooden stick with a wooden ball attached to one end. The experimenter modeled (a) hanging the bell from the bar (Step 1) and hitting it with the stick (Step 2) making a ringing sound.

Questionnaires. Prior to arrival at the laboratory, parents completed three questionnaires about their infant's health and behavior. A general information questionnaire (Appendix C), developed by our lab, was used to obtain information regarding the age and general health of the infant, ethnic and racial designations of the parents, parents' level of highest education, and parents' handedness measures. In addition, non-mnemonic individual differences in temperament characteristics and language development were collected upon enrollment with the IBQ-r (Garstein & Rothbart, 2003) and the MCDI-I (Fenson et al., 1994).

Procedures

Upon entrance to the laboratory, parental permission was obtained (see Appendix E). Parents and infants visited the laboratory on two consecutive days (visits were scheduled 24 hours, +/- 2 hours apart). During each visit, behavioral and electrophysiological measures were collected by the same experimenter in the same room.

EEG Recordings. Continuous electroencephalogram (EEG) was collected throughout both sessions of this study to assess brain electrical activity from the scalp during cognitive processing. EEG recordings were made from 8 left and 8 right scalp sites: frontal pole (Fp1, Fp2), medial frontal (F3, F4), lateral frontal (F7, F8), anterior temporal (T3, T4), posterior temporal (T7, T8), central (C3, C4), parietal (P3, P4), and occipital (O1, O2) (Pivik, Broughton, Coppola, Davidson, Fox, & Nuwer, 1993). All electrode sites were referenced to Cz. EEG was recorded using a 21-lead stretch cap (extra-small sized Electro-Cap) with electrodes placed in the International 10/20 system pattern (Jasper, 1958). After the cap was placed on the infant's head, its ear straps were snapped into place on an infant-sized elastic chest belt with Velcro closure on the infant's back. EEG gels were dispensed into each recording site from a 5-ml plastic syringe equipped with a blunt tip. A small amount of abrasive (NuPrep) gel was placed into each recording site and the scalp site was gently rubbed with the smooth wooden end of a cotton swab. Following this, conductive gel provided by the cap manufacturer was placed in each site. Electrode impedances were measured and accepted if they were below 5K ohms. The electrical activity from each lead was amplified using separate SA Instrumentation Bioamps and bandpassed from 1 to 100 Hz. Activity for each lead was displayed on the flat-screen monitor of a 100-MHz acquisition computer located in the testing room. The EEG signal was digitized online at 512 samples per second for each channel so that the data were affected by aliasing.

Continuous 6-9 Hz EEG recordings occurred during a baseline period in each visit and during the cognitive tasks. This frequency band was selected because infants 8 months of age have a dominant frequency between 6 and 9 Hz (Bell & Fox, 1994), and this particular frequency band discriminates baseline EEG from task EEG (Bell, 2001), as well as correct from incorrect responses (Bell, 2002), during an infant working memory task.

During the electrode application, a research assistant entertained and distracted the infant by playing with age-appropriate toys. This entertainment period also served to help the infant to "warm-up" to the laboratory setting. Immediately prior to the first cognitive task, EEG electrodes were applied and 1-minute of baseline physiology was recorded as the infant sat on the mother's lap. During baseline recordings, the experimenter alternated manipulating a toy containing brightly colored balls and tapping her nails on the top of the testing table (measuring 90 cm (L) x 60 cm (W) x 75 cm (H) and located 1.1 meters in front of the infant[1]). This procedure was used to quiet the infant during baseline recording (e.g., Bell, 2001, 2002); in addition, it yielded minimal eye movement and gross motor movements. Parents were instructed not to talk to infants during the baseline recording. Immediately after the baseline period, the first cognitive task was administered. Event marks were placed on the physiological record so that the EEG recordings could be synchronized with the stimulus presentations of the cognitive tasks. After the final task of each visit, the EEG cap was gently removed and the gels were washed from the infant's hair.

Visit 1. During the first session of the study, infants were presented with 3 novel 2-step event sequences (for schematic representation of the imitation protocol, see Table 3). For each event sequence in turn, infants observed a set of props placed upon the far surface of the testing table for 10 seconds. The props were then moved so that they were within the infant's reach and

the experimenter provided a general verbal label for the event. Infants were allowed to manually explore the props so that each infant could establish his/her own baseline for the spontaneous production of target actions for each sequence. This baseline period lasted 60 seconds or until the infant became bored or fussy. The experimenter then took back the props and on the surface of the testing table, modeled the event sequence twice, verbally labeling the event and narrating each step. Infants were not permitted to touch the props again or imitate these actions. These toys were then removed from the testing table and the process was repeated with the props for the remaining two event sequences.

The presentation of sequences was block randomized such that each infant saw 1 of 2 available sets of 3 event sequences during the first visit (sets ABC and DEF were counterbalanced across subjects). The block of event sequences used during the first day's exposure session (ex. ABC) were the stimuli to be used in the delayed recall at visit 2 (after the 24 hour delay). The other block of event sequences (ex. DEF; to be modeled on day 2) served as the stimuli for the assessment of immediate recall during visit 2.

Visit 2. Infants and parents returned to the laboratory 24 hours (+/- 2 hours) for the second day of the study. Infants were tested by the same experimenter and in the same room as in the exposure session. Continuous EEG was again collected during a baseline period and all cognitive tasks. During visit 2, infants were tested for both delayed and immediate recall of event sequences. The order of these two tasks was be counter-balanced to control for whether previous manipulation of the first set of props resulted in a heightened generalized exploration of the second set of props which may have impacted performance.

Delayed Recall Assessment. For each of the three event sequences modeled during visit 1, infants again observed the set of props being placed upon the far surface of the testing table

for 10 seconds. The props were then moved within infant's reach and the experimenter provided a general verbal label for the event. During this delayed recall test, infants were allowed to manipulate the props for approximately 60 seconds. Behavioral measures of interest for each event included: the number of target actions produced (max = 2), the number of ordered pairs of actions produced (max=1), and the latency for the infant to touch the props (ranging from 0 to 60 seconds).

If, during this delayed recall test, the infant did not demonstrate ordered recall (both actions imitated in the correct sequence), an assessment of savings from relearning was conducted. Once again, experimenter took back the props demonstrated the event sequence once more, verbally labeling the event and narrating each step. The toys were given back to the infant and the general verbal label was provided for the event. Infants were once again allowed to manipulate the props for approximately 60 seconds. This procedure was conducted for the remaining 2 sets of toys only if infants did not display ordered recall during the delayed recall tests of those events. As before, behavioral measures of interest for savings assessment included: the number of target actions produced (max = 2), the number of ordered pairs of actions produced (max=1), and the latency for the infant to touch the props (ranging from 0 to 60 seconds).

Immediate Recall Assessment. Because the immediate recall assessment required a zero delay between initial modeling of the events and recall testing, both the modeling and testing for this assessment were conducted during visit 2. As was the case for the modeling portion of the delayed recall procedures, infants were presented with 3 novel 2-step event sequences (ex. Toys D, E, & F). For each event sequence in turn, infants observed a set of props placed upon the far surface of the testing table for 10 seconds. The props were then moved so that they were within

the infant's reach and the experimenter provided a general verbal label for the event. Infants were allowed to manually explore the props so that each infant could establish his/her own baseline for the spontaneous production of target actions for each sequence. This baseline period lasted 60 seconds or until the infant became bored or fussy. The experimenter then took back the props and on the surface of the testing table, modeled the event sequence twice, verbally labeling the event and narrating each step. In contrast to the delayed recall procedures, however, these props were then immediately returned to the infant, and he/she was allowed to imitate the action sequences that were recently modeled. During this immediate recall test, infants were allowed to manipulate the props for approximately 60 seconds. Behavioral measures of interest for each event included: the number of target actions produced (max = 2), the number of ordered pairs of actions produced (max=1), and the latency for the infant to touch the props (ranging from 0 to 60 seconds).

Data Reduction

Electrophysiological Data. EEG data were examined and analyzed using software developed by James Long Company. First, the data were re-referenced offline via software to an average reference configuration. This average referencing weighed all the electrode sites equally and eliminated the need for a noncephalic reference. Active (F3, T3, etc.) to reference (Cz) electrode distances vary across the scalp and without this re-referencing it would be unclear if power values reflect differences in inter-electrode distance or valid brain-electrical activity. The data were then artifact-scored for eye movements using a peak-to-peak criterion of 100 uV or greater. Artifact associated with gross motor movements over 200 uV peak-to-peak was also scored. These artifact-scored epochs were eliminated from all subsequent analyses. The data were then analyzed with a discrete Fourier transform (DFT) using a Hanning window of 1-sec

width and 50% overlap. Power values were computed at each scalp site during each task within the 6- to 9-Hz frequency band. Power was expressed as mean square microvolts and the data were transformed using the natural log (ln) to normalize the distribution.

Behavioral Data. Each session was videotaped for later coding. The author recorded 100% of infants' behaviors, noting both the production of target actions and the order in which they were completed. For each event-sequence, infants could produce two target actions in one correct order. For example, in the event "make a gong", the infant must have hung the bell (step one) and then hit it with the stick (step 2) to ring it (end state). Only the first occurrence of each target action was coded so as to reduce credit that might be received due to chance or trial and error. This provided a more conservative measure of memory performance. All subsequent analyses define memory performance as the proportion of correctly ordered pairs of actions per block (either delayed or immediate).

Results

In this section, I restate and test my a priori hypotheses regarding behavior expressions of immediate and delayed recall, electrophysiological patterns during each phase of the recall assessments, and performance-related differences in the biobehavioral expression of infant recall.

Behavioral Results

All infants contributed complete data for immediate and delayed recall assessments. However, for one infant, these data were inaccessible due to experimenter error with the video record. Therefore, all behavioral results involve the remaining 47 infants.

Hypothesis 1. In the first portion of this hypothesis, I expected infants to produce a greater number of ordered pairs of actions during the post-modeling tests than during the pre-modeling baseline periods. In order to examine whether the modeling procedure was successful

at promoting recall memory at both the zero delay (immediate) and the 24-hour delay (delayed), I conducted paired samples t-tests[2] to compare action sequence production before and after the modeling displays (see Figure 1). Within the immediate recall task, the proportion of correctly-ordered pairs of actions observed before the modeling was significantly different than that observed during the post-modeling test (t (46) = 2.24, p=.03; η^2= .10). Indeed, relative to the pre-modeling baseline (M= .06, SD= .13), infants produced more ordered pairs after observing the modeled display (M= .14, SD= .25). Similarly, within the delayed recall task, modeling of the display significantly increased (t (47) = 3.90, p<.01; η^2= .25) the number of action sequences produced (M=.17, SD= .24) relative to the pre-modeling baseline (M= .04, SD= .11). Therefore, the modeling procedures were successful in creating event memories that were recalled by infants both immediately and after a 24-hour delay.

In the second part of hypothesis 1, I stated that I expected that approximately 50% of infants would show delayed ordered recall (one or more correctly order pairs of target actions) after 24-hours. Descriptive analyses were conducted to examine the frequency of infants displaying ordered recall during the immediate and delayed assessments. Within this sample, the percentage of infants producing at least one complete action sequence during the imitation test was as follows. During the immediate test, 14 infants (30%) produced at least one correctly ordered pair. In addition, for the delayed test, 19 infants (40%) produced at least one correctly ordered pair. Therefore, the proportion of infants displaying ordered recall after the 24-hour delay was lower than hypothesized.

For subsequent analyses comparing memory performance with physiology and/or other individual difference measures, 2 performance groups were created and included a zero-recall group and an ordered-recall group. Group membership was dependent on the presence of at least

1 correctly-ordered pair of actions during the recall assessments (Bauer et al., 2003, Carver et al., 2000, Lukowski et al., 2005). Within analyses, the low value (0) was assigned to the group of infants who did not show ordered recall on that task, relative to the group of infants who produced at least 1 ordered pair of events (this group received a score of 1 for successful performance).

Hypothesis 2. This hypothesis stated that infants would show better memory performance on the test of immediate recall rather than delayed recall. To test this, I conducted a paired-samples t-test between the proportional scores of ordered-pairs for each assessment. There was no difference between immediate and delayed recall test performance (t (46) = -.51, p=.61). Infants recalled ordered-pairs of actions equally well regardless of whether recall was assessed immediately after encoding or if it was delayed 24 hours.

Hypothesis 3. In this hypothesis, I predicted that infants who expressed difficulty in delayed recall performance (did not correctly produce and order actions) would benefit from additional modeling, and would thus demonstrate savings in relearning. However, in the cases where savings through relearning was assessed, there was no significant increase in production of sequences related to additional demonstration of the display when compared to initial delayed recall performance (t (44) =1.6, p=.12).

Hypothesis 4. This hypothesis stated that non-mnemonic individual differences would not predict memory performance, specifically, that infant temperament characteristics (IBQ-r) and language development measures (MCDI-I) would be unrelated to recall performance. To examine whether non-mnemonic individual differences contributed to recall performance group membership, measures of temperament and language development were regressed on task performance. Across both the immediate and delayed assessments, only one infant temperament

scale was significantly associated with recall performance grouping. Correlation coefficients are provided in Table 4. Lower maternal ratings of low-intensity pleasure were related to higher delayed recall performance group, r (47) = -.29, p=.04. Several other temperament scales were marginally significant in relation to immediate memory performance group including negative correlations with fear (r (46) = -.27, p=.07) perceptual sensitivity (r (46) = -.28, p=.06) and vocal reactivity (r (46) = -.26, p=.08). However, subsequent regression equations indicated that infant temperament was not predictive of either immediate (R^2= .20, χ^2 (13) = 15.80, p= .26) or delayed group status (R^2= .33, χ^2 (13) = 19.02, p= .16).

In addition, there were no significant bivariate correlations between infant language (as measured by the MCDI) and immediate recall group. Using binary logistic regression, regressing comprehensive language on immediate recall group also was not significant (R^2= .20, χ^2 (10) = 9.32, p= .50). Two language measures were correlated with delayed memory performance, vocabulary comprehension (r (46) = -.39, p=.01) and gestural imitation (r (46) = -.32, p=.03). However again, using binary logistic regression, regressing language on delayed recall group also was not significant (R^2= .31, χ^2 (10) = 15.62, p= .11).

As an additional note, latency to touch the toys for each assessment was recorded every time a set of toys was given to the infant for either a baseline assessment or recall test. For every infant in the study, regardless of performance, the latency to touch the toys was under 2 seconds. Because this measure included virtually no variability, it was not included in behavioral analyses.

Electrophysiological Results

Forty-seven infants (98%) contributed electrophysiological data for immediate and delayed recall assessments. One infant refused to wear the EEG electrode cap. Of these 47

infants, one infant's day1 data and another's day 2 data were inaccessible due to experimenter error with the physiological record. Therefore, the sample size for the following electrophysiological analyses was 45 infants.

Hypothesis 5. I expected that infants would show baseline-to-task 6-9Hz EEG power activation at frontal and/or temporal locations during each phase of the imitation assessments. In order to examine patterns of brain-electrical activity during the two separate phases of the 3 assessments (demonstration and test) un-related to performance, I conducted separate 2 (Condition: baseline vs. task) x 8 (Region: Fp1/2, F3/4, F7/8, T3/4, T7/8, C3/4, P3/4, O1/2) x 2 (Hemisphere: left vs. right) repeated measures MANOVA's for baseline-to-task EEG power changes during each phase of the immediate recall, delayed recall, and savings assessment procedures. For each phase of these three assessments, there were significant main effects for region where posterior scalp sites had higher EEG power values than anterior sites (*F*'s ranged from 97.45 to 159.99, all *p's*<.001; η^2 ranged from .95- .97). This pattern of increasing regional power values from front to back on the scalp is considered to be standard in psychophysiological testing and has been previously reported in infant studies (Bell, 2002). All subsequent results focus on Condition x Region interactions in each phase of the recall assessments. These findings are summarized in Table 5.

For the immediate recall assessment, there was a significant Condition x Region interaction during the demonstration (encoding) phase, (Hotelling's Trace: F (7, 39) = 2.92, p=.02; η^2=.34). Regional analyses indicate that these effects were present at sites T7/8 (F (1, 44) = 4.49, p=.04; η^2=.09) and P3/4 (F (1, 44) =4.26, p=.04; η^2=.09). Examination of the means indicated that during the demonstration condition of the immediate recall procedures power

values at temporal (T7/8) and parietal (P3/4) scalp locations (M=3.11, SEM=.06 and M=2.86, SEM=.06) decreased relative to baseline (M=3.20, SEM=.07 and M=2.97, SEM=.08).

In addition, there was a significant Condition x Region interaction during the testing (recall) phase of the immediate recall assessment, (Hotelling's Trace: F (7, 39) = 21.54, p<.001; η^2=.79). Regional analyses indicate that these effects were present at sites F3/F4 (F(1, 44)= 16.15, p<.01; η^2=.26), T7/T8 (F(1, 44)= 3.93, p=.05; η^2=.08), C3/C4 (F(1, 44)= 14.21, p<.01; η^2=.24), P3/P4 (F(1, 44)= 17.60, p<.01; η^2=.28), and O1/O2 (F(1, 44)= 4.25, p=.04; η^2=.09). Examination of the means indicated that during the test phase of the immediate recall procedures power values at frontal (F3/F4), temporal (T7/T8), central (C3/C4), and parietal (P3/P4) scalp locations decreased relative to baseline. Uncharacteristic of this pattern, power values at occipital sites (O1/O2) increased from baseline to task.

For the delayed recall assessment, there was a significant Condition x Region interaction during the demonstration (encoding) phase, (Hotelling's Trace: F (7, 38) = 6.84, p<.001; η^2=.56). Regional analyses indicate that these effects were present at sites C3/C4 (F (1, 44) = 13.86, p<.001; η^2=.24) and P3/4 (F (1, 44) =6.96, p=.01; η^2=.14). Examination of the means indicated that during the demonstration condition of the delayed recall procedures power values at central (C3/C4) and parietal (P3/4) scalp locations decreased relative to baseline.

In addition, there was a significant Condition x Region interaction during the testing (recall) phase of the delayed recall assessment, (Hotelling's Trace: F (7, 38) = 14.74, p<.001; η^2=.73). Regional analyses indicate that these effects were present at sites F3/F4 (F(1, 44)= 8.63, p=.01; η^2=.16), T7/T8 (F(1, 44)= 4.15, p=.05; η^2=.09), C3/C4 (F(1, 44)= 20.08, p<.001; η^2=.31), and P3/P4 (F(1, 44)= 11.63, p<.001; η^2=.21). Examination of the means indicated that during

the test phase of the immediate recall procedures power values at frontal (F3/F4), temporal (T7/T8), central (C3/C4), and parietal (P3/P4) scalp locations decreased relative to baseline.

Finally, for the savings assessment, there was a significant main effect for region where more posterior scalp sites had higher EEG power values during the reminder (re-encoding) phase (F (7, 36) = 159.99, p<.001; η^2=.97), as well as during the re-test phase (F(7, 36) =144.17, p<.001; η^2=.97). In addition, there was a significant Condition x Region interaction during the reminder (re-encoding) phase, (Hotelling's Trace: F (7, 36) = 4.24, p<.01; η^2=.45). Regional analyses indicate that these effects were present at sites F3/F4 (F(1, 42)= 6.56, p=.01; η^2=.14), T7/T8 (F(1, 42)= 4.61, p=.04; η^2=.10), C3/C4 (F(1, 42)= 9.04, p<.01; η^2=.18), and P3/P4 (F(1, 42)= 5.30, p=.03; η^2=.11). Examination of the means indicated that during the reminder condition of the savings assessment power values at frontal (F3/F4), temporal (T7/T8), central (C3/C4) and parietal (P3/4) scalp locations decreased relative to baseline.

In addition, there was a significant Condition x Region interaction during the re-testing phase of the savings assessment, (Hotelling's Trace: F (7, 36) = 9.23, p<.001; η^2=.64). Regional analyses indicate that these effects were present at sites F3/F4 (F (1, 42) = 8.63, p=.01; η^2=.17), C3/C4 (F (1, 42) = 15.98, p<.001; η^2=.28), and P3/P4 (F (1, 42) = 7.83, p=.01; η^2=.16). Examination of the means indicated that during the test phase of the immediate recall procedures power values at frontal (F3/F4), central (C3/C4), and parietal (P3/P4) scalp locations decreased relative to baseline. In summary, across each phase of each recall assessment, there was a dominant pattern of baseline-to-task decreases in EEG power during cognitive processing.

Biobehavioral Results

Hypothesis 6. This hypothesis had 3 components. First, I expected that infants who showed ordered-recall (one or more pairs of correctly-ordered actions) would have different

patterns of baseline-to-task EEG activation than those who did not. In order to examine differences in patterns of brain-electrical activity based on recall performance, I conducted separate 2 (Group: no ordered pairs vs. at least 1 ordered pair) x 2 (Condition: baseline vs. task) x 2 (Hemisphere: left vs. right) mixed groups repeated measures MANOVA's the encoding and test phases of each of the three assessments (immediate, delayed, and savings). Regional analyses focused exclusively on frontal and temporal sites, as memory effects were hypothesized at sites F3/F4 and T3/T4.

Second, based on previous work in our lab (Bell, 2001, 2002; Wolfe & Bell, 2004, Morasch & Bell, *under review*) I expected that better cognitive performance would be supported by increases in 6-9 Hz EEG power from baseline-to-task. Therefore, I specifically hypothesized that, at least in the case of the delayed assessment, infants who evidenced ordered-recall would have greater temporal (T3/T4) activation during encoding and/or greater frontal (F3/F4) activation during retrieval. Finally, for both the immediate and delayed assessments, I expected that infants who did not evidence ordered-recall would display no significant baseline-to-task changes in EEG power during either phase of the recall assessments. Subsequent post-hoc analyses expanded these regional investigations to include additional frontal and temporal sites.

Immediate Assessment. During the demonstration (encoding) phase of the immediate recall assessment, repeated measures MANOVA revealed marginally significant effects of recall group on baseline-to-task changes in EEG power at sites F3/F4 (see Figure 2). A Group x Condition interaction approached significance (Hotelling's Trace: F (1, 43) = 3.34, p=.07) at region F3/F4. A condition effect was present within the zero-recall group (F (1, 31) = 12.38, p<.001; η^2=.29) where these infants displayed lower power values during the demonstration phase (M=2.60, SEM=.09) relative to baseline (M=2.79, SEM=.10). In contrast, the infants who

succeeded in producing at least one correctly-ordered sequence, the ordered-recall group, did not have any baseline-to-task changes in EEG power values during the demonstration phase at region F3/F4 ($F(1, 12)=.19$, $p=.67$).

Post-hoc analysis expanded regional investigations to include other frontal and temporal sites. At region F7/F8, there was a significant Group x Condition x Hemisphere interaction (Hotelling's Trace: $F(1, 43) = 4.66$, $p=.04$; $\eta^2=.10$). Simple effects analyses revealed that infants in the zero-recall group evidenced significant differences in EEG power from baseline to task at site F8 ($t(31)= 1.98$, $p=.05$; $\eta^2=.11$) but not site F7 ($t(31)= .72$, $p=.48$). At scalp location F8, infants in the zero-recall group displayed lower power values during the demonstration phase (M=2.53, SD=.43) relative to baseline (M=2.62, SD= .52). In contrast, infants in the ordered-recall group did not display any baseline to task changes in EEG power values during the demonstration phase at site F7 ($t(12) =1.25$, $p=.24$) or F8 ($t(12) =1.08$, $p=.30$).

During the testing phase of the immediate assessment, a significant Group x Condition x Hemisphere interaction was revealed (Hotelling's Trace: $F(1, 43) = 10.79$, $p=.002$; $\eta^2=.20$) at region F3/F4 (see Figure 3). Simple effects analyses revealed that infants in the zero-recall group evidenced significant differences in EEG power from baseline to task at sites F3 ($t(31)= 2.76$, $p=.01$; $\eta^2=.20$) and at site F4 ($t(31)= 3.29$, $p=.003$; $\eta^2=.26$). At both locations, power values during the immediate recall test (F3: M=2.62, SD=.61; F4: M=2.55 , SD=.44) decreased relative to baseline (F3: M=2.81, SD=.69; F4: M=2.76, SD=.60). Infants in the ordered-recall group also showed differences at right frontal site F4 ($t(12) = 3.15$, $p=.008$; $\eta^2=.45$) where power values during the immediate recall test (M=2.3, SD= 1.09) decreased relative to baseline (M=2.72, SD=.74). However, infants who were in the ordered-recall group did not show any

baseline-to-task related changes during the immediate recall test at left frontal site F3 ($t(31)= .30$, $p=.77$).

To summarize, for the immediate recall assessment, group-related differences were observed at frontal regions where better performance, almost exclusively, was indicated by no baseline-to-task changes, but weaker performance was marked by baseline-to-task decreases in EEG power.

Delayed Assessment. During the demonstration (encoding) phase of the delayed recall assessment, repeated measures MANOVA revealed no significant effects of recall group on baseline-to-task changes in EEG power at hypothesized frontal regions F3/F4 or temporal regions T3/T4. Additional post-hoc examinations expanding these analyses to include other frontal and temporal sites were also non-significant during the demonstration phase of the delayed assessment.

In addition, during the delayed recall test, no group related effects were observed at hypothesized sites F3/F4 or T3/T4. However, post-hoc examinations expanding to other frontal and temporal sites revealed a significant Group x Condition interaction during the recall test at region F7/F8 (Hotelling's Trace: F (1, 44) = 5.71, $p=.02$; $\eta^2=.12$). These findings are presented in Figure 4. Infants in the zero-recall group evidenced no significant differences in EEG power from baseline to test at region F7/F8 (F (1, 26) = 2.11, $p=.16$). However, infants in the ordered-recall group showed differences in EEG power from baseline to test at region F7/F8 (F (1, 18) = 4.38, $p=.05$; $\eta^2=.20$) where power values during the delayed recall test (M=2.65, SEM=.09) increased relative to baseline (M=2.54, SEM=.11).

A marginally significant Group x Condition x Hemisphere interaction at region T7/T8 (Hotelling's Trace: F (1, 44) = 3.34, $p=.07$). Simple effects analyses revealed that infants in the

zero-recall group evidenced no significant differences in EEG power from baseline to test at site T7 ($t(26)= 1.08$, $p=.29$) and a significant difference at site T8 ($t(26)= 3.31$, $p<.001$; $\eta^2=.30$). At right temporal site T8, power values during the delayed recall test (M=3.11, SD=.38) decreased relative to baseline (M=3.30, SD=.38). In contrast, infants in the ordered-recall group showed no task-related differences at left temporal site T7 (t (18) = .35, $p=.73$) or T8 (t (18) = .09, $p=.93$).

In summary, for the delayed recall assessment, ordered-recall was noted by 2 patterns: baseline-to-task increases at bilateral frontal sites and by no baseline-to-task changes in temporal activity. However, poor recall performance was marked by baseline-to-task decreases in EEG power at right temporal cortex, and by no change in frontal activity.

Savings Assessment. During the reminder (re-encoding) phase of the savings assessment, repeated-measures MANOVA revealed marginally significant effect of performance group on baseline-to-task changes in EEG power at region T3/T4 (see Figure 5). A Group x Condition x Hemi interaction approached significance (Hotelling's Trace: F (1, 41) = 3.49, $p=.07$). Simple effects analysis indicated that infants in the zero-recall group evidenced no significant differences from baseline to task at sites T3 ($t(27)=.60$, $p=.56$) and T4 ($t(27)=.23$, $p=.82$). Infants in the ordered-recall group also did not show any baseline-to-task changes in EEG power values during the reminder phase at site T3 ($t(14)=.37$, $p=.72$) but did show a marginal difference at site T4 ($t(14)= 1.80$, $p=.09$). At this location, infants in the ordered-recall group showed marginally decreased power values (M=2.89, SD=.37) relative to baseline (M=3.04 SD=.35).

Finally, during the re-test phase of the savings assessment, a significant Group x Condition interaction was found at region T3/T4 (Hotelling's Trace: F (1, 41) = 4.56, $p=.04$; $\eta^2=.10$). However, simple effects analyses did not reveal significant condition effects for either group. Infants in the zero-recall group evidenced no significant differences from baseline to task

at region T3/T4 (F (1, 27) =2.80, p=.11). Infants in the ordered-recall group also did not show any baseline-to-task changes in EEG power values during the reminder phase at region T3/T4 (F (1, 14) =1.71, p=.21).

To summarize, for the savings assessment, group-related differences were marginal and observed only at right temporal site T4, where better performance was indicated by a marginal decrease in baseline-to-task EEG power, and zero-recallers displayed no baseline-to-task changes.

Hypothesis 7. I expected that, within subjects, correct and incorrect performance (determined by the presence or absence of ordered recall) for each assessment would be related to different patterns of frontal (F3/F4) and or temporal (T3/T4) activity during encoding and retrieval. To examine different patterns of brain-electrical activity underling within-subjects performance at the response level, I conducted 2 (Performance: correct vs. incorrect) x 2 (Hemisphere: left vs. right) repeated measures MANOVAs for both the demonstration and testing phases of the immediate and delayed assessments at regions F3/F4 and T3/T4. Analyses were only conducted if the same infant displayed at least one correct and one incorrect trial within the assessment block.

Ten infants (21%) met this criterion for the immediate assessment. Because of the reduced sample size, baseline-to-task power values were transformed into change scores (task-baseline) to maximize degrees of freedom. In this situation, negative change scores indicate decreases in EEG power values from baseline to task, and positive change scores indicate increased power values from baseline to task. There were no significant main effects or interactions associated with performance (within-subjects) on any of the analyses involving the immediate recall assessment (F's(1,9) ranged from .01 to 2.66, p's ranged from .16 to .91).

Data from 18 infants (38%) met the criteria for the delayed recall assessment. Again 2 (Performance: correct vs. incorrect) x 2 (Hemisphere: left vs. right) repeated measures MANOVAs were conducted for both the demonstration and testing phases at regions F3/F4 and T3/T4 using EEG change scores (see Figure 6). During the demonstration phase of the delayed recall assessment, there was a significant main effect of response performance at region F3/F4 (Hotelling's Trace: $F(1, 17)= 4.57$, $p=.05$; $\eta^2=.21$). Demonstration trials that would go on to be forgotten after 24 hours were evidenced by larger negative change scores (task-related decreases; M= -.30, SD= .13) relative to trials that would be correctly recalled (imitated in the modeled order after the 24-hour delay; M= -.17, SD= .11). No response performance effects were observed at region T3/T4 during the demonstration phase ($F(1, 17)=.06$, $p=.80$).

Additionally, no response performance effects were observed during the test phase of the delayed recall assessment at region F3/F4 ($F(1, 17)= .002$, $p=.97$), however, there was a marginally significant main effect of response performance at region T7/T8 during the delayed testing phase (Hotelling's Trace: $F(1, 17)= 4.13$, $p=.06$). Demonstration trials that would go on to be correctly recalled after 24 hours were evidenced by larger positive change scores (task-related increases; M= .26, SD= .12) relative to trials that would be forgotten (M= .07, SD= .07).

In summary, correct recall trials were signified by 2 EEG patterns: reduced decreases (less negative changes) in power values from baseline to task at medial frontal region F3/F4 during *encoding* (relative to incorrect responses from the same infant) and greater increases in power values at anterior temporal region T3/T4 during the retrieval *test* (relative to incorrect responses from the same infant).

Hypothesis 8. In an effort to examine individual differences in the relation between ordered-recall and brain-electrical activity (an analysis unprecedented in the infant recall

literature), I hypothesized that frontal and temporal changes in EEG power during recall tasks would account for a significant portion of variance in ordered-recall performance. The purpose of this analysis was determine whether infants who correctly ordered multiple sequences had larger changes in EEG power from baseline to task.

Linear regression analyses were conducted to examine the proportion of variance in recall performance accounted for by task-related changes in EEG power. To this end, frontal (F3/F4) and temporal (T3/T4) change scores from each phase of the assessments were regressed on the proportion of correctly-ordered event sequences for the immediate and delayed assessments. This is in contrast to previous analyses in which performance was grouped as either zero-recall (no correctly-ordered pairs) or ordered-recall (at least one, but up to 3 correctly ordered pairs).

Within the ordered-recall group for the immediate assessment, there were no relations between the number of ordered-pairs displayed and changes in EEG power during either the demonstration phase (R^2=.29, F(4, 12)= .82, p=.55) or the test phase (R^2=.19, F(4, 12)= .49, p=.75). Similarly, within the ordered-recall group for the delayed assessment, there were no relations between the number of ordered-pairs displayed and changes in EEG power during either the demonstration phase (R^2=.33, F(4, 16)= 1.5, p=.28) or the test phase (R^2=.18, F(4, 18)= .78, p=.56). Post-hoc analyses including other frontal and temporal regions were also not significant. Therefore, in terms of biobehavioral patterns underlying infant recall, it was the presence of the ability to order actions (see results for hypothesis 6) rather than individual variability in the magnitude of this skill that was related to changes in brain-electrical activity.

Discussion

This study represents the first investigation to concurrently examine continuous brain-electrical activity throughout the encoding and retrieval stages involved in infant explicit

memory. The first portion of this discussion will focus on my a priori hypotheses regarding infant recall behavior, electrophysiological indices of encoding and retrieval processes, and finally biobehavioral hypotheses linking concurrent memory performance with EEG. I then discuss the major contributions of this work specific to the study of biobehavioral mechanisms involved in infant recall and to developmental cognitive neuroscience in general. Following this, I will reflect on limitations of this study and finally conclude by highlighting some future directions in developmental science that the outcomes of this work may inspire.

Behavioral Hypotheses. In Hypothesis 1, I stated that I expected infants to produce a greater number of ordered pairs of actions during the post-modeling test than during the pre-modeling baseline period. My data suggest that this was the case for both the immediate and delayed recall assessments. In both cases, infants produced more correctly-ordered pairs of actions after observing the modeling procedure than before (during the behavioral baseline period). The published means for imitation procedures, whether the design is between- (Barr et al., 1996; Collie & Hayne, 1999); Gross, Hayne, Herbert & Sowerby, 2002; Hayne, 2004) or within-subjects (Bauer et al., 2001; Bauer et al., 2003; Bauer, 2005, 2006; Carver & Bauer, 1999; Carver et al., 2000; Lukowski et al., 2005), are consistently low, and the means for this study were no different. Typically, baseline procedures in either protocols are close to zero, thus, addition of non-parametric analyses[2] was imperative in interpreting these findings. My inclusion of effect-size estimates for these findings, which have not been included in any published studies of infant recall work, may inform the field of the relative impact of the modeling procedure as a learning experience.

In addition, consistent with previous research (Bauer et al., 2001; Bauer et al., 2003; Bauer, 2005, 2006; Carver & Bauer, 1999; Carver et al., 2000; Lukowski et al., 2005), I expected

that approximately 50% of infants would show delayed ordered recall (one or more correctly order pairs of target actions) after the 24-hour delay. In my data, this percentage was lower; approximately 40% of the 10-month-olds in this study went on to correctly order at least one pair of actions. However, this reduced representation of ordered-recallers in my sample may be a function of the one-session of learning provided, as opposed to a 3-consecutive day training session as is used in the studies that report 50% of their sample producing ordered-pairs. The methodological decision to include only one training session for this investigation was made for both practical reasons (attempting more than 2 days of consecutive physiological recordings would have been additionally challenging for recruitment) and conceptual reasons.

From this study, it is clear that a tremendous amount of encoding occurred during the demonstration phase of the protocol, as can be observed in successful infant recall performance. In addition, the behavioral data indicate that for some infants, observing a second session of a demonstration (the reminder treatment in the savings assessment) was very helpful in subsequent recall tests. Also, from the comparing different patterns of brain-electrical activity associated with initial encoding and secondary re-encoding during the reminder treatment, it is clear that the neurological processing during a second encoding session is not identical to the first, most likely because the infant is undergoing both new encoding and retrieval (recognition and/or recall) of this now familiar event. Although fewer infants produced ordered-pairs of actions than I expected, the methodological adjustment that may have contributed to this distribution also allows for more conservative, and potentially accurate, interpretations of when and how infants encoded the modeled displays. This is especially crucial to consider when making statements highlighting the primary role of encoding processes in a biobehavioral model of early explicit memory (Bauer, 2005, 2006).

In Hypothesis 2, I stated that infants would show better memory performance on the test of immediate recall rather than delayed recall. This was not the case behaviorally, and based on the electrophysiological data from each phase of the assessments, it was unlikely that it was the case physiologically. Similar patterns of baseline-to-task EEG were observed regardless of delay. We know that adult and child explicit memory is susceptible to forgetting as a function of delay and distraction between encoding and retrieval. It may be that the 24-hour delay was not time to challenge the memory traces these infants created in the first visit, or it may be that during the delayed test, they compensated by capitalizing on retrieval cues in the testing environment (such as experimenter, and memorable characteristics in the testing room). It was clear that the task phase (demonstration vs. test) had much more of an impact than delay on behavior, physiology, and the combination of the two.

Hypothesis 3 stated that infants with difficulty in delayed recall performance would benefit from additional modeling, thus demonstrating savings in relearning. In my sample, it was not the case that the relearning procedure helped to improved delayed recall performance. Better recall performance following a relearning procedure (i.e. how much of the memory trace is present despite lack of initial recall), is often interpreted as the forgetting was due to failure in retrieval mechanisms. This sample's lack of a relearning effect conceptually suggests that when forgetting occurred, it was due to failures in initial encoding processes, rather than the degradation of a memory trace. It is unclear whether others studying infant recall abilities have found similar effects with their savings assessments, as although this procedure is often mentioned in published methodologies, not one of these investigations has published electrophysiological or behavioral data pertaining to the savings assessment (all note that first authors should be contacted for those results; Bauer et al., 2003, 2006; Lukowski et al., 2005).

In Hypothesis 4, I stated that memory performance would be unrelated to other sources of individual differences in infant socio-emotional and cognitive development including temperament scales (from the IBQ-r) and language development (MCDI-I). Although a few mild-to-moderate bivariate correlations were observed between recall performance and infant temperament scales and language measures, these predictors were not significant contributors to infant recall performance. These relations included poorer immediate recall being associated with higher levels of fear and perceptual sensitivity. In the testing situation, however, all infants, regardless of reported temperament, were quick to engage with the toys for the task. For infants high in fear, or who were highly perceptually sensitive, perhaps the social engagement necessary for observational learning of another's actions or the disengagement necessary to cease object exploration and move on to object action were difficult challenges. Similarly, infants who were rated as high on low-intensity pleasure by mothers also had poorer ordered-recall performance. The tasks for this study were designed to end in a surprising or rewarding end-state for the infant and many of the modeled actions involved circumstances that could be argued as higher intensity (shaking the rattle, hitting the gong, making the bunny jump by hitting the spatula).

For mnemonic correlations with some of the language measures, the direction of these effects was somewhat counterintuitive. Infants reported by mothers as more imitative (gesturally) and having higher language comprehension were likely to have displayed poorer recall memory. However, reported means on these measures were strikingly higher than reported norms (both were at least 25% above the published norms for 10 months of age; Fenson, et al., 1994), so care should be taken when interpreting language relations within this sample.

Electrophysiological Hypothesis. Hypothesis 5 pertained to an electrophysiology-only analysis of EEG during each phase of the recall assessments (immediate, delayed, and savings). I hypothesized that infants would show baseline-to-task increases in 6-9Hz EEG power at frontal and/or temporal locations during each phase of the imitation tasks. However, almost exclusively, the baseline-to-task changes that were observed, across phases and assessments, were decreases from baseline to task. Thus, the direction of the findings were in direct contrast to the direction of effects hypothesized (i.e. increases from baseline to task at scalp locations were observed in previous work from our lab; Bell, 2001, 2002; Wolfe & Bell, 2004; Morasch & Bell, *under review*). There were several frontal and temporal effects observed, but there was also consistent involvement of parietal (spatial), central (motor), and occipital (visual) regions, which, when given the demands of how infants were asked to display their event recall, was not surprising. In order to express their recall, infants had to interact with the toys, and thus had changes in activity at pre-motor cortical areas (central sites) and also had to represent and recreate the required actions which recruited cortical regions dominantly involved in visuo-spatial processing (parietal and occipital areas). Appropriate involvement of these cortical regions in these tasks suggest that this pattern of baseline-to-task decreases in EEG power may indicate a new pattern of physiology underlying cognitive processing that is specific to explicit memory separate from what can be expected from tasks and patterns describing infant working memory. This point will be further discussed in the context of recall performance in the next section.

Biobehavioral Hypotheses. From an a priori standpoint, hypothesis 6 was the major test hypothesis of the study, combining both behavior and physiology. I will discuss my findings first with regard to the immediate, then delayed, and finally the savings assessment. Just as in the analyses, recall performance will be discussed in group terms, with infants belonging to

either the zero-recall group (no ordered pairs produced) or the ordered-recall group (at least 1 pair or ordered-actions produced).

The first component of this hypothesis 6 stated that infants who showed ordered-recall (one or more pairs of correctly-ordered actions) would have different patterns of baseline-to-task EEG activation than those who did not. Significant and consistent differences were observed between the two recall performance groups across the three assessments. However, the direction of performance-related changes in EEG power that were observed were not the same as those hypothesized. Consistent with previous developmental cognitive neuroscience work examining baseline-to-task changes with successful cognitive performance in infants (Bell, 2001, 2002; Wolfe & Bell, 2004; Morasch & Bell, *under review*), in hypothesis 6, I expected that infants who evidenced ordered-recall would have displayed more regional activation during the recall tasks when compared to infants who were unsuccessful. Specifically, I hypothesized that ordered recallers would show increases in EEG power from baseline at temporal (T3/T4) sites during encoding and/or frontal (F3/F4) sites during retrieval. Additionally, I expected that infants who did not evidence ordered recall would show no significant baseline-to-task activation during either the encoding or retrieval phases. As discussed below, the data, however, show that an opposite pattern of performance-related changes in EEG power was observed, where generally, poor recall performance was evidenced by significant baseline-to-task decreases in EEG power, and successful ordered-recall was evidenced by no change in EEG power from baseline to task (with one exception of a baseline-to-task increase).

For the assessment of immediate recall, I found several biobehavioral effects during both the demonstration and testing phase. Within the demonstration phase, infants in the zero-recall group displayed a pattern of baseline-to-task changes in EEG power marked by significant task-

related decreases relative to their own baseline physiology. This occurred at bilateral medial and lateral frontal sites. In contrast, the ordered-recallers showed a different pattern of EEG activity, where better recall performance was indicated, not by baseline-to-task increases, but by no change from baseline-to-task at these frontal locations. Additionally, within the test phase of this assessment, I also found that the zero-recall group showed patterns of baseline-to-task decreases at bilateral medial frontal sites. While this was also the pattern for ordered-recallers at the right medial frontal site (F4), again, better recall performance was indicated by a lack of change from baseline to task at left medial frontal site (F3). To summarize, in the context of the immediate recall assessment, group related differences were observed at frontal regions where better performance, almost exclusively, were indicated by the lack of baseline-to-task changes, but weaker performance was marked by baseline-to-task decreases in EEG power.

For the delayed assessment, there were no observable effects at any frontal or temporal sites during the demonstration phase. However, during the delayed recall test, infants showed a similar pattern to that during the immediate recall test phase. Infants in the zero-recall group had baseline-to-task decreases at right lateral frontal and right posterior temporal sites. Infants in the ordered-recall group, however, displayed either no change from baseline to task (as was the case at bilateral posterior temporal sites) or task-related increases (at the lateral frontal cortex). Again, rather than the zero-group experiencing no EEG power changes from baseline-to-task, weaker performance was indicated by task-related decreases, whereas successful performance was noted by either no change or increases from baseline to task.

Finally, in the savings assessment, this pattern was reversed. Unlike the other two assessments, all performance-related effects were found at exclusively temporal locations. Within both the reminder condition and the subsequent re-test, the zero-recall group displayed no

baseline-to-task changes in EEG power, whereas the ordered-recall group displayed baseline-to-task decreases at right anterior temporal site T4. This pattern may reflect the fact that what is often labeled as a re-encoding episode (the reminder treatment) actually involves both encoding and retrieval processing, and that the re-test, as a second opportunity to recall a specified event from long-term memory, is also being affected by the previous imitation failure as well as short-term processing of the reminder treatment. In sum, the behavioral, and thus physiological demands during the savings assessment are fundamentally different than those in the encoding or test phase of both the immediate or delayed assessments, and care should be taken with any direct comparisons.

Thus, except for the savings assessment, better performance was indicated in most instances by the lack of a change from baseline-to-task EEG power and poor performance was almost exclusively indicated by baseline-to-task decreases in EEG power. Once again, this pattern is in contrast to the only published pattern of task-related EEG changes during cognitive tasks in infancy. However, this work, by Bell and colleagues, has always used infant working memory tasks as the cognitive measure in studying infant brain-electrical activity (for a review, see Bell & Morasch, 2007; Bell, 2001, 2002, Wolfe & Bell, 2004; Morasch & Bell, *under review*). Perhaps because recall tasks target a separate mnemonic system, it may be that the observed pattern of electrophysiological changes during cognitive processing in this study represent an appropriate pattern for infant explicit memory performance, as opposed to one omnibus pattern underlying generalized successful infant cognition. In the adult psychophysiological literature, Klimesch, Schimke, & Schwaiger (1994) argue that care should be taken to interpret directional task-related changes in EEG based on the context of the cognitive system being assessed as well as the frequency bands used for analysis. Klimesch et

al. (1994) argue that for working memory and explicit memory adult studies, different directions of effects are seen dependent on performance, and whether analyses are within the alpha or theta frequency bands. Late within the first year, the dominant frequency band for infants is 6-9 Hz (Bell & Fox, 1994) which has been shown to discriminate correct and incorrect performance in infant working memory (Bell, 2001). In infancy, this band shares some qualities with those of adult alpha and adult theta, but at the current time, not enough is known about frequency bands in developmental psychophysiology to label this infant band as directly representative of either. Therefore, just as in adulthood, expected patterns of biobehavioral expression of cognitive tasks in infancy should be qualified based on the type of memory system being assessed.

Hypothesis 7 referred to an exploration at the individual response level, to see whether different patterns of continuous EEG *within the same infant*, are present for correct and incorrect responses. I expected that within subjects, correct and incorrect performance (determined by the presence or absence of ordered recall) for each assessment would be related to different patterns of frontal (F3/F4) and or temporal (T3/T4) activity during encoding and retrieval. There were no observable differences within the immediate assessment, however, some interesting patterns representing correct and incorrect performance emerged from the delayed recall assessment. Specifically, a correct response was represented by 2 patterns: a less-negative decrease in frontal EEG (relative to an incorrect response from the same infant) as well as a larger increase in temporal EEG (again relative to an incorrect response from the same infant). It is interesting that this same pattern of negative task-related EEG power corresponded to poor performance on both the immediate and delayed recall assessments as well as incorrect performance within an individual child. This finding that across group performance and within individual responses, poor memory was discernable from successful memory (as was noted in other developmental

cognitive neuroscience investigations, Bell, 2001), lends weight to the argument that this pattern is valid in the biobehavioral expression of explicit memory in infancy.

Finally, Hypothesis 8 stated that frontal and temporal changes in EEG power during recall tasks would account for a significant portion of variance in ordered-recall performance. This hypothesis was tested to examine individual, rather than group, differences in recall performance and its' relations with frontal and temporal EEG. However, un-grouping the ordered-recallers to examine potential individual differences especially with those infants who correctly ordered pairs of actions above the requisite 1-pair for group membership, resulted in null biobehavioral results at each phase of both the immediate and delayed recall assessments. This analytical approach was novel to the study of infant recall processes and has not been reported in any of the literature on infant recall development, perhaps because what is more telling than how *many* sequences infants can correctly order, is whether or not they can display memory for temporal order of events at all.

Major Contributions. This study contributes to our understanding of the biobehavioral expression of infant explicit memory in three main ways. First, changes in frontal lobe activity are directly involved in explicit memory processing both during event encoding as well as retrieval. Prior to this work, evidence for frontal lobe involvement in this classically temporal-lobe task was inferred from evoked potential analyses between encoding and retrieval (responses were based on recognition of 2-dimensional photos of the stages of the event sequences; Bauer et al., 2003; Carver et al., 2000). It is now clear that changes in both frontal and temporal EEG power are involved in each processing stage of successful and unsuccessful infant recall performance.

Second, this work provides evidence of a developmentally appropriate and valid pattern of electrophysiology never before evidenced. Because so little work has been done to map continuous patterns of brain-electrical activity during cognitive tasks with developmental populations, it was understandable to expect a similar pattern of performance-related results in this study. However, in keeping with the caveats from adult psychophysiology, infant brain-electrical work should not be expected to hold to one single pattern. This study is the first to provide any data to trace continuous electrophysiological change in infant explicit memory processing during the creation of new event memories and short-and long-term recall of events.

Finally, this study bridges the gap between a classic behavioral task of infant memory (which has been conceptually linked to neuropsychological data) and current developmental cognitive neuroscience. A majority of the current studies examining infant brain mechanisms of cognition focus exclusively on evoked responses and recognition memory. However, I feel this particular technique (of averaging evoked responses to familiar and unfamiliar displays) is both un-naturalistic in the setting of infant recall processes, and because it requires the averaging of several (sometimes several hundred) individual responses to familiar and novel displays, it completely eliminates any opportunity to study one trial/session learning of an event, a hallmark of true explicit memory. Therefore, by examining continuous EEG during actual behavior, the interpretations of neurological systems central to infant memory are more valid.

Limitations. Important limitations of this study include the under-representation of ordered-recall within the sample, for reasons discussed above. This may have limited my ability to examine individual differences in successful ordered-recall performance and an analysis of multiple pathways to achieve successful recall. This may be rectified by choosing tasks that are easier for infants to perform and/or by giving infants additional demonstrations with each

modeled sequence. Also, because this is the first study to examine continuous physiology supporting recall processing, more work needs to be done to replicate the observed pattern of biobehavioral expression of infant recall. Finally, demographically the sample was quite homogenous, and though it reflected the racial and ethnic make-up of the surrounding area, according to the distribution of parents' educational status, this sample is not representative of a wide range of SES. Expanding the sample to include more variety on this dimension will help to increase generalizability.

Future Directions. Future studies based on this work should focus on age-related changes in the biobehavioral expression of infant explicit memory. Because we know that infant recall is newly emergent at 6 months and continues to develop and become more robust by the end of the first year, and that the frontal lobe, which this study shows is integral to this ability, also shows a dramatic developmental pattern across this time, examining the relation between the two from 6-12 months would be extremely informative in our understanding of the development of this memory system. Also, from an electrophysiological standpoint, investigating not only EEG power but coherence (the integration of two or more cortical systems) measures may also be informative in examining neurological development associated with infant recall. Finally, following infants into the early childhood period, when this type of memory is more commonly assessed with verbal recall, may tell us about the stability of this cognitive function in early development.

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End Notes.

1. This distance was calculated to be both within infants' visual range but beyond the distance where they will strain to reach for objects. Therefore, this distance reduces motor artifact during cognitive processing (Bell, 2001).

2. Because for any particular test in this study many infants did not produce any correctly-ordered pairs (and thus had proportional scores of zero), non-parametric assessments were also conducted to examine these within-subjects comparisons. By repeating the parametric analyses using the Wilcoxon Signed Ranks Test, a non-parametric technique designed to compare 2 related samples, an identical pattern of results was revealed. The modeling procedure significantly increased ordered-pair production in both the immediate assessment (Z=-2.01, p= .04) and the delayed assessment (Z= -3.27, p<.01). However, there was no effect of savings through relearning (Z= -1.46, p=.14).

Table 1. Neurological Correlates of Explicit Memory

Region	Function (s)	Development	Citations
Medial Temporal Lobe *(Hippocampus, Amygdala Para- hippocampal Cortex, Entorhinal Cortex, Dentate Gyrus*)*	Explicit Memory: • Encoding • Consolidation • Storage	Structurally and functionally mature early within the first year (0-3 months) (Except for the dentate gyrus which is more protracted)	(Nelson, 1995; Squire, 1992; Cohen & Squire, 1980; Zola & Squire, 2000; Eckenhoff & Rakic, 1991; O'Neil et al., 1986)
Prefrontal Cortex	• "Central Executive" • Retrieval of long-term memories • Temporal sequencing of actions	Structurally and functionally *delayed* (associated with performance by 8 months) and *protracted* (development continues into early adulthood).	Wheeler et al., 1997; Goldman-Rakic & Leung, 2002; Luria, 1966; Fuster, 2002; Nyberg et al., 1996; Liston & Kagan, 2002; Bachevalier, 1992; Chugani, 1994; Bell, 2001, 2002; Bell & Fox, 1994; Diamond, 2001, Bauer, 2004)
Temporal-Cortical Circuit *(MTL & PFC)*	Ordered-Recall Memory: • Encoding • Consolidation • Storage • Retrieval	Although some components would be mature early (MTL), the overall circuit's development would be driven by the protracted development of its other components (PFC & DG).	Bachevalier, 1992; Nyberg et al., 1996; Bauer, 2004.

Table 2. Behavioral & Electrophysiological Indices of Recall

Age	Recognition Test (ERP)	Retention Interval	Key Results	Citation
9 mos.	1 week after final exposure	5 weeks	Infants who showed delayed ordered recall, had delayed ERP's which differentiated between novel and familiar stimuli. Infants who did not show ordered recall did not differentiate between novel and familiar photos 1 week after encoding.	Carver, Bauer, & Nelson (2000)
9 mos.	Immediately after final exposure & 1 week after final exposure	5 weeks	Infants who showed delayed ordered recall, had immediate and delayed ERP's which differentiated between novel and familiar stimuli Infants who did not show ordered recall showed novel and familiar photos 1 week after encoding. Therefore, differences in delayed recall were not due to differences in encoding.	Bauer, Wiebe, Carver, Waters, & Nelson (2003)
9 & 10 mos.	After second exposure	1 month	All infants showed immediate recognition (ERP's distinguished between novel and familiar stimuli). However, these ERP differences were more striking at 10 months, suggesting developmental differences in encoding. Only 10-month-olds showed ordered recall. Therefore, differences in delayed recall may be due to developmental differences in encoding.	Bauer, Wiebe, Lukowski, Haight, Waters, & Nelson (2005)

Table 3. Schematic Representation of the Research Protocol

Day	Baseline Assessment	Modeled Events	Recall Testing	Remodeled Events	Savings Assessment
1	ABC	ABC	--------	--------	--------
		24 Hour	Delay	(+/- 2 hrs.)	
2	--------	--------	ABC (delayed)	ABC	ABC
	DEF	DEF	DEF (immediate)	--------	--------

Note: For this example, on Day 1, infants were presented with event sequences ABC (this was counterbalanced with events DEF). The retention interval was 24 hours. On day 2, the testing session, infants were tested for both immediate recall (of day 2 events DEF) and delayed recall (of day 1 events ABC; the order of these 2 tests were also be counterbalanced). Additionally, savings from relearning of day 1 events ABC was assessed.

Table 4. Correlations between Recall Performance and IBQ-r Temperament Scales

	Immediate Recall	Delayed Recall
Activity Level	.08	-.15
Approach	.06	-.15
Cuddliness	-.04	-.10
Distress	-.12	.12
Falling Reactivity	.20	.11
Fear	-.27^{+}	.01
High-Intensity Pleasure	.11	-.18
Low-Intensity Pleasure	.09	-.29*
Orienting	-.04	-.20
Perceptual Sensitivity	-.28^{+}	-.16
Sadness	-.11	.02
Smiling	-.12	-.24
Soothability	-.24	.13
Vocal Reactivity	-.26^{+}	.04

Note: All temperament correlations with immediate recall group had a sample size of n=47. Correlations between delayed recall group and temperament had n=48.

* $p < .05$

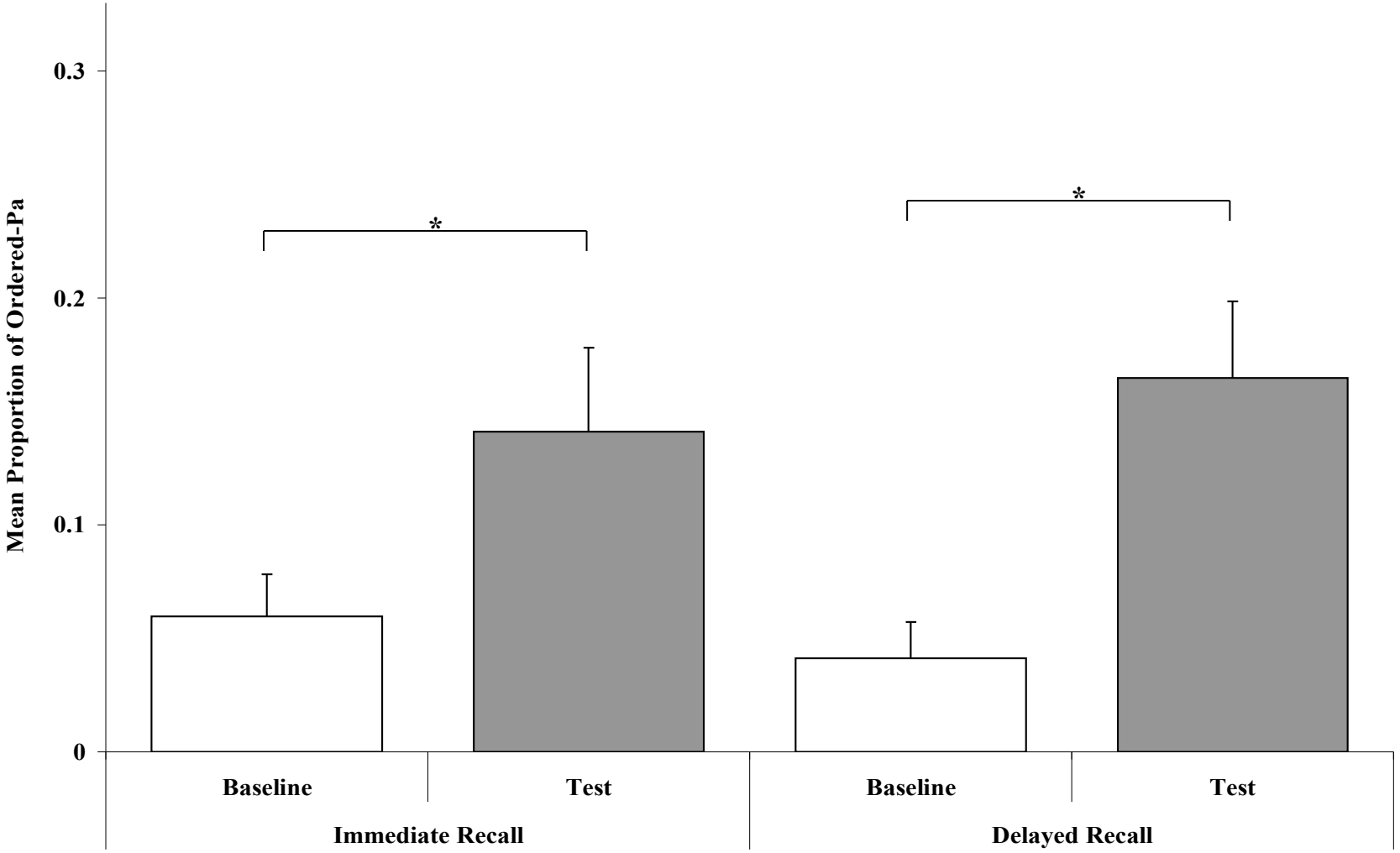
$^{+}$ $p < .10$

Table 5. Condition x Region Interaction Effects for each Phase and Recall Assessment

	Demonstration Phase		Test Phase	
Immediate Recall: df (7, 39)				
	F	η^2	F	η^2
Fp1/Fp2	0.09		.16	
F3/F4	2.47		16.15*	.26
F7/F8	1.25		.01	
T3/T4	0.08		2.33	
T7/T8	4.49*	.09	3.94*	.08
C3/C4	0.77		14.21*	.24
P3/P4	4.26*	.09	17.60*	.28
O1/O2	0.04		4.25*	.09
Delayed Recall: df (7, 38)				
Fp1/Fp2	0.52		0.50	
F3/F4	1.37		8.63*	.16
F7/F8	1.09		0.17	
T3/T4	0.05		0.01	
T7/T8	1.24		4.15*	.09
C3/C4	13.86*	.24	20.08*	.31
P3/P4	6.96*	.14	11.63*	.21
O1/O2	0.54		1.18	
Savings Assessment: df (7, 36)				
Fp1/Fp2	0.18		0.05	
F3/F4	6.56*	.14	8.48*	.17
F7/F8	0.06		0.08	
T3/T4	0.13		0.01	
T7/T8	4.61*	.10	1.91	
C3/C4	9.04*	.18	15.98*	.28
P3/P4	5.30*	.11	7.83*	.16
O1/O2	0.40		1.30	

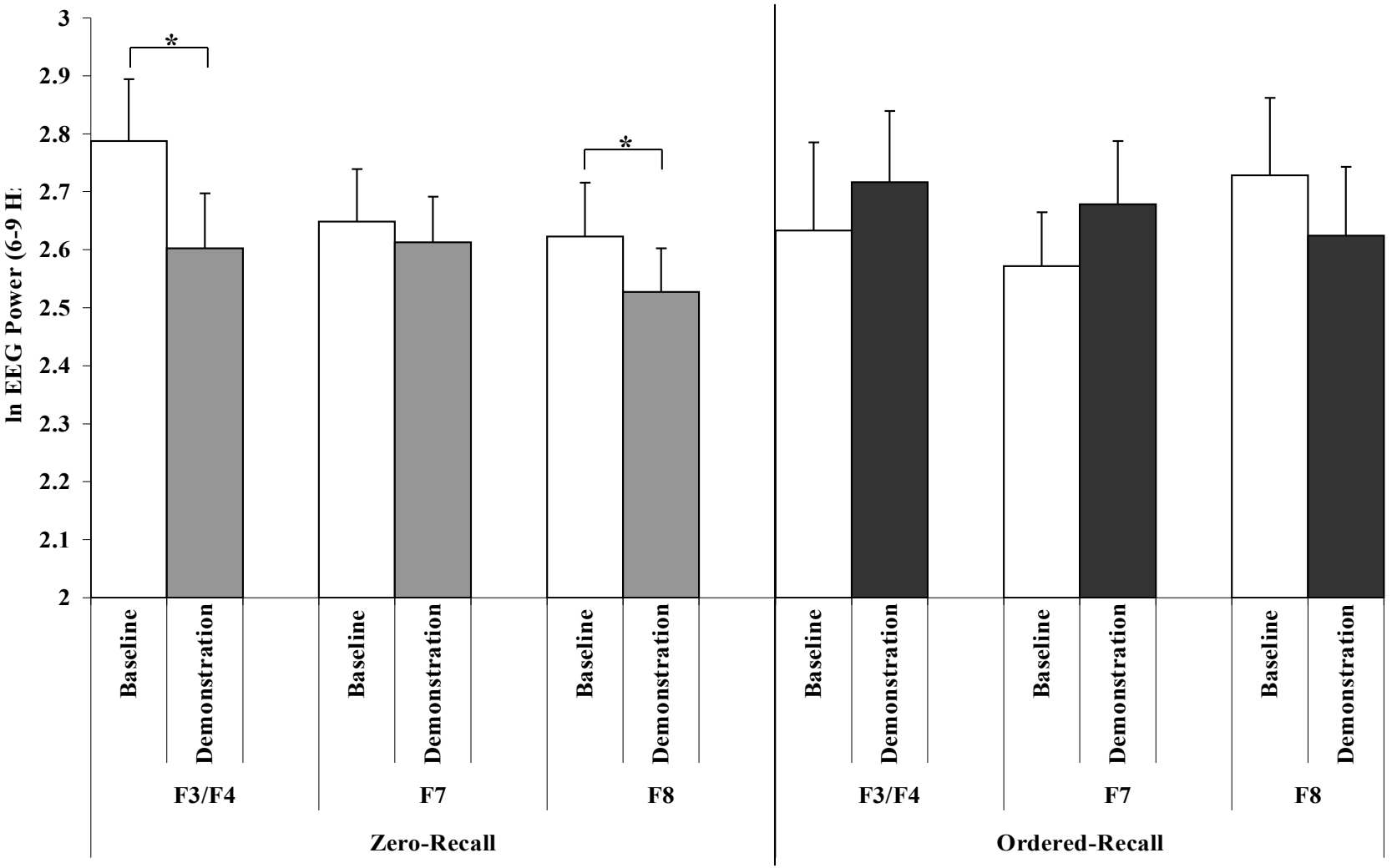
* $p < .05$

Figure 1. Recall Performance on Immediate and Delayed Assessments



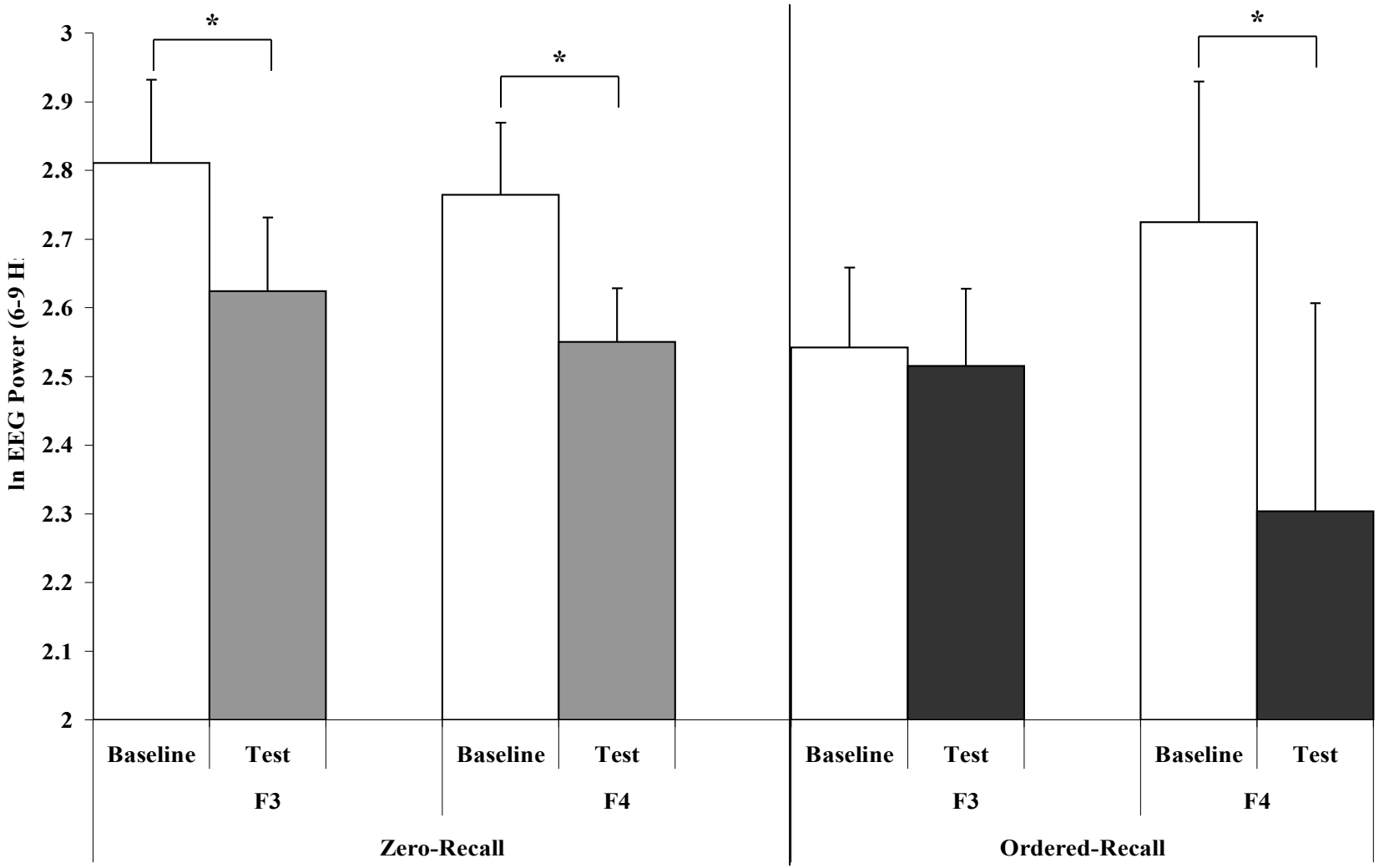
* p<.05

Figure 2. Baseline-to-Task EEG and Immediate Recall Performance (Demonstration Phase)



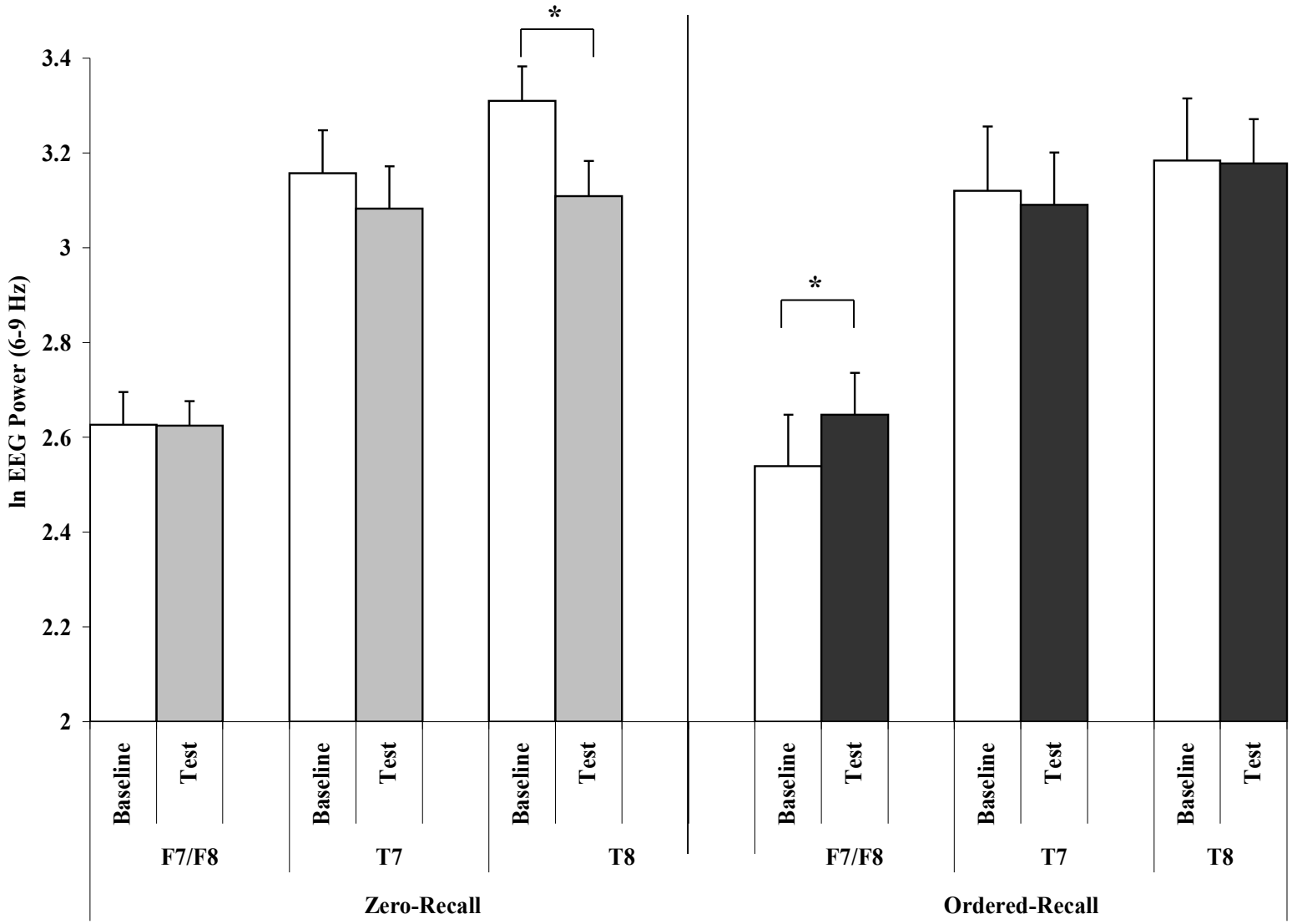
*p<.05

Figure 3. Baseline-to-Task EEG and Immediate Recall (Test Phase)



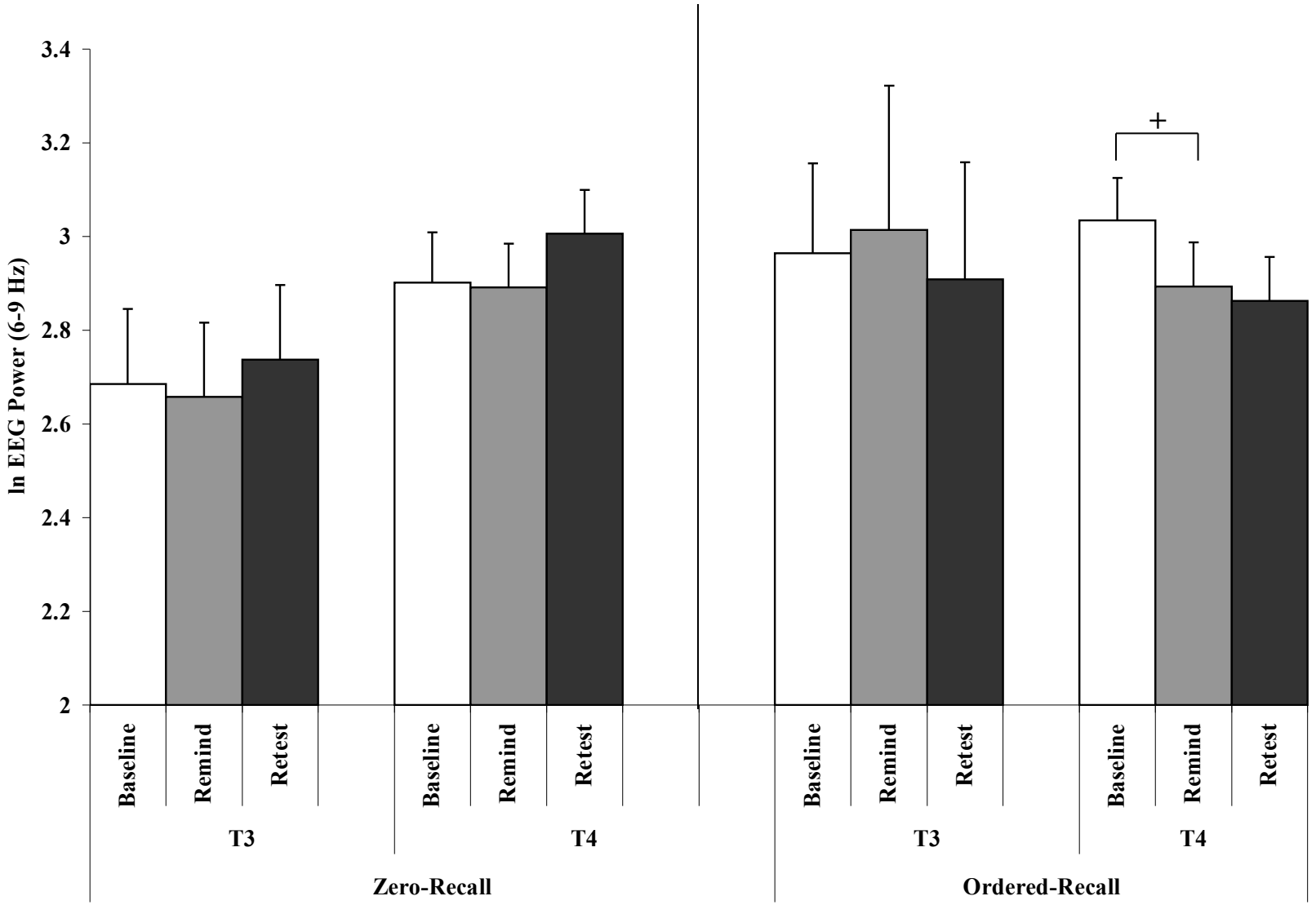
- p<.05

Figure 4. Baseline-to-Task EEG and Delayed Recall Performance (Test Phase)



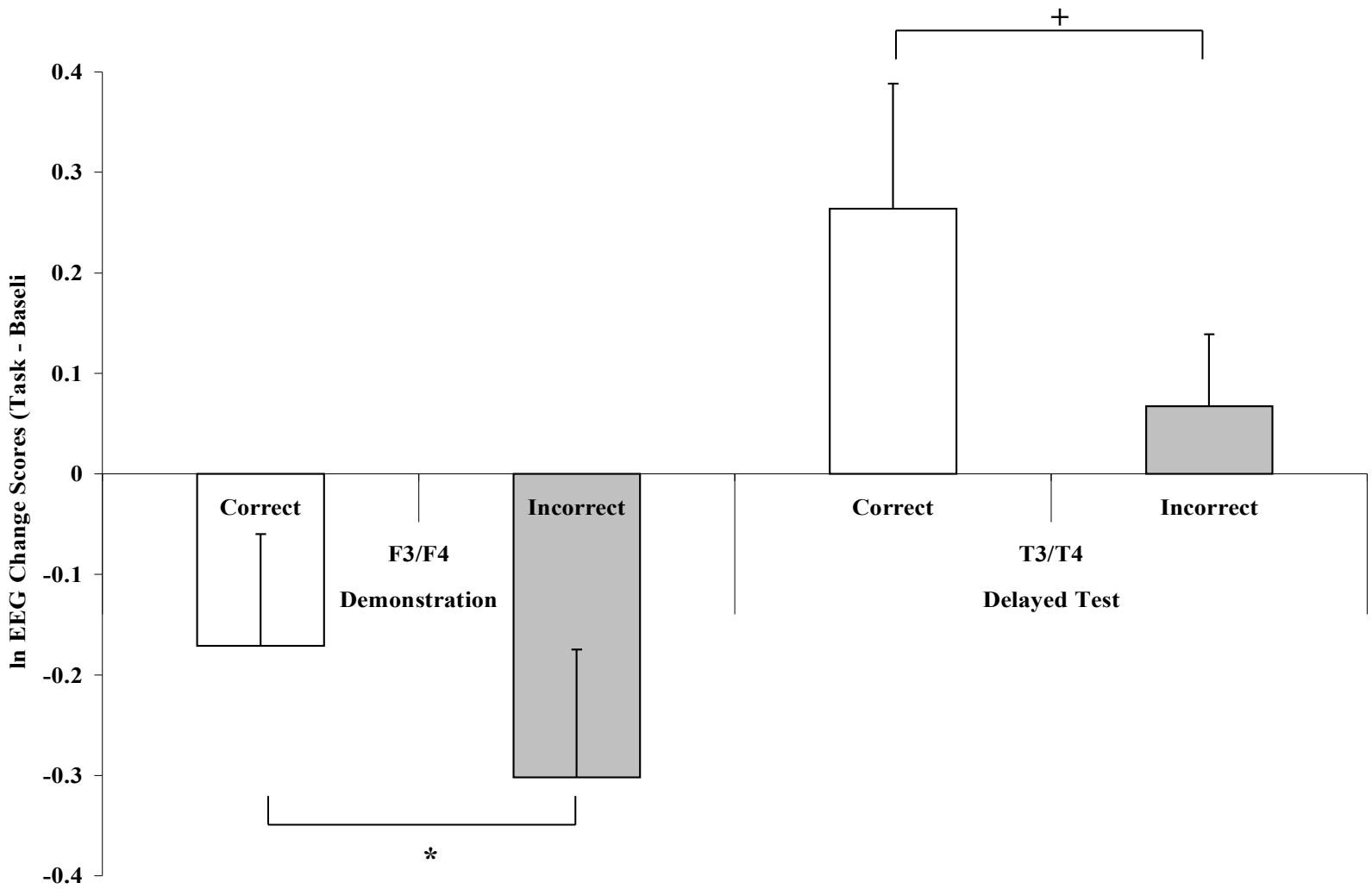
*p<.05

Figure 5. Baseline-to-Task EEG and Savings Performance (Reminder and Retest Phases)



+ p<.10

Figure 6. Correct and Incorrect Performance on the Delayed Recall Assessment



* p<.05

+ p=.06

Appendix A. Recruitment Letter

April 6, 2007

Dear Parents:

Isn't it amazing how quickly the first year flies by! By now, your infant is a "professional thinker", building memories for all of the exciting people, places, and experiences of their lives. This growing skill opens a whole new world of possibilities and adventure for you and your infant. Here at the Psychology Department at Virginia Tech, we are studying infant memory and attention with 10-month-olds in the New River Valley area. We are looking for 50 infants and their parents to join us in this study. We would like to invite you and your child to participate!

In this project, we involve your infant in several games where they search and reach for hidden toys, explore some new and old puppets and toys, and remember experiences from one day to the next. Most of these games will be played whereas your infant wears our little stretchy EEG cap, so we can see what the brain is doing whereas he/ or she plays our games. Also, little patches will be applied to your infant's chest so that we can see what the heart is doing whereas he/ or she plays our games. There will also be some questions that we will ask you to answer about your infant's general patterns of behavior and language understanding.

This is a two-part study in which parents and infants visit the C.A.P. Lab at Virginia Tech on **two consecutive days** and will spend a total of 90 minutes with us (45 min. for visit one, and 45 min. for visit two). Because this is a student dissertation project, your infant will receive 2 small gifts as a "thank you" for participating in the study (a personalized hand-painted piggy bank and a T-shirt).

Our research lab is in Williams Hall (on the Virginia Tech campus) and is located on the Drill Field, where we have reserved parking for participants in our research projects. You may already be familiar with our infant and child research program at Virginia Tech if your child participated in our ongoing Child Development Project or other studies in the Developmental Sciences Research suite.

Would you be interested in hearing more about this study? One of us will be calling you within the next few days to talk with you about our study. Agreeing to talk with us over the phone does not obligate you to participate. We want to tell you all the details before you decide whether or not you and your infant would like to participate. In the meantime, feel free to visit the web site for our research lab. You can read about similar *Infant Development Projects* and see photos of infants who have been involved with our studies.

http://www.psyc.vt.edu/devcogneuro

If you wish, feel free to call us at your convenience. We can be reached at 231-2320 (research lab) and by e-mail (Mrs. Morasch's address is *kacolo@vt.edu)*. Thank you and we look forward to talking with you!

Sincerely,

Katherine C. Morasch, M.A.
Graduate Student of Psychology

Martha Ann Bell, Ph.D.
Associate Professor of Psychology

Appendix B. Descriptions of Events Used in Recall Testing

1. *Make a gong.* This event consisted of a U-shaped base with a thin dowel connecting the 2 side panels, a small brass bell attached to an S-hook, and a wooden dowel with a ball attached to one end. The experimenter modeled (a) hanging the bell from the bar (Step 1) and hitting it with the dowel (Step 2) making a ringing sound.

2. *Find the froggy.* This event consisted of a small wooden box with a hinged lid and transparent sides and top and a rubber frog toy placed inside the box. The experimenter modeled (a) opening the lid (Step 1) and removing the frog from inside the box (Step 2).

3. *Pop-up Book*: This event consisted of a child's pop-up book adjusted so that it would only open to the designated pages, and a flap to open on that page. The experimenter modeled (a) opening the book (Step 1) and (b) opening the flap (Step 2) making the picture of the fish pop up.

4. *Make a rattle.* This event consists of a flexible plastic tube with orange rubber covers on both ends (one end has a small opening in the rubber) and a small plastic figurine. The experimenter modeled (a) inserting the figurine into the rubber end of the plastic tube (Step 1) and (b) shaking the tube (Step 2) making a rattling sound.

5. *Water the flowers*. This event consisted of a small plastic flower pot, a small yellow plastic watering can, and a short pick of artificial red carnations. The experimenter modeled (a) inserting the flowers into the pot (Step 1) and (b) lifting and tilting the can (Step 2) giving the flowers a "drink".

6. *Make the bunny jump*. This event consists of a round piece of PVC pipe (1.5" diameter) affixed beneath the bent handle of a flexible kitchen spatula with a large flat base, and a lightweight plastic bunny toy. The experimenter modeled (a) placing the bunny on the base of the spatula (Step 1) and (b) pushing down on the opposite end (Step 2) causing the bunny to "jump" in the air.

Appendix C. General Information Questionnaire

Infant ID number _____

Date of visit _____

1. Sex of baby: M F

2. Date of birth _____

3. Weight at birth _____

4. What was the expected due date? _____

5. Did your child receive any oxygen at birth or soon thereafter?
   _____ no
   _____ yes

6. Has your child experienced any serious illness or problems in development?
   _____ no
   _____ yes-----brief explanation_____

7. Has your child ever had any neurological problems, such as epilepsy, or seizures of any kind?
   _____no
   _____yes-----brief explanation _____

8. Has your child received any long term medication?
   _____ no
   _____ yes-----brief explanation_____

9. Is your child ill or on any medications now?
   _____no
   _____yes------brief explanation _____

10. Has your child shown an allergic reaction to anything?
    _____no
    _____yes-----brief explanation_____

11. Has your child ever had any skin irritations?
    _____ no
    _____yes

12. Age of parents at infant's birth:
    Mother_____
    Father _____

13. Ethnic group of parents:

Mother _____ Hispanic or Latino
_____ not Hispanic or Latino

Father _____ Hispanic or Latino
_____ not Hispanic or Latino

14. Racial group of parents:

Mother_____ American Indian / Alaska Native
_____ Asian
_____ Native Hawaiian or Other Pacific Islander
_____ Black or African American
_____ White

Father _____ American Indian / Alaska Native
_____ Asian
_____ Native Hawaiian or Other Pacific Islander
_____ Black or African American
_____ White

15. Highest level of education completed: (please note any "in progress")

Mother_____ High school
_____ Technical school
_____ College
_____ Graduate school

Father _____ High school
_____ Technical school
_____ College
_____ Graduate school

16. Which hand do you <u>prefer</u> to use for each of these activities?
Please put **R** (right hand), **L** (left hand), or **E** (either hand).

	Mother	**Father**
a. Writing	_____	_____
b. Drawing	_____	_____
c. Throwing	_____	_____
d. Scissors	_____	_____
e. Toothbrush	_____	_____
f. Knife (without fork)	_____	_____
g. Spoon	_____	_____
h. Broom (upper hand)	_____	_____
i. Striking match (to hold match)	_____	_____
j. Opening jar (hand on lid)	_____	_____
* * * * * * *		
k. Which foot do you prefer to kick with?	_____	_____
l. Which eye do you use when using only one?	_____	_____

Appendix D. Parental Permission Form

VIRGINIA POLYTECHNIC INSTITUTE AND STATE UNIVERSITY
Parental Permission for Participants
of Investigative Projects

Title of Project: "Cognitive Developments in Infancy: The Impact of Cortical Reorganization"
Investigators: Katherine C. Morasch, M.A. & Martha Ann Bell, Ph.D.

I. Purpose of this Research

You and your infant have been invited to participate in a research project investigating the development of several types of memory at 10 or 12 months of age. Specifically, we are examining how brainwave activity is associated with infant attention and memory task performance. The information we gather in this research study will further our knowledge of how infants develop important attention and memory skills. A total of 96 infants and parents will be participating in this study.

II. Procedures

This study involves two visits to the Infant Development Project Lab (Williams Hall 348) at Virginia Tech. The visits will occur when your child is 10 or 12 months of age, and will occur over two consecutive days. The first visit will last 45 minutes, and the second visit will last 45 minutes. Your infant will sit next to you in a high chair throughout the visit. The entire session will be video taped. This study also involves 3 questionnaires (General Information Questionnaire, Infant Behavior Questionnaire, and the MacArthur Communicative Development Inventory). We ask that you to try to complete these brief forms at home prior to your infant's visit to our research lab.

First Visit: The first visit will last approximately 45 minutes. During the first visit, electrophysiological measures will be taken during three attention and memory games. A small green cap will be placed on your infant's head in order to help us study brainwave activity. This cap looks and fits like an infant swimming cap. In order to collect brainwave activity, gel will be applied to your infant's hair through several small holes in the cap. These procedures are similar to those used in a doctor's office and are not harmful to your infant. We will gladly make a Polaroid photo of your infant in our "Science Baby brainwave and heart rate" attire so that you have a snapshot for your scrapbook. We will also ask you if we can make a second photo for out lab bulletin board. In addition, two adhesive patches will be placed on your infant's chest to record heart rate activity. While brainwave activity and heart rate activity are being recorded for 1 minute, your infant will be looking at an infant toy with colored balls being tumbled in it. We have found that 10 and 12-month-old infants like this toy and will sit quietly and watch it. Brain-wave activity and heart rate activity will also be recorded during the rest of the research session.

We will play four attention and memory games with your infant while he/or she is seated next to you in the high chair. First, we will observe how your infant pays attention to two puppets and toys he/or she has never seen before. Next, we will study how your infant searches for a hidden toy. We will be making note of where your infant looks to determine the strategy your child uses in searching for the toy. Finally, we will present your infant with two puppets to look at; an old one he/or she has already seen, and a new one they have not yet seen. We will be making note of where your infant looks to determine which puppet he/or she prefers. After these recordings, the cap and sticky patches will be removed and the gel will be washed from your infant's hair with warm water and a clean washcloth. After this last task is completed, we will give you and your child a small gift as a token of our gratitude.

Second Visit: The second visit will last approximately 45 minutes. During the second visit, we will play two attention/memory games with the electrophysiological measures as before. While your infant is sitting on your lap, we will observe how your infant interacts with the puppet and toys he/or she saw during the first visit. Next, we will again study how your infant searches for a hidden toy, but this time, we will watch where your infant reaches to determine the strategy your child uses in searching for the toy. After this last task is completed, we will give you and your child a small gift as a token of our gratitude.

III. Risks

There is minimal risk associated with this research project. The brainwave procedures are similar to that done in a doctor's office and are not harmful. All brain-wave equipment is disinfected after each use. The heart rate equipment is disposable. Toys are disinfected after being handled by each infant. If your child has an allergy to skin lotions, please inform us so that we can discuss the allergy and determine if any procedural changes need to be made. Our EEG gels are water based, but do contain the same preservatives that are used in everyday skin lotions.

IV. Benefits of This Research

There are no tangible benefits for you or your infant. No promise or guarantee of benefits has been made to encourage you and your infant to participate in this study. In a scientific sense, however, this research study will give developmental specialists more information about the development of memory during infancy. Upon completion of this study, we will send you a letter briefly outlining the findings of this research.

V. Extent of Confidentiality

Information gathered for this study will be confidential and the information from each individual baby will be identified by code number only. Information linking infant name and code number will be kept in a file and locked in a file drawer. Only Dr. Bell and her graduate Research Assistants will have access to the file. Your infant will be videotaped during the lab procedure. This allows us to go back at a later date and code your infant's search behaviors. Videotapes will identify infants only by code number. Tapes will be stored in the research lab and will be accessible only to Research Assistants associated specifically with this research project. Dr. Bell will supervise the confidentiality of the videotapes. Tapes will be erased five years after publication of the results of this study.

VI. Compensation

You and your infant will receive a hand-painted personalized piggy bank and an infant T-shirt for participation in the laboratory visits for this research study.

VII. Freedom to Withdraw

You may withdraw your infant from participation in the lab visit portion of this research study at any time without penalty. You and your infant will still receive the small gifts.

VIII. Review of the Research Protocol by the IRB

Federal laws and regulations governing the protection of human subjects involved in research require that study procedures be reviewed by an institutions' Institutional Review Board (IRB). The Virginia Tech IRB has reviewed this research study, and has found that its methods and procedures comply with the

requirements/standards established in applicable federal laws and regulations governing the protection of human subjects. In deciding whether to participate in this study, you must determine for yourself whether the risks and benefits of participation are acceptable.

IX. Parent's Responsibilities

We will ask you to complete questionnaire data.

X. Parent's Permission

I have read and understand the Informed Consent and conditions of this research study. I have had all my questions answered. I hereby acknowledge the above and give my voluntary consent for my infant to participate in this project. I understand that I may withdraw from participation at any time without penalty. I understand that I will be given a copy of this consent form.

_____ _____

Parent's signature Date

Should I have any questions about this study, I may contact:

1) Katherine C. Morasch, M.A.
   Investigator, Graduate Student of Psychology, 231-2320
2) Martha Ann Bell, Ph.D.
   Investigator, Associate Professor of Psychology, 231-2546
3) David W. Harrison, Ph.D.
   Chair, Psychology Department Human Subjects Committee, 231-4422
4) David Moore, Ph.D.
   Chair, IRB, CVM Phase II, 231-4991

Photographer's Release

I understand that the photographs taken of my child are the property of Virginia Tech. These photographs will be displayed in a public place, specifically, on a bulletin board in the Research in Developmental Processes Suite in the Department of Psychology. These photographs will be used to illustrate Department of Psychology research at professional conferences, in professional publications, and/or in university/departmental literature (print and internet).

_____ _____

Parent's signature Date

KATHERINE COLONA MORASCH, Ph.D.
Virginia Polytechnic Institute and State University
Department of Psychology (0436)
329 Williams Hall
Blacksburg, VA 24061
Primary Phone: (540) 250-0712
Office Phone: (540) 231-2320
Email: kacolo@vt.edu

EDUCATION

2007 Ph.D. Virginia Polytechnic Institute and State University Blacksburg, VA
Mentor: Martha Ann Bell, Ph.D.
Concentration: Developmental Psychology
Dissertation: "Explicit Memory and Brain-Electrical Activity in 10-month-old Infants"

2003 M.A. The College of William & Mary Williamsburg, VA
Mentor: Pamela S. Hunt, Ph.D.
Concentration: General Experimental Psychology
Thesis: "The Effects of Postnatal Ethanol Exposure on the Behavioral Development of Adolescent Rats: Primary and Secondary Deficits"

2001 B.S. The College of William & Mary Williamsburg, VA
Mentor: Pamela S. Hunt, Ph.D.
Concentration: Biological Psychology

ACADEMIC POSITIONS

2005-2007 Instructor, Virginia Polytechnic Institute and State University
Courses Taught:
The Psychology of Learning
Developmental Psychology
Advanced Developmental Psychology

2004-2006 Graduate Research Assistant, Program for Developmental Sciences across the Lifespan, Virginia Polytechnic Institute and State University

2003-2005 Teaching Assistant, Virginia Polytechnic Institute and State University
Courses Taught:
Developmental Psychology Laboratory
Introductory Psychology Recitation

2001-2003 Teaching Assistant, The College of William & Mary
Courses Taught:
Introduction to Statistics Laboratory
Introduction to Research Methods Laboratory
Research Methods in Comparative Psychology Laboratory
Research Methods in Physiological Psychology Laboratory

2002 Graduate Research Assistant, The College of William & Mary

Behavioral Neuroscience Laboratory

2002 Network Administrator, The College of William & Mary, Williamsburg VA
Research Participation Pool- Experimetrix System

RESEARCH

Published Papers

Hunt, P.S. & **Morasch, K.C.** (2004). Modality-specific deficits in response habituation following postnatal binge ethanol. *Neurotoxicology & Teratology, 26, 3,* 451-459.

Book Chapters

Bell, M.A. & **Morasch, K.C.** (2007). Individual differences in the development of working memory during infancy. In L.M. Oakes & P.J. Bauer (Eds.) *Short and Long-term Memory in Infancy and Early Childhood*, pp. 27-50. New York:Oxford University Press.

Papers under Review

Morasch, K.C. & Bell, M.A. (under review). Behavioral and electrophysiological associations among recognition, recall, and working memory at 12 months. Submitted to *Infant Behavior and Development.*

Papers in Preparation

Morasch, K.C. & Bell, M.A. (in preparation). Brain electrical activity and infant recognition memory. To be submitted to *Developmental Psychobiology.*

Invited Talks

Morasch, K.C. (April, 2007). Biobehavioral indices of recognition and recall in infants, toddlers, and adults. Invited talk given to at the Datapalooza speaker series in the Developmental and Biological Psychology area of the Department of Psychology at Virginia Tech, Blacksburg,VA.

Morasch, K.C. (March, 2007). Cognitive Development: The Role of Information Processing. Invited talk given to the Department of Psychology, Roanoke College, Salem, VA.

Morasch, K.C. (February, 2007). Biobehavioral indices of explicit memory performance in infants, toddlers, and adults. Invited talk given to the Child and Family Research Section of the National Institute of Child Health and Human Development (NICHD), Bethesda, MD.

Morasch, K.C. & Bell, M.A. (August, 2006). EEG patterns during recognition memory in 12-month-old infants. Student paper symposium held at the annual meetings of the American Psychological Association, New Orleans, LA.

Oral presentations given at professional meetings

Morasch, K.C. & Bell, M.A. (October, 2006). Relations between infant brain-electrical activity and toddler recognition memory. NIH travel awardee talk given at the meetings of the International Society for Developmental Psychobiology, Atlanta, GA.

Bell, M.A. & **Morasch, K.C.** (April, 2005). The development of working memory during infancy and early childhood. In L.M. Oakes & P.J. Bauer (Chairs), *Short- and Long-Term Memory in Infancy and Early Childhood: Taking the First Steps Toward Remembering*. Symposium held at the biennial meetings of the Society for Research in Child Development, Atlanta, GA.

Morasch, K.C. & Bell, M.A. (June, 2004). Frontal and temporal EEG differences and novelty preferences in 5-month-old infants. NIH travel awardee talk given at the meetings of the International Society for Developmental Psychobiology, Aix-en-Provence, France.

Colona, K.A. & Hunt, P.S. (October, 2002). Binge ethanol exposure during the brain growth spurt causes persistent deficits in response habituation in pre- and post- weanling rats. NIH travel awardee talk given at the meetings of the International Society for Developmental Psychobiology, Orlando, FL.

Posters presented at professional meetings

Morasch, K.C. & Bell, M.A. (March, 2007). EEG correlates of toddler recognition and recall memory performance. Paper to be presented at the meetings of the Society for Research on Child Development, Boston, MA.

Morasch, K.C. & Bell, M.A. (March, 2007). The central role of 2-year-old inhibitory control: Relations among cognition, emotion, and brain-electrical activity. Paper to be presented at the meetings of the Society for Research on Child Development, Boston, MA.

Bell, M.A., **Morasch, K.C.** & Wolfe, C.D. (March, 2007). Individual differences in cognition at 24 Months: Contributions from self-regulatory factors at 10 and 24 months. Paper to be presented at the meetings of the Society for Research on Child Development, Boston, MA.

Smith, C.L., Bell, M.A., **Morasch, K.C**. & Wolfe, C.D. (March, 2007). Stability in frontal lobe asymmetry as a predictor of toddlerhood internalizing and externalizing behaviors. Paper to be presented at the meetings of the Society for Research on Child Development, Boston, MA.

Buonomano, L., **Morasch, K.C.**, & Bell, M.A. (March, 2007). Paper to be presented at the meetings of the Society for Research on Child Development, Boston, MA.

Morasch, K.C. & Bell, M.A. (October, 2006). Relations between infant brain-electrical activity and toddler recognition memory. Paper presented at the meetings of the International Society for Developmental Psychobiology, Atlanta, GA.

Buonomano, L., **Morasch, K.C.**, & Bell, M.A. (October, 2006). Performance of 2-year-old children on the dimensional change card sort task. Paper presented at the meetings of the International Society for Developmental Psychobiology, Atlanta, GA.

Morasch, K.C. & Bell, M.A. (June, 2006). Differential patterns of frontal EEG and novelty preferences in 10-month-old infants. Paper presented at the Biennial International Conference on Infant Studies, Kyoto, Japan.

Bell, M.A., Wolfe, C.D., **Morasch, K.C.** & Cardell, A.C. (June, 2006). Self-regulation and individual differences in infant cognition. Paper presented at the Biennial International Conference on Infant Studies, Kyoto, Japan.

Smith, C.L., Bell, M.A., Wolfe, C.D., & **Morasch, K.C**. (June, 2006). Infant EEG as a predictor of toddlerhood behavior problems. Paper presented at the Biennial International Conference on Infant Studies, Kyoto, Japan.

Morasch, K.C. & Bell, M.A. (November, 2005). Relations among spatial working memory, recognition memory, and explicit memory in 12-month-old infants. Paper presented at the meetings of the International Society for Developmental Psychobiology, Washington, D.C.

Morasch, K.C. & Bell, M.A. (August, 2005). Electrophysiological Correlates of Deferred Imitation in 12-month-old Infants. Paper presented at the meetings of the American Psychological Association, Washington, D.C.

Morasch, K.C. & Bell, M.A. (April, 2005). Visual recognition memory and brain electrical activity in 5- & 10-month-old infants. Paper presented at the meetings of the Society for Research on Child Development, Atlanta, GA.

Morasch, K.C. & Bell, M.A. (June, 2004). Frontal and temporal EEG differences and novelty preferences in 5-month-old infants. Paper presented at the meetings of the International Society for Developmental Psychobiology, Aix-en-Provence, France.

Morasch, K.C. & Bell, M.A. (June, 2004). Frontal and temporal EEG differences and novelty preferences in 5-month-old infants. Paper presented at the joint meetings of the Behavioral Genetics Association and the International Society for Developmental Psychobiology, Aix-en-Provence, France.

Morasch, K.C. & Hunt, P.S. (May, 2004). Binge-like postnatal alcohol exposure induces alterations in response habituation: The duration and modality specificity of effects. Paper presented at the meetings of the International Society on Infant Development, Chicago, IL.

Miranda, D.O., Rima, B.N., **Colona, K.A.** & Hunt, P.S. (May, 2003). Neonatal binge ethanol affects olfactory, but not auditory, memory. Paper presented at the meetings of the Society of Young Neuroscientists and Professors in the South East (SYNAPSE), Harrisonburg, VA.

Colona, K.A. & Hunt, P.S. (October, 2002). Binge ethanol exposure during the brain growth spurt causes persistent deficits in response habituation in pre- and post- weanling rats. Paper presented at the meetings of the International Society for Developmental Psychobiology, Orlando, FL.

Colona, K.A., Miranda, D. O., Islam, S.N., & Hunt, P.S. (June, 2002). The persistence of response habituation deficits following PD 4-9 binge ethanol exposure. Paper presented at the meetings of the Research Society on Alcoholism, San Francisco, CA.

Colona, K.A. & Hunt, P.S. (November, 2001). Temporal determinants of alcohol-induced deficits in response habituation. Paper presented at the meetings of the International Society for Developmental Psychobiology, San Diego, CA.

Hunt, P.S., **Colona, K.A.,** Hrushka, M., & Hillard, M. (June, 2001). Attention deficits arising from neonatal alcohol exposure: Evaluation of response habituation. Paper presented at the meetings of the Research Society on Alcoholism, Montreal, Quebec, Canada.

HONORS, PRIZES, & AWARDS

Student Travel Award to attend the Biennial Meetings of SRCD	2007
Early Career Research Travel Grant from the APRICA Childcare Institute of Japan	2006
Virginia Tech Nominee for the APA Dissertation Research Award	2006
Virginia Tech Nominee for the APF/COGDOP Dissertation Research Award	2006
APA International Affairs Travel Grant	2006
APA Convention Student Travel Award	2006
Graduate Research Development Project Award, Virginia Tech	2006
Psychology Representative at the VT College of Science Alumni Roundtable	2006
VT Graduate Student Association Travel Fund Award, annually	2004- 2007
Galper Graduate Fund Award, Department of Psychology, Virginia Tech	2004, 2005, 2006
National Institutes of Health Student Travel Awards to annually attend ISDP	2001-2006
The College of William and Mary Arts and Sciences Minor Research Grant	2002
Research Society on Alcoholism Student Merit/ Junior Investigator Award	2002
The College of William & Mary Student Affairs Conference Travel Award	2001, 2002
Member of Psi Chi (The National Honor Society in Psychology)	2001-2007

SERVICE

Membership in professional organizations

American Psychological Association (APA)	2004-2007
American Psychological Association of Graduate Students (APAGS)	2004-2007
Association for Psychological Science (APS)	2006-2007
International Society for Developmental Psychobiology (ISDP)	2001-2007
International Society on Infant Studies (ISIS)	2003-2007
Research Society on Alcoholism (RSA)	2001-2003
Society for Research in Child Development (SRCD)	2004-2007

Leadership in professional organizations

ISDP	Student Representative on the Executive Committee	2006-2008
APA Division 7	Student Representative on the Executive Committee	2004-2006

Campus and Community Outreach

Morasch, K.C. & Bell, M.A. (October, 2006). Memory Developments in Infancy. Invited lecture given at Radford University, Department of Psychology. Radford, VA.

Whack, A.L., **Morasch, K.C.**, & Bell, M.A. (July, 2006). Cognitive and Emotional Development in Infancy: Relations among Memory, Temperament, and Language. Talk given at the 14th Annual Minority Academic Opportunities Program Symposium, Blacksburg, VA.

Morasch, K.C. & Bell, M.A. (March, 2006). Visual Recognition Memory and Brain-Electrical Activity in 12-month-old Infants. Paper presented at the 21st Annual Graduate Research Symposium, Blacksburg, VA.

Adkins, D.R. & **Morasch, K.C.** (March, 2006). Intelligence is more than a number. Invited talk given to the Blacksburg chapter of Mothers of Preschoolers (MOPS).

Morasch, K.C. & Bell, M.A. (May, 2005). Visual recognition memory and brain electrical activity in 5- & 10-month-old infants. Paper presented at the 50th Anniversary celebration of the Masters of Arts program at the College of William & Mary, Williamsburg, VA.

Colona, K.A. & Hunt, P.S. (January, 2002). The duration of fetal alcohol effects on response habituation. Talk given at the 1st Annual Graduate Research Symposium, Williamsburg, VA.

OTHER PROFESSIONAL ACTIVITIES

Research

- Primary experimenter of the 24-month-old assessment phase of a NIH-funded longitudinal project
- Primary experimenter of the parent assessment phase of a project examining the familial relations among EEG, cognition, and emotion
- Secondary experimenter on several other longitudinal and cross-sectional investigations of cognitive development during infancy, and early and late childhood
- Served as a graduate student liaison between developmental research labs in the Departments of Psychology and Human Development at Virginia Tech
- Organized annual events to show appreciation for longitudinal study participation
- Organized a recruitment booth to expand cross-departmental participant recruitment efforts

Teaching

- Trained two graduate students and 17 undergraduate student researchers in lab procedures, task administration, and maintenance of electrophysiological and behavioral data.
- Aided fellow graduate students in course preparation
- Served as a resource and reference for students applying to graduate programs
- Annually presented for Cave Springs High School Psychology classes visiting Virginia Tech

REFERENCES

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