LORAZEPAM-INDUCED AMNESIA: EXAMINATION OF DELAYED FREE RECALL AND PRIMING EFFECTS

by

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I certify that this thesis contains no material which has been accepted for the award of any other egree or diploma in any university, and that to the best of my knowledge and belief, the thesis contains no copy or paraphrase of material previously published or written by another person, except where due reference is made in the text of the thesis

Mark Stanton

TABLE OF CONTENTS

LORAZEPAM-INDUCED AMNESIA: PRIMING EFFECTS AND DELAYED FREE RECALL

PART A: LITERATURE REVIEW

PART B: JOURNAL ARTICLE

Abstract

Benzodiazepines are a class of drugs that produce a number of side effects including temporary memory impairments which appear to parallel organic amnesia. This raises the possibility that benzodiazepines can be utilised as another method of exploring amnesia and consequently normal memory functioning. This review outlines the current models of memory and amnesia, including the temporary benzodiazepine-induced amnesia. Priming is one memory function that has been shown clearly to be preserved in organic amnesia but not so clearly in benzodiazepine-induced amnesia. This review outlines this research and suggests the need for further research to clarify the exact status of priming in benzodiazepine-induced amnesia.

Benzodiazepines are a class of drugs that produce a range of side effects including temporary and selective memory impairments (Curran, 1986). These temporary effects upon memory appear to be similar to the permanent memory deficits found in organic amnesia (Ghoneim & Mewaldt, 1990). Research into the types of memory impairments in organic amnesia is important in understanding the mechanisms underlying memory itself (Brown, Brown, & Bowes, 1988). Whether or not a similarity exists between benzodiazepine-induced amnesia and organic amnesia impacts on the type of contribution benzodiazepine-induced amnesia can make to our understanding of amnesia and memory (Lister, 1985; Curran, 1991).

This review attempts to outline current understandings of memory and amnesia. This will involve examining the current models of memory, with an emphasis on the widely accepted multistore model and some of its developments. The types of memory impairments found in amnesia and the underlying neuroanatomical substrates will be outlined to further develop an understanding of memory, as will some existing explanations of amnesia . Also, the nature of benzodiazepine-induced amnesia will be explored to determine the degree to which it parallels organic amnesia. One particular area this comparison will focus on is the phenomenon of priming. Priming is the enhanced performance in a processing task because of prior exposure to the information involved in the processing task (Graf & Schacter, 1985). It has been well established that priming is preserved in organic amnesia but the picture is not so clear with benzodiazepine-induced amnesia. Further research into the status of priming in benzodiazepine-induced amnesia could help

identify the relationship between benzodiazepine-induced amnesia and organic amnesia.

An incidental issue that arises from the benzodiazepine-induced amnesia research is the time pattern of delayed recall impairments. Severe impairments in the delayed recall of visual and verbal information has been widely observed in both organic and temporary benzodiazepine-induced amnesia. However, little research has focussed on how soon after the information is presented do the impairments in recall start. This review attempts to outline some of this research and suggests the need for further research to define the time onset of delayed recall impairments.

MODELS OF MEMORY

Psychological research into memory has been dominated by two different theoretical approaches. One approach emphasises the underlying structures involved in memory whilst the other explains memory in terms of the cognitive processes that information undergoes. This section outlines these two approaches and some attempts to develop or synthesise them.

MULTISTORE MODEL OF MEMORY

The structural approach to approach to memory is best represented by Atkinson and Shiffrin's (1968) multistore model. Atkinson and Shiffrin (1968) proposed that information flows through three distinct, but functionally related, structural components. These three memory components are sensory memory, the short-term store, and long-term memory.

Sensory Memory

According to the multistore model, information from the environment enters into a parallel series of transient sensory memory stores (eg: visual, auditory, tactile, olfactory) (Broadbent, 1958). These sensory buffers consist of, at least, two subcomponents (Turvey, 1973). For example, the first component of the visual buffer mediates perception of light and the second component is responsible for the process of identification or pattern recognition and is associated with the secondary visual cortex (Phillips,1974). Pattern recognition occurs when an almost identical somatotopic representation of the environment is analysed for its main features and compared to information stored in long-term memory (Martindale, 1991). A comparable process appears to occur in the auditory sensory buffer (Cowan, 1984). The three main characteristics of sensory memory appear to be that it has a very large capacity (Sperling, 1960), that it has a very short duration of between 50 and 500 ms (Phillips, 1974), and that it is pre-attentive (Baddeley, 1990).

Short-term Store

The short-term store (STS) was conceptualised by Atkinson and Shiffrin (1968) as the executive component of the memory system and that it is related to consciousness. The STS acts as the locus of control because it directs the flow of information within the memory system and performs a number of important functions. A prime function of the STS is to transfer the information from the sensory buffers to the STS itself, before the rapidly decaying information in the sensory memory

buffers is permanently lost. The STS forms part of the wider cognitive system that is responsible for learning, reasoning, and comprehending (Baddeley, 1993). Therefore, another important STS function is to hold information temporarily to assist in the performance of these wide range of cognitive functions. For example, comprehension of a sentence requires holding the first part of a sentence whilst the last part of it is processed. Information is maintained temporarily within the STS by the process of rehearsal, where the information is repeated vocally or subvocally to refresh the decaying trace (Atkinson & Shiffrin, 1968). Rehearsal serves another STS function by mediating the transfer of information from the STS to the long-term memory. Finally, the STS is also responsible for the retrieval of information from long-term memory.

The STS has a limited span of about five to nine items, as demonstrated by the number of digits or words that subjects can repeat immediately after presentation (Miller, 1956). More precisely, STS span is assumed to be limited by the number of "chunks" that can be immediately recalled (Murdoch, 1961). A chunk is an integrated piece of information. An alternative interpretation of STS span is that it is limited by the spoken duration of the items to be recalled (Hitch, Haliday, and Littler, 1984). This suggestion is based on the research of Ellis and Hennelly (1980) who found Welsh-speaking children had a shorter digit span than English-speaking children because Welsh digits took longer to articulate. When the speed of articulation was accounted for, the Welsh-speaking children's digit span was the same as English-speaking children. Further research indicates digit span can be defined as the number of items that can be

articulated in two seconds (Hitch et al., 1984; Hoosain & Salili, 1988).

The most widely used measure of STS storage duration is the Brown-Peterson task which prevents rehearsal, and therefore, the capacity to maintain the information within the STS. Rehearsal is prevented by requiring subjects to perform a filler task, like counting backwards in threes, immediately after presentation. Prevention of active rehearsal can result in marked forgetting of information by as soon as two seconds (Brown, 1958; Peterson & Peterson, 1959; Bjork & Healy, 1974) or as late as 30 seconds (Baddeley & Warrington, 1970), after presentation.

Working Memory model

A simple concept of the STS, as proposed by Atkinson and Shiffrin (1968), is not capable of accounting for some research findings. Perhaps the most significant of these findings is that a simultaneous digit span task does not significantly impair reasoning performance (Baddeley, 1968) or the recency effect in free recall (Baddeley & Hitch, 1977). These findings are not consistent with the concept of a simple STS because dual task performance should exceed the limited capacity of the STS, and therefore, significantly reduce performance. In consequence, Baddeley and Hitch (1974) proposed a multi-component working memory (WM) to supersede the STS, where a central executive supervises and co-ordinates at leasttwo subsystems: an articulatory loop and a visual-spatial scratch pad.

At the core of the multi-component WM is a central executive that acts as an integrator and controller of the slave

subsystems. Baddeley (1990) describes the central executive more as an attentional system than a memory store. This is best illustrated by Norman and Shallice's (1986) supervisory activating system (SAS) model of attention. The model assumes ongoing action and cognitive tasks are controlled or directed by two separate, but interacting, attentional processes. The first type of attentional control is used with well learned skills or tasks, in which repeated practice allows the system to maintain the ongoing activities relatively automatically (eg: driving a car). Therefore, decisions made at this level are also relatively automatic. Norman and Shallice called this contention scheduling. An important characteristic of contention scheduling is that a large number of well learnt activities can be concurrently performed with little interference (eg: talking and driving). The second attentional process functions more as the operation of the will or conscious awareness. Norman and Shallice (1986) termed this process the supervisory activating system (SAS). The SAS functions by interrupting automatic functioning and focussing attentional resources on complex or novel tasks.

The phonological loop is a major WM subsystem and acts as a limited capacity mechanism fundamentally involved in the processing of speech-based information (Baddeley, 1990). The phonological loop consists of a phonological store, where speech-based information is held for one to two seconds before fading, and an articulatory control process which captures the memory traces in the phonological store, refreshes them, and feeds them back into the phonological store. This forms the basis of verbal memory span. The phonological loop contributes to a number of

cognitive activities, including reading, vocabulary, and comprehension (Baddeley, 1983). The concept of a phonological store is capable of explaining acoustic similarity effects. The phonological store encodes acoustically, therefore similar acoustic items will be harder to discriminate in recall (Baddeley, 1983). Also, there is some research that indicates simultaneous presentation of non-meaningful speech-based information and word lists impairs recall of the word lists. For example, concurrent presentation of passages in another language (Colle & Welsh, 1976) or nonsense syllables (Salame & Baddeley, 1989), but not silence or non-speech based noises (eg: music), impair immediate recall performance of word lists. This suggests the existence of a temporary store that holds predominantly acoustic based, but not semantic, information. Another concurrent task that impairs immediate recall of verbally presented information is requiring subjects to repeat vocally or subvocally an irrelevant word. Repetition of an irrelevant word is assumed to occupy the articulatory control process, which reduces the number of words that can enter it (Baddeley, Lewis, & Valler, 1984b). Repetition of irrelevant material appears to affect specifically the phonological loop and not more general attentional processes. Non-speech based concurrent tasks that require equivalent levels of attentional resources as do repeating irrelevant words have little effect on digit span performance (Baddeley et al., 1984b).

The visual-spatial scratch pad is the visual version of the phonological loop, holding and processing visual image representations. Research on this working memory component is not extensive but it is assumed to be responsible for

manipulating visual-spatial images for a variety of discrete visual and spatial functions (Baddeley, 1990).

Long-Term Memory

Long-Term Memory (LTM) is defined as the memory system whose function is to store information, on a relatively permanent basis, once the information can no longer be held within WM (Parkin, 1987). LTM can be divided into three different structures, which though distinct, combine to form a highly interactive system (Tulving, 1986). First is episodic memory which represents an autobiographical record of life events or, as Tulving (1983) describes it, as a memory of personal experiences. Episodic memories include a spatio-temporal context (Ghoneim & Mewaldt, 1990). Second is semantic memory which is the store for general knowledge, concepts, rules and language (Parkin, 1987). Semantic memory differs from episodic memory in that the knowledge in semantic memory exists without a spatial-temporal context or reference to the source of information. For example, people may remember a familiar piece of music without specific reference to a time that they actually heard it. Third is procedural memory and represents the learning of visual, motor, and cognitive skills (Squire, 1986). Procedural memory is revealed by performance and is not necessarily consciously retrievable (Shimamura, 1986).

LTM functioning is dependent upon three processes; consolidation, storage, and retrieval (Ghoneim & Mewaldt, 1990). Consolidation represents the development of a relatively durable and permanent memory trace. This is assumed to involve deeper semantic and associative processing (Tulving, 1986). Storage

refers to the process of maintaining the memory trace in such a form that it will not fade or be replaced. Retrieval is the process by which the memory traces are bought out of storage and applied to some cognitive operation (Baddeley, 1993).

Perhaps a useful theoretical organisation of LTM is one which distinguishes between explicit and implicit memory functions (Graf & Schacter, 1985). Explicit memories are those that can be consciously or explicitly referred to. Explicit memory is revealed when performance of a task requires conscious recollection of a previous learning experience (Schacter, 1987). In contrast, implicit memory is revealed when previous experience, or learning, facilitates performance of a task but does not require conscious recollection of those previous learning experiences (Graf, Shimamura, & Squire, 1985). Explicit memory functioning is mediated by episodic and semantic memory whereas implicit memory functioning is mediated by procedural memory (Ghoneim & Mewaldt, 1990). Cohen and Squire (1980) suggest this division of LTM represents the crucial distinction between knowing how (implicit) and knowing that (explicit).

Evidence for a multistore model

A considerable amount of evidence supporting the argument for a structurally separate WM appears to arise from the fact that some memory tasks consist of two components that behave in different ways. Perhaps the best example of this is the free recall task (Baddeley & Warrington, 1970; Baddeley, 1990). In the free recall task subjects are presented with a list of words and are requested to recall these words immediately in any order.

The probability of the words being recalled is a function of its position in the list. Words in the beginning of the list have a greater probability of being recalled than those in the middle and those at the end have the greatest probability of recall (Glanzer & Cunitz, 1966). This is known as the serial position curve, with the greater recall of words at the beginning of the list termed the primacy effect and the greatest recall of words at the end of the list titled the recency effect (Atkinson & Shiffrin, 1968; Baddeley & Warrington, 1970). Cognitive tasks demonstrate differential effects upon the primacy and recency effect. For example, the recency effect disappears but the primacy effect remains if subjects are required to perform the Brown-Peterson task (Glanzer & Cunitz, 1966). Alternatively, reduction of the primacy effect is achieved by altering the speed of presentation (Glanzer, 1972), performance of a concurrent distracting task (Murdoch, 1965), presenting nonsense words (Glanzer, 1972), or presenting abstract words (Baddeley, 1990), whereas the recency part of the curve remains unaffected. There even appears to be a difference in processing time as subjects are faster in their responses for words at the end of the serial position than those at the beginning (Waugh, 1970).

Another line of argument for a structurally separate WM is based on findings that suggest WM encodes the acoustic characteristics of information and LTM encodes the semantic elements of the information. Encoding refers to the process by which information is retained within the stores (Atkinson & Shiffrin, 1968). Evidence of acoustic encoding in WM is found with studies that demonstrate WM encoding errors are of an acoustic nature. For example, subjects are more likely to make

errors in recalling consonants that are acoustically similar (eg: substituting P for V) with an immediate recall task (Conrad, 1960). Similarly, subjects are more likely to recall inaccurately acoustically similar sequences of consonants than acoustically dissimilar sequences of consonants (Conrad & Hull, 1964) or acoustically similar words than semantically similar words (Baddeley, 1966), when the number of items to recall are within WM span. In contrast, the encoding of information into LTM appears to be more semantically based. Subjects recall less semantically similar words than semantically dissimilar words, when there is a delay in recall and the word lists are more than 10 items (Baddeley, 1966). Baddeley (1990) suggests acoustic characteristics are no longer important in long-term learning and they are discarded. Only the meaning of the information is retained.

Furthermore, the fact that encoding produces differential effects upon memory tasks also suggests different memory structures are involved. In particular, acoustic encoding affects the ST component of the task and semantic encoding affects the LT component. For example, semantic similarity in the items reduces the primacy effect whereas acoustic similarity reduces the recency effect in free recall (Kintch & Buschke, 1969). In another example, Sachs (1967) presented prose passages to subjects and requested they identify any changes in sentences that were repeated. Subjects were very good at noticing both syntactic and semantic changes when tested immediately. However with a delay subjects semantic recall remained good but recall of the syntactic changes significantly decreased.

The types of memory impairments that brain-injured patients reveal suggests a double-disassociation between WM and LTM (Schacter, 1986). One group of patients, whose brain damage is generally associated with the medial temporal area, demonstrate normal WM functioning but are greatly impaired in long-term recall of new information (Milner, 1956; Baddeley & Warrington, 1970; Squire & Shimamura, 1985). With these patients, the recency effect is intact, digit span is normal, and they perform well on the Brown-Peterson task. However, these amnesic patients demonstrate a severely reduced primacy effect and are impaired in the delayed recall of word lists. In contrast, another group of patients, with damage more associated with left hemisphere functions, appears to have normal long-term learning, but severely impaired immediate recall (Shallice & Warrington, 1970; Baddeley, 1983). It is assumed the first group of patients represent, what is commonly thought to be, amnesia and the second, and much rarer group, experience another type of acquired brain-injury.

LEVELS OF PROCESSING APPROACH

An alternative theoretical framework was developed by Craik and Lockhart (1972). Rather than information flowing through structurally separate memory stores, Craik and Lockhart (1972) argued information proceeds through a hierarchy of stages, involving an increase in the depth to which the information is processed. Depth is defined by the degree of semantic or cognitive analysis the information undergoes. Information processed by its superficial sensory characteristics results in short-lived traces. Phonological processing results in

slightly more durable encoding,= and deeper level processing, involving encoding of semantic qualities, results in a "more elaborate, longer lasting and stronger trace" (Craik & Lockhart, 1972). The important characteristic of the levels of processing approach its emphasis on these processing stages existing as a continuum of analysis, not as structurally separate components.

An inadequacy of the multistore model is that it offers too simplistic an explanation of long-term learning. The multistore model argues that new information enters LTM via the STS and rehearsal is the process responsible for this (Atkinson & Shiffrin, 1968). Therefore, brain-injured patients with impaired WM (or STS) should have great difficulty learning. However this has not been empirically supported (Baddeley & Wilson, 1988). Furthermore, normal subjects can maintain information in WM but it does not necessarily encourage transfer to LTM (Tulving, 1966). Craik and Lockhart (1972), alternatively, suggest memory traces are the by-product of the perceptual processes the information undergoes, with deeper processing enriching the traces with semantic and associative characteristics which results in better learning. Support for this proposition can be found in incidental learning tasks, where subjects perform orienting tasks that produce different levels of processing without the expectation of recall. Processing information for its physical characteristics (eg: is the word printed in capitals) results in poor recognition but semantic processing (eg: whether the word is the name of an animal) results in extremely good recognition (Craik & Tulving, 1975). Interestingly, semantic processing requires significantly more response time for

recognition than physical processing suggesting a deeper form of processing (Craik & Lockhart, 1972).

A synthesis of the two approaches would assume there are two separate rehearsal processes (Baddeley, 1993). Information can be maintained within WM for relatively brief periods of time, by refreshing the decaying trace. Refreshing a memory trace involves continued processing of the information at a shallow depth, which only temporarily maintains the information in WM and does not lead to long-term learning. Once the process is terminated the information is lost. A second more elaborative rehearsal process involves an increase in the depth of processing and, therefore, an enrichment with semantic and associative characteristics. This results in a more permanent trace. Baddeley (1990) suggests the levels of processing approach to rehearsal primarily relates to the manner in which long-term learning occurs and, as such, represents a complementary position to the multistore model. This synthesis is consistent with the serial position curve. The primacy effect results from elaborative (deeper semantic) rehearsal, as the subject is aware future items are to come (Craik & Lockhart, 1972). The final words require only acoustic processing to be maintained temporarily for recall and, therefore, receive only maintenance rehearsal (Baddeley, 1983).

It is also clear that the multistore model's neat separation of WM and LTM, on the basis of the type of encoding, is too simplistic. It has been found WM can encode semantic and visual characteristics (Schulman, 1974). For example, Baddeley and Levy (1971) found semantic associate pairs were better recalled than semantically dissimilar pairs in an immediate recall task.

Baddeley (1993) summarises the current status of acoustic and semantic encoding by suggesting subjects will encode semantically if they are able but if time or the information does not allow this, then the subjects will encode acoustically.

AMNESIA AND MEMORY

Research on amnesic patients contributes to current concepts of memory functioning in three important ways. First, it offers the possibility of identifying the neuroanatomical correlates of proposed memory functions. Second, identifying the memory functions that are preserved or impaired in amnesia provides further opportunity to identify normal memory functions. Third, attempts to explain amnesia with cognitive models contribute to the theoretical explanations of normal memory functioning. The increased understanding of memory functioning from amnesia research reciprocally develops concepts of amnesia.

NEUROANATOMICAL STRUCTURES UNDERLYING MEMORY

Research into amnesia provides a useful methodology to identify the neuroanatomical substrates underlying normal memory functioning (Squire, 1982). Currently, the neuroanatomical structures that mediate memory functions have not been clearly defined (Tulving, 1985). A major difficulty is the variety of etiologies for amnesia (Parkin, 1992). Some of the most common causes of amnesia are Korsakoff's syndrome, viral infections that invade the brain, anoxia, closed head injuries, and neurosurgery (Lezak, 1983). Amnesia from different origins does

result in damage to a similar set of brain structures but there are some differences and these differences have not been outlined well (Mayes, 1992). For example, Korsakoff's syndrome, a product of a thiamine (vitamin B1) deficiency (usually caused by excessive long-term alcohol consumption and inadequate diet), appears to involve lesions in the mamillary bodies, nuclei of the thalamus, and the dorsolateral part of the frontal cortex (Lezak, 1983; Levin, 1986). In contrast amnesia caused by viral infections that invade the brain can often result in damage to the medial temporal area, hippocampal formation, the amygdala, and orbito-frontal part of the frontal lobe (Squire, 1986). These viral infections includes herpes simplex encephalitis, tuberculosis meningitis, and neurosyphilis (Lezak, 1983).

Furthermore, few patients experience only discrete lesions to well identified memory structures. In particular, many amnesics experience damage to the prefrontal cortex, which adversely affects many other cognitive functions as well as memory (Mayes, 1992). Amnesic patients also vary in the severity of the memory impairments and there is no generally accepted method of measuring severity (Hirst, 1982). It is not clear to what degree the severity of amnesia represents a qualitative impairment to distinct structures or is a quantitative measure of the combined damage to all, or some, of these structures (Parkin, 1992).

In spite of these difficulties, there is general agreement about some of the structures that are involved in memory and a rudimentary understanding of their role (Moscovitch, 1992). Cortical modules are the neural structures underlying the first subcomponent of sensory memory. These modules within the

neocortex receive domain-specific environmental information and process it at a shallow presemantic level (Fodor, 1985). 'Central systems' act on the output of these modules to attach meaning and significance to the somatotopic representation of the environment (Moscovitch, 1992), that is, the central systems mediate the process of pattern recognition. Both these processes are assumed to be located in the posterior and midlateral neocortex areas which are responsible for perceptual analysis and identification (Squire, 1986). This information is attended to by the WM and results in deliverance of this information into the hippocampus and related limbic and medial temporal structures (Mayes, 1988). This formation of structures binds this information to form a memory trace within the neocortex. At the same time, it also produces an index of the trace within the hippocampus. The frontal cortex appears to supply the contextual component to the memory trace (Moscovitch, 1992). Contextual memory can be defined as the global information about when and where specific information was obtained and the temporal relationship of one episode to another (Schacter, 1987).

Amnesic patients appear to experience damage to a common set of neuro-anatomical structures. In particular, amnesia involves damage to the neural circuit connecting the medial temporal area, frontal lobes, hippocampus, mamillary bodies, thalamus and hypothalamus (Squire, 1986). It has been argued that damage to the diencephalon and to the medial-temporal area represents two separate forms of amnesia (Squire, 1986; Mayes, 1988) Medial-temporal damage is believed to cause more rapid forgetting but less impaired contextual memory. This is frequently found in patients who have contracted herpes simplex

encephalitis. Diencephalon damage may result in less rapid forgetting but more impaired contextual memory. This type of damage is commonly found with Korsakoff patients.

The concept of a single amnesia is further challenged by evidence of marked yet specific deficits in contextual memory resulting from frontal lobe damage. Several studies have found amnesics with impaired frontal lobe functioning, as demonstrated by poor performance on the Wisconsin card sorting task and Benson word fluency test, exhibit poor contextual memory relative to recall and recognition (Huppert & Piercy, 1978; Squire, 1982a; Meudell, Mayes, Ostergaard, & Pickering, 1985). Poor contextual memory relative to recognition or recall is indicated by amnesics who showed comparable performance to controls on recognition tests but were impaired in identifying where and when they received the information. This is illustrated by Squire (1982) who presented two word lists to subjects followed by a recognition task and instructions to identify which of the lists a recognised word came from. Korsakoff patients were significantly impaired in discriminating between which of two presentation lists particular words came from compared to normal controls, even though both groups were equivalent in recognition. A strong correlation exists between performance on tasks sensitive to frontal damage and list presentation discrimination but not with recognition performance (Squire, 1982; Schacter, Harbluck, & McLaclan, 1984).

PRESERVED AND IMPAIRED MEMORY FUNCTIONS

In most forms of amnesia WM appears to remain unaffected. Preserved WM in amnesic patients has been demonstrated by normal digit span (Milner, 1971), normal recency effect, and normal performance on the Brown-Peterson task (Baddeley & Warrington, 1970). In some cases, amnesic patients can have above average WM performance (Baddeley, 1990). In contrast, amnesic patients recall significantly less of the first presented words than normals in the free recall task, suggesting some form of impairment to LTM functioning (Baddeley & Warrington, 1970). LTM is not impaired as a whole, rather, it is specific components within LTM that are affected. Reduced primacy effect on the free recall task represents an impairment to episodic memory (Walsh, 1987). Further suggestions of impaired episodic memory is found with amnesic patients whose autobiographical memory for life events before the onset of amnesia is well preserved but recall of events after amnesia onset are severely impaired (Walsh, 1987). Unimpaired language ability and general intellectual functioning in amnesic patients both suggest intact semantic memory (Hirst, Johnson, Phelps, & Volpe, 1988).

Procedural memory is also spared in amnesia. A large body of research suggests amnesic patients are capable of learning a number of motor, visual, and cognitive skilled tasks. The best known example is the pursuit rotor task. The pursuit rotor task is a hand-eye co-ordination test that involves following a dot on a revolving disk with a stylus (Weiskrantz & Warrington, 1979). Similar learning is demonstrated in mirror drawing tasks (Brooks & Baddeley,1976), in the Tower of Hanoi puzzle (Cohen, 1984) and in complex puzzle tasks (Baddeley & Wilson, 1988). The unique

feature of amnesic patients' performance in these tasks is that they demonstrate normal learning of skilled tasks in spite of an inability to recall the learning experiences. Moscovitch (1984) claims all these tasks have a number of common elements. The tasks are structured and, therefore, it is easy to understand the requirements of the task. The behaviours required to perform the task are ones which the amnesic patients have already demonstrated (eg., moving blocks, assembling a puzzle). Most importantly, the tasks can be achieved without explicitly having to refer to a past experience.

COGNITIVE EXPLANATIONS OF AMNESIA

A number of theories have been offered in an attempt to explain what has been termed a 'core' amnesic syndrome. These explanations are in accordance with related theories of general memory function. This next section outlines three of the most noted theories.

Consolidation

One of the earliest theories of amnesia derived from the memory deficits that werenoted with patient H.M., who had received a bilateral removal of medial temporal lobes, hippocampus, and amygdala (Milner, 1971). H.M.'s inability to acquire new information was explained as deficits in transferring information from WM and consolidating it as a stable, more permanent, trace within LTM. Milner (1956) identified the hippocampus as the vital structure responsible for consolidation. However, the consolidation hypothesis has been strongly criticised for its inability to account for the effects of

increased recall through retrieval cues, good performance on recognition tasks, and proactive interference (Stern, 1981; Baddeley, 1990). Also, it offers no explanation of intact perceptual and procedural skills in amnesics (Hirst, 1982).

Encoding

An alternative hypothesis, based on the levels of processing approach, proposes amnesics do not spontaneously encode information semantically and thus fail in the deeper level processing necessary for adequate learning (Cermak, Butters, & Gerrin,1973; Cermak & Reale, 1978). Impaired semantic processing by Korsakoff patients may represent supporting evidence for this proposition. This is illustrated by studies where Korsakoff patients were impaired at detecting successive words from the same semantic category but not words that rhyme or are repeated (Cermak et al., 1973; Cermak & Morienes, 1976). These results appear to indicate amnesic patients encode information based on physical or acoustic characteristics. It is believed encoding at superficial levels is more susceptible to interference effects that impair new learning (Cermak, Butters, & Morienes, 1974). Further support arises from the fact that Korsakoff patients show no levels of processing effects (Cermak & Reale, 1978). There are further suggestions that Korsakoff patients cannot utilise other semantic processes. For example, Cermak et al. (1974) found that Korsakoff patients can only obtain release from proactive interference using shifts in superficial features of the information, whereas normal subjects can obtain release shifts in both superficial and semantic features. However, Meudell, Mayes, and Neary (1980) found

increased learning with deeper processing of drawings and cartoons.

Retrieval

Interference theory assumes information is encoded in LTM but that increased competition of information results in a failure to retrieve the accurate item. Initial evidence suggested that cuing techniques (priming) assisted in reducing interference effects and, therefore, increased learning (Warrington & Weiskrantz, 1968; 1970).

A more sophisticated interference framework attributes memory dysfunction to insufficient association of the material with cues necessary for subsequent retrieval. The information has insufficient cues to separate it from other information when retrieval is required (Mayes, 1992). Evidence for this is found with amnesic patients who are comparable to normals in recognising previously presented pictures but are significantly impaired at identifying when it was presented (Huppert & Piercy, 1976). Also, amnesics tend to be impaired more in their capacity to remember where they acquired new information than what the subject matter was (Schacter et al., 1984; Shimamura & Squire, 1987). This point is well illustrated in the experiment of Huppert and Piercy (1978) who found normal controls were more likely to correctly identify the time of presentation of pictures as the previous day if the pictures had been presented twice. On the other hand Korsakoff patients were more likely to mistakenly assert the pictures had been presented that day if they had been presented twice on the previous day. It appears Korsakoff patients rely more on the general strength of the trace

and normal controls increase the strength of the trace by directly encoding temporal information (Meudell et al., 1985). Baddeley (1991;1993) develops the concept of contextual processing further to suggest that amnesics may in fact lack the ability to create cognitive links between two separate bits of information. In particular, the deficits in amnesia extend to difficulties in organising connections between the new items of information, other new information, and old, already encoded, information within LTM.

BENZODIAZEPINE-INDUCED AMNESIA

Benzodiazepines are a class of minor tranquillisers used extensively for their hypnotic, anti-anxiety, and muscle relaxant effects (Kanto, 1985; Curran, 1986). In addition, benzodiazepines produce specific, although temporary, memory impairments that appear to parallel the permanent memory deficits found in organic amnesia (Brown, Brown, Horn, Lewis, & Bowes, 1982). If benzodiazepine-induced amnesia has many properties similar to the organic amnesia syndrome, then it represents an invaluable methodology to explore amnesia and memory structures. Benzodiazepines could be used to explore and test both the types of memory impairment experienced in amnesia, and, various models of amnesia and memory functioning (Danion, Weigertner, File, Jafard, Sunderland, Tulving, & Warburton, 1993). Brown, Brown, and Bowes (1989) suggest that benzodiazepine-induced amnesia offers some specific advantages in the study of amnesia and memory functioning. For example, it allows the opportunity to experimentally manipulate a number of variables to a degree

not possible with the organic amnesia syndrome. These variables include testing pre-amnesia performance, matching subject variables (eg: age, sex, education, IQ), and experimental design.

NEUROBIOLOGICAL EFFECTS OF BENZODIAZEPINES

Benzodiazepines act directly upon benzodiazepine specific receptor sites. Although benzodiazepine receptor sites have been identified through-out the body, it is the receptor sites in the central nervous system upon which benzodiazepines have their greatest effect (File, 1988). The highest density of benzodiazepine receptors are located in the CNS, especially in the cortex, limbic structures, thalamus and hypothalamus, and possess pharmacologically distinct properties compared to benzodiazepine receptors located elsewhere (Mohler & Okada, 1977; Greenblatt, Shader, & Abernethy, 1983).

Benzodiazepines enhance the inhibition effect of the neurotransmitter GABA (gamma-aminobutyric acid). GABA is a major inhibitory neurotransmitter that acts by opening neuronal membrane to chlorine ions. Chlorine ions, when allowed to enter neurones, alter the electrical potential in such a way as to make it more difficult for the neuron to excite. GABA and benzodiazepine receptors possess similar pharmacological characteristics. Consequently, benzodiazepines can potentiate the binding of GABA to neuronal membranes and, conversely, GABA can potentiate the binding of benzodiazepines. The presence of benzodiazepine enhances the inhibitory action of GABA and, thereby, benzodiazepine mediates neuronal inhibition. Enhancing the inhibitory action of GABA may then produce a feedback mechanism that potentiates the binding of

benzodiazepine to its receptor sites (Dundee & Haslett, 1970; Greeblatt et. al., 1983). Some attempts have been made to neurochemically isolate the specific effects of benzodiazepines. For example, two types of benzodiazepine specific receptor sites have been identified (Braestrup & Nielson, 1980). Type 1 receptors are found extensively throughout the brain and are thought to be associated with the anxiolytic effects of benzodiazepines. Type 2 receptors are more localised in the limbic area and are thought to be more associated with the sedation effects of benzodiazepines (Davies, 1985).

AMNESTIC PROPERTIES OF BENZODIAZEPINES

Benzodiazepine administration appears to produce selective memory impairments that are similar to those in organic amnesia. WM, measured by performance on digit span test (Brown et al., 1982), Brown- Peterson task (Baddeley & Wilson, 1988), and free recall recency effect (Wilson & Baddeley, 1988), is unimpaired. In contrast, long-term episodic memory for newly presented information, measured by performance on delayed free recall, recognition, and cued recall tasks, is severely impaired (Ghoneim & Mewaldt, 1975; Subhan, 1984). Deficits in delayed recall and recognition have been found with visual stimuli (Dundee & Wilson, 1986; Miller, Bullard, & Patrissi, 1989) and verbal information (File & Lister, 1982; Borbely, Schlapfer, & Trachsel, 1988). However, benzodiazepines do not impair recall or recognition for material presented prior to drug administration (Brown et al., 1983). In fact, in some cases benzodiazepines may facilitate retrograde retrieval of word lists (Hinrichs, Ghoneim, & Mewaldt, 1984). Also

benzodiazepines do not appear to impair semantic memory, as indicated by normal performance on verbal fluency tests and procedural memory, as indicated by preserved learning of viusal-motor skills (Brown et al., 1983)

PHARMODYNAMIC EFFECTS OF BENZODIAZEPINES

A large variety of benzodiazepines are currently in medical use. Virtually all benzodiazepines produce anxiolytic, sedative, and anticonvulsant effects and have been associated with temporary amnesic effects (Greenblatt et al., 1983; Ghoneim & Mewaldt, 1990). There are, however, substantial differences in the duration and potency of the effects produced by these derivatives (Dundee & Haslett, 1970). A number of factors mediate these differences in amnesic effects. It appears the factor most clearly related to duration and degree of amnesic effects is dose (Ghoneim & Mewaldt, 1984). Dose-related deficits have been observed with lorazepam (Preston, Brooks, Traub, Ward, Poppleton, & Stahl, 1988), diazepam (Kothary, Brown, Pandit, Samra, & Pandit, 1981), and midazolam (O'Boyle, Harris, Barry, McCreary, bewly, & Fox, 1989). Increases in dose tend to increase mainly the duration of effects with diazepam and lorazepam but to increase the magnitude of effects with midazolam (O'Boyle, 1988; Ghoneim & Mewaldt, 1990). Route of benzodiazepine administration also clearly determines the degree and duration of amnesic effects. The most rapid, strong, and durable effects are observed with intravenous administration. In descending order, slower and less intense effects occur with intramuscular, subcutaneous, and oral administration (Ghoneim & Mewaldt, 1990). Weight and age are

two important subject characteristics that influence onset, intensity, and duration of effects (Curran, 1986). Increased age and decreased weight produce greater sensitivity to benzodiazepines.

The benzodiazepine derivatives possess different rates of absorption, distribution, and elimination (Ghoneim & Mewaldt, 1984). This results in different onset, intensity, and duration of effects. With oral administration, the rate of absorption is determined by the gastrointestinal tract and the lipid solubility of the benzodiazepine. Therefore, highly lipid soluble benzodiazepines like diazepam and midazolam have a rapid onset but relatively short duration. Low midazolam and diazepam doses administered intravenously, produce amnesic effect within two minutes, peaking within two-five minutes, and disappearing within 20-40 minutes (Dundee & Wilson, 1980. Oral administration delays the onset time. In contrast, less lipid soluble benzodiazepines, like lorazepam and oxazepam, have a later onset but longer lasting effects. Lorazepam starts demonstrating amnesic effects within 20-30 minutes, peaking for two hours, but lasting for six to eight hours (Preston et al, 1988).

PRIMING EFFECTS

Priming is enhanced or changed performance in a processing task because of prior exposure to the information involved in the processing task (Tulving, Schacter, & Stark, 1982; Squire, Shimamura, & Squire, 1987). Priming has been demonstrated in a wide variety of processing tasks. One of

these is the lexical decision task, where subjects are required to decide if presented strings of letters are real or nonwords. Priming is demonstrated by decreased response times in the lexical decision making for previously presented strings of letters compared to new strings of letters (Mckoon & Ratcliff, 1979; Durgunoglu & Neely, 1985). Another processing task is word identification, which involves identifying tachistoscopically presented words for very brief periods (300ms). Priming is reflected by increased accuracy in identifying previously presented words in an unrelated orienting task compared to new words (Jacoby & Dallas, 1981). Perhaps the most common priming task is word completion. This involves completing a word from a fragment (eg: A_c__u_t_n_ = Accountant) or stem (eg: mot = mother) with the first word that comes to mind. Priming is indicated by the greater completion of fragments or stems that form previously presented words compared to fragments or stems that form new words (Graf, Squire, & Mandler, 1984; Slomon, Hayman, Ohto, Law, & Tulving, 1988).

CHARACTERISTICS OF PRIMING

A considerable amount of research has attempted to outline the nature of the priming effects. Priming has been widely demonstrated with simple pre-existing memory representations such as words (Graf et al., 1984). Priming effects may not to be restricted to simple representation but also occurs with pre-existing semantic associations between words. Traditionally semantic associations have been examined by presenting subjects with semantically related word pairs (table-chair) or

semantically associated word pairs (sour-grape). In these instances priming has been demonstrated by the greater tendency for subjects to complete successfully word stems (sour-gr.....) that form previously presented word pairs than word stems that form previously unpresented word pairs (Shimamura & Squire, 1984: Graf & Schacter, 1985). Priming effects may even extend to situations where different but semantically related words are used in the orienting task and word completion task (Shimamura & Squire, 1984). For example the word *child* may produce priming in a word stem that could form the word *baby.*

One explanation of priming effects with semantic associations is framed within a semantic network model (Collins & Loftus, 1975). The semantic network model states that each familiar concept is represented as a node within a semantic network. The properties that define that node represent the links to other related nodes within the network. For example the concept of *Dog* would exist in semantic network for *Animals* and would be linked to the concept of *Cat* by the properties they share, such as fur, tail, pet. Therefore, the model assumes that accessing one node within the framework results in activation not only for that node but also nodes linked by shared defining properties. Lupker (1984) suggests the semantic network model alone cannot explain priming effects with semantic associations. Rather, the major factor involved in explaining priming effects is the degree of direct association between separate representations. Direct association represents the linking of separate representations on the basis of episodic pairing rather than shared semantic properties.

New semantic associations are formed when previously unrelated or unassociated pre-existing representations are paired together (Schacter, 1986). In priming studies the most common method of creating new semantic associations is by presenting unrelated words (grape-poor) in a paired associate task. Priming has been demonstrated with new semantic associations (Graf & Schacter, 1986; Shimamura & Squire, 1989).

Priming effects may extend beyond the modality in which the information is initially processed and occur with completion tasks in another modality. Graf, Shimamura, and Squire (1985) found both visually and verbally presented information in a semantic processing task produced priming effects in a written word stem completion task. Similar findings of priming with modality shifts have been demonstrated with a word fragment completion task (Roediger & Blaxton, 1987). These results contrast with suggestions that priming is modality specific (Clark & Morton, 1983). It should be noted, however, that greater priming effects are demonstrated with processing and word completion occurring within the same modality. This may suggest priming is mediated by a set of related sensory processes which are responsible not only for priming but also for the transfer of information across modalities.

Currently, there is little agreement concerning duration of priming effects. There is some evidence to suggest priming effects are transient, disappearing in two hours (Graf et al., 1984; Shimamura & Squire, 1984). However, these findings contrast with other research which has detected priming effects after seven days (Tulving et al., 1982). Duration of priming may be dependent, at least in part, upon the type of processing task

utilised. Some lexical decision making (Scarborough, Cortese, and Scarborough, 1977) and word identification tasks (Jacoby & Dallas, 1981) have demonstrated relatively long priming effects. In contrast, word stem completion performance has declined within ten minutes and has returned to guessing levels within two hours (Graf & Mandler, 1984; Graf et al., 1984). Unfortunately, no systematic attempt has been made to delineate functional differences between these tasks and how these differences may explain the observed variability in priming effect duration (Shimamura, 1987).

It is clear, however, that priming effect duration is not completely task-dependent. Findings of considerable variability in priming effect duration with different word completion task studies complicate explanations based upon functional differences amongst tasks. This variability is demonstrated by a number of studies which found priming effects had disappeared within two hours (Graf et al., 1984: Graf & Schacter, 1985) whereas other studies found some degree of priming after a week and after 12 months (Slomon et al., 1988). Perhaps, the only relatively consistent finding is priming effects showing some degree of reduction within 10 minutes (Graf et al., 1985).

Shimamura (1986) suggests that the conflicting data with word completion tasks may arise from the type of words used. Word completion tasks that have resulted in more transient priming effects have utilised words whose stem could form a number of other words (Graf et al., 1984; Schacter, 1985). A number of studies reporting longer-lasting priming effects have used words whose stem or fragment could form only one word (Tulving et al., 1982; Sloman et al., 1988). Shimamura (1986)

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notes that it is easier for subjects to complete stems or fragments which form many words with the first word that comes to mind than it is to complete stems or fragments which form only one word. Therefore, when subjects are not able to complete stems or fragments quickly they access explicit memory to recall the previously presented words. Consequently, word completion of stems or fragments that form only one word may represent explicit memory facilitation and, therefore, increased duration. Significantly, Squire et al. (1987) found semantic processing of words whose stem could form only one word resulted in more priming after four days than nonsemantic processing. Improved priming as the product of elaborative processing may suggest the involvement of explicit memory.

Explicit memory facilitation of priming with word specific primes may help explain some of the variable findings of priming effect duration . On the other hand, evidence of long-lasting priming effects with amnesic patients suggests the need for further explanation. For example, the brain-injured patient K.C. who demonstrated virtually no explicit memory functioning, was able to demonstrate priming effects for 12 months (Tulving, Hayman, & macDonald, 1991). Furthermore, there is some suggestion that amnesics do not utilise explicit memory functions in priming. This is based on findings that amnesics show impaired priming when the primes are processed semantically (Graf et al., 1984; Graf & Schacter, 1985). Further studies are required to determine the influences on the duration of priming effects.

PRIMING AND EXPLICIT MEMORY

Research has also focused on attempts to identify priming as an implicit memory function and the implications priming may have for an implicit and explicit memory distinction. Evidence for such a distinction comes from both neuropsychological examination of amnesics and research with normal subjects.

Amnesic patients

Preserved memory functions in amnesia suggest that these preserved memory functions are dissociable from those that are in fact impaired (Baddeley, 1991). Perhaps the most significant source of evidence for a dissociation between priming and explicit memory is differential preservation of these types of memory in amnesics. Amnesics are impaired significantly on traditional tests of explicit memory, such as free recall, cued recall, and recognition tasks (Cohen, 1981; Moscovitch, 1982; Paller, 1990). However, in a substantial number of studies amnesic patients demonstrate preserved priming effects (Graf et al., 1984; Squire et al., 1987; Tulving, 1991).

More specific evidence for a dissociation of priming and explicit memory in amnesia is found when word completion test instructions are manipulated deliberately to tap either explicit or implicit memory. Amnesic patients demonstrate preserved priming effects when they are provided with the implicit instructions "to write the first word that comes to mind" but priming effects are impaired when subjects are provided with the explicit instructions "to use the stems as cues to remember the recently presented words" Graf et al. (1984). The effect of implicit and explicit instructions upon priming effects can also

be illustrated clearly by comparing the performance of amnesic patients and normal subjects with paired-associate tasks. Amnesic patients demonstrate comparable priming effects to control subjects when provided with implicit instructions but only amnesic patients' performance is reduced with explicit instructions (Shimamura & Squire, 1984; Graf & Schacter, 1985). Impairment of explicit memory functioning only with amnesic patients is consistent with the idea of two separate memory processes that are affected differentially by the cognitive demands placed upon them.

Normal Subjects

Some studies have attempted to demonstrate a dissociation between priming and explicit memory by exploring the differential effects of a variety of cognitive variables upon priming and explicit memory. One of these variables is manipulation of the level of processing involved in the original processing task. More elaborative processing of the priming information increases explicit memory performance, as measured by recognition or recall, but does not influence the level of priming in word completion tasks or completion of common idioms (Jacoby & Dallas, 1981). Graf and Mandler (1984) directly demonstrated the differential effect of processing depth upon implicit and explicit memory by manipulating the types of instructions given to subjects. Elaborative processing improved significantly word completion performance when subjects were instructed explicitly to complete the word stems from the previously presented words but elaborative processing did not improve performance when subjects were provided with implicit

instructions to complete the word stems with the first word that came to mind.

Also, cross-modality priming effects may represent support for an explicit memory and priming dissociation. Whilst priming effects are detected when there is a shift in modality from the processing task to the word completion task, they are not as substantial as those found when there was no shift in modality (Graf et al., 1985). In contrast, such a modality shift does not produce a similar reduction in free recall (Jacoby & Dallas, 1981; Graf et al., 1985). Some have concluded from these results that priming is mediated by a different set of related sensory processes from that of explicit memory (Graf et al., 1985; Schacter, 1986).

Proactive interference describes the situation in which previously presented information disrupts the learning of newly related information. For example, learning an association between A-B may interfere with new learning of an association between A-C. Historically, proactive interference has been demonstrated with explicit memory functions such as recall (Postman & Underwood, 1973). However, Graf and Schacter (1987) suggest there is a dissociation between priming and explicit memory with findings of interference effects with a cued recall task but not with a word stem completion task. Graf and Schacter (1987) explain differential proactive interference effects in terms of the different performance requirements of the tasks. Explicit memory tasks require subjects to access specific previously presented words. As such, memory performance depends upon the ability to distinguish between items within the presented list. The distinctiveness of each

item is lowered under conditions of proactive interference. In contrast priming tasks require subjects to respond with the first word that comes to mind and, consequently, does not depend upon distinguishing between items in the previously presented list. Priming performance is not impaired, therefore, as subjects only have to relate the two newly associated words.

Some researchers have attempted to demonstrate a dissociation between priming and explicit memory with evidence of differential duration of priming and recognition or recall. For example, Jacoby and Dallas (1981) detected word identification priming effects but significantly reduced recognition after a week. Another example is Tulving, Schacter, and Stark (1982) who found significantly diminished recognition after seven days but relatively unchanged word completion priming effects. However, attempts to argue for a distinction between priming and explicit memory based on differential durations are complicated by findings of persistent recognition but no priming effects by two hours (Graf & Mandler, 1984; Graf et al., 1984). These results suggest a dissociation between priming and explicit memory but in the opposite direction. No systematic framework has been provided to explain how the variable direction of these differential duration effects in fact supports a distinction between priming and explicit memory. The situation is complicated further by uncertainty as to the duration of priming effects and the factors that influence duration (Shimamura, 1986).

EXPLANATIONS OF PRIMING EFFECTS

Theoretical explanations of priming effects have focused on attempts to account for the characteristics of priming and explain how priming operates as an implicit memory function. Currently no single model achieves this satisfactorily. This section outlines three approaches that are capable of accounting at least for some of the data.

Multiple Memory Systems

Implicit memory, therefore priming, and explicit memory functions are explained as the product of different underlying memory structures. This conceptualisation is compatible with Tulving's (1983) distinction of episodic and semantic memory structures. Episodic memory is considered the structure underlying explicit memory functions and semantic memory the structure underlying implicit memory functions. Another variation of this approach is Cohen's (1984) declarative and procedural memory distinction. Here it is the declarative memory structure which mediates explicit memory functions and the procedural memory structure which mediates implicit memory functions. The neatness of the model's explanation of priming effects stands as its main strength. The model's account for impaired explicit memory and preserved implicit memory in amnesics as the product of different neuroanatomical structures (Schacter, 1987). This also represents its failing as it does not provide any detailed account for a variety of findings. For example, it provides no explanation of differential duration of priming effects and explicit memory, or, priming with new associations.

Processing Model

An alternative approach explains implicit and explicit memory as the result of the relationship between encoding and the type of retrieval process utilised (Schacter, 1987). Both implicit and explicit memory are the product of encoded episodic representations. Therefore, both implicit and explicit memory for newly presented information are mediated by the same episodic memory representation (Jacoby & Witherspoon, 1982). However, the difference is that explicit memory reflects the processes of elaboration, organisation, and reconstruction, whereas implicit memory reflects other processes that are determined by the needs of the data (Jacoby & Dallas, 1981). Implicit and explicit memory are distinguished also by different retrieval processes. Thus, the same episode is retrieved with awareness of its spatio-temporal context for an explicit task but is retrieved without awareness for an implicit task (Jacoby & Witherspoon, 1982).

Emphasis on the episodic origins of priming means that the processing approach is consistent with the following findings; long-lasting priming effects (Tulving et al., 1982), preserved priming with new associations with normal subjects (Shimamura & Squire, 1984; Graf & Schacter, 1985), and effects of processing variables such as shifts in modality (Graf et al., 1985). However, the approach does not account for findings of transient priming effects (Graf et al., 1984) or learning of new associations with amnesic patients (Shimamura & Squire, 1984).

Activation

Activation is probably the most common explanation of priming. Priming effects are the result of the temporary activation of pre-existing memory representations (Mandler, 1984). Processing of a word results in an automatic activation of its pre-existing memory representation. This process increases temporarily the availability of the word and, therefore, will bias or facilitate performance in a priming task. Activation occurs automatically and does not contain the contextual information that is necessary for establishing a durable episodic memory trace (Rozin, 1976). Consequently, subjects may be impaired in recognising or recalling the word. In contrast, explicit memory functions are explained by the process of elaboration (Rozin, 1976). Elaboration is the conscious process of relating the word to a spatio-temporal context, linking it with associations, and developing other cognitive links (Schacter, 1986). Elaboration results in a durable memory so if required the subject is able to recall the word or to identify it as one that was just presented.

Activation provides the best explanation for findings of priming effects in the absence of elaborative processing (Jacoby & Dallas, 1981). Furthermore, suggestions that activation results in the temporary availability of the words is consistent with findings of transient priming effects (Graf et. al., 1984; Squire et al., 1985). In particular, activation predicts the very rapid deterioration of priming effects which is detected in some studies (Squire et al., 1985; Tulving et al., 1991). Finally, activation theory explains the findings of no priming effects for

new associations in that the new associations possess no pre-existing representations to be activated (Rozin, 1976).

Activation does not represent a comprehensive explanation of priming effects because there are some findings which are not consistent with activation theory. For example, the automatic activation of the pre-existing representation is generally assumed to be only temporary and, therefore, has difficulty in accounting for long -lasting priming effects (Sloman et al., 1988). Also, findings of priming effects with new associations in both normal subjects and amnesics is inconsistent with the idea that it is only pre-existing representations that are made temporarily available (Graf & Schacter, 1987).

PRIMING EFFECTS AND AMNESIA

Priming is preserved in organic amnesia. In amnesia, priming effects have been found with preexisting memory representations, such as words, and the semantic associations between representations (Shimamura & Squire, 1984; Graf et al., 1985). Cross modality priming effects have also been found in amnesia. Priming effects have been demonstrated in amnesic patients with a wide variety of etiologies, including electroconvulsive therapy (Squire et al., 1985), anoxic encephalopathy (Graf et al., 1985), Korsakoff syndrome (Squire et al., 1985; Squire et al., 1987), closed head injury (Tulving et al., 1991), encephalitis (Graf & Schacter, 1986), bilateral removal of the medial temporal area (Tulving, 1991), and an hypotensive incident (Graf et. al, 1985).

Priming effects appear dependent, at least in some part, on the severity of amnesia. For example, priming with new semantic associations is preserved with mildly but not severely impaired amnesic patients (Graf & Schacter, 1986; Shimamura & Squire, 1989). One explanation of impaired priming of new associations with only severe amnesics has been framed within a levels of processing approach (Shimamura 1986). Severely amnesic patients are less capable of utilising the explicit memory function of elaborative processing and, therefore, are less able to form semantic associations between the items. Whereas priming for new associations may be dependent upon the severity of memory impairments, little research has been conducted in regard to priming for pre-existing representations. Findings of preserved priming effects but impaired explicit memory in amnesic patients have been explained by the theory that activation is spared in amnesia but elaboration is not (Squire & Shimamura, 1984).

It is not so clear whether priming effects are preserved in benzodiazepine-induced amnesia. Fang, Hinrichs, and Ghoneim (1987) compared performance in a group of subjects who received 0.3mg/kg diazepam and a group of subjects who received a placebo on a free recall, cued-category generation, and stem word completion task. Both the diazepam group and placebo group completed significantly more stems forming words from a recently presented word list than stems forming new words. However, the diazepam group showed significant impairment in the free recall task. Similar results were found with a cued-category recall priming task. Danion, Zimmermann, Willard-Schroeder, Grange, and Singer (1989) also found priming effects

with a word completion and cued category recall task when subjects were administered 0.2mg/kg diazepam. Fang et. al. (1987) conclude these results demonstrate that priming tasks utilise implicit memory structures and free recall tasks are mediated by impaired explicit memory.

Further studies result in a more complicated picture. A number of studies demonstrate impaired priming with lorazepam. For example, Brown et al. (1989) found impaired word completion and cued-category recall with 3mg of orally administered lorazepam. Danion, Peretti, Grange, Bilik, Imbs, and Singer (1992) similarly found 2.5 mg of orally administered lorazepam produced impaired priming with a word completion task. Sellel, Danion, Kauffmann, Grange, Imbs, Linden, and Singer (1992) directly compared the effects of lorazepam and diazepam upon word and picture completion priming and found only lorazepam impaired performance on the word completion task. Diazepam produced only minor effects upon the more sensitive picture completion task. These results suggest that lorazepam and diazepam have differential amnesic effects.

Currently, no widely agreed explanation exists to reconcile these findings. It is possible the differential effects of diazepam and lorazepam are dose related. However, manipulations of dosage in the above studies have not revealed any illuminating pattern of dose effect (Danion et al, 1993). This may be because too few studies manipulating dosage have been conducted. More importantly, it would be difficult to determine dosage effects across drugs because little information exists that is directly comparable (Curran, 1986; O'Boyle, 1988). Therefore, from the small amount of existing data, it does not

seem differential benzodiazepine effects are attributable to dose (Sellel et al., 1992). An alternative hypothesis is that differential benzodiazepine effects are task dependent, that is, lorazepam-impaired priming occurs only with particular priming tasks. However lorazepam impaired priming has been found with both with a word and picture completion task (Sellel et al., 1992). This suggests it is the priming effect in general that is impaired rather than the nature of word completion task.

To further complicate the situation Brown et al. (1989) found impaired word completion and cued-category recall priming, but preserved recognition performance with 3 mg of orally administered lorazepam. A finding of impaired priming is consistent with other findings of impaired priming with lorazepam, but no other lorazepam study found impaired recognition. Brown et. al. (1989) conclude these results indicate a "partial" double dissociation between priming and recognition in the organic amnesia syndrome and lorazepam-induced amnesia. In the organic amnesia syndrome priming implicit memory functioning is preserved and explicit memory functioning is impaired. The opposite occurs in lorazepam-induced amnesia, with explicit memory preserved and implicit memory impaired. One explanation of this double dissociation is framed within the activation approach. Activation explains how priming is preserved when explicit memory is impaired severely in organic amnesia. Lorazepam may inhibit activation and, therefore, impair priming (Brown et al., 1989).

TIME COURSE OF DELAYED RECALL IMPAIRMENT

The free recall task has been used to examine deficits in LTM in organic and temporary benzodiazepine-induced amnesia. Recollection of word lists is assumed to be a product of episodic memory and therefore represents an explicit memory function. It has been established that delayed free recall performance is impaired severely in organic amnesia (Baddeley & Warrington, 1970) and temporary benzodiazepine-induced amnesia (Subhan, 1983).

The majority of pharmokinetic studies have examined delayed recall impairment with considerable delays. Impairment in delayed recall of word lists have been found at 12, 20, 24, 45, 60, 110 minutes, 3-5 hours, 10 hours, the next morning, and a week after presentation (Brown et al., 1982; Subhan & Hindmarch, 1983; Borbely et al., 1988). This pattern of results is consistent amongst the benzodiazepine derivatives (Curran, 1986).

Only a few studies have attempted to examine the pattern of delayed recall impairment within 10 minutes of presentation. Some studies have shown impairments in the recall of visual stimuli within 10 minutes of presentation. For example, Dundee and Wilson (1980) found subjects administered 0.1mg/kg midazolam intravenously recalled only 35% of pictures 10 minutes after their presentation. Also, Luyk, Boyle, and Ward-Booth (1987) found dental patients receiving diazepam or midazolam were impaired in recalling four photographs five minutes after presentation. There is some suggestion that a similar pattern occurs with word lists (Hennessy, Kirkby, &

Montgomery, 1991). Brown et al. (1983) found impairments in recalling lists of 12 words 1.5 minutes after presentation when subjects were administered 2.5 mg lorazepam intravenously. Further research is required to clarify the time course of delayed free recall impairments. Clarifying the time course of impairment in the delayed free recall task could expand our theoretical understanding of the nature of the impairment.

SUMMARY AND CONCLUSION

Benzodiazepines produce specific but temporary memory impairments that are similar to the permanent effects of organic amnesia. The primary effect of benzodiazepines is to impair episodic memory, as indexed by severe deficits in the acquisition of newly presented information. In contrast, the semantic and procedural components of LTM appear to be relatively unaffected, as indicated by preserved general intellectual functioning and learning of new cognitive, and visual-motor skills respectively. WM also appears preserved, with normal performance on a wide variety of WM tasks. Graf and Schacter (1985) suggested LTM could be divided into explicit and implicit memory functions, of which episodic and semantic memory mediate the former and procedural memory mediates the latter. It appears that explicit memory is more affected than implicit memory in benzodiazepine-induced amnesia (Sellal et al., 1992).

The type of memory impairments that different benzodiazepines produce appear to be qualitatively similar (Danion et al., 1992). Amnesia differs from one benzodiazepine to another in terms of the time of onset, duration, and potency of effects. These variations are a function of the drug itself, the

dose, the route of administration, and the characteristics of the subjects receiving the drug (Lister, 1985; Sellal et al., 1992). It is not entirely clear how benzodiazepines produce these memory impairments but there is some suggestion it involves benzodiazepine specific receptors in similar CNS areas to those associated with amnesia.

The degree to which benzodiazepine-induced amnesia and organic amnesia share similar features impacts upon the contribution benzodiazepine-induced amnesia has in the understanding of amnesia and memory. If they share similar deficits then benzodiazepine-induced amnesia may provide a useful model in investigating organic amnesia. Furthermore, benzodiazepine-induced amnesia would represent a valuable methodology for examining amnesia because of the greater control that is possible with organic amnesia. Benzodiazepine-induced amnesia would still represent an important area for research if there are differences between benzodiazepine-induced amnesia and organic amnesia, as it would offer the opportunity to further separate memory functions based upon its distinct modes of effect.

Priming may be one memory function that does not demonstrate the same performance in organic and benzodiazepine-induced amnesia. Priming has been shown clearly to be preserved in organic amnesia but further research would be useful to detail the status of priming in benzodiazepine-induced amnesia.

Another issue that arises from benzodiazepine-induced amnesia research is the time course of delayed recall deficits. There are suggestions deficits occur as early as three minutes

but few studies have examined this. Further research is required to outline the time course of delayed recall impairments in the first five minutes after presentation.

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Abstract

Benzodiazepines produce temporary memory impairments that are similar to those found permanently in organic amnesia (Ghoneim & Mewaldt, 1990). This experiment examined priming, a memory function found to be preserved in organic amnesia (Graf, Squire, & Mandler, 1984), to determine if it is preserved in benzodiazepine-induced amnesia. Healthy volunteers were assigned into either a lorazepam (2 mg) or placebo group and presented a stem word completion priming task. The time course of impairments in the delayed free recall of word lists was also examined. Word lists were presented for free recall immediately, one minute, three minutes, or five minutes after presentation. The digit span test and a sedation questionnaire were presented to examine WM functioning and sedation effects respectively . These tasks were presented at pre and post drug test sessions. Lorazepam impaired the priming effect but not as substantially as had been found in previous research. This result may be the product of the specific elements of the priming task used in this experiment. Lorazepam did not appear to impair digit span. Free recall was impaired at the one, three, and five minute delay conditions but not at immediate recall. This confirms earlier research that suggested impaired delayed recall within three minutes of presentation (Brown, Brown, & Bowes, 1983; Hennessy, Kirkby, & Montgomery, 1991). Sedation ratings were increased by lorazepam, but were weakly correlated with memory.

Benzodiazepines are a class of minor tranquillisers used extensively for their hypnotic, anti-anxiety, and muscle relaxant effects which appear to produce temporary memory impairments similar to those associated with organic amnesia (Ghoneim & Mewaldt, 1990). The apparent similarity between temporary benzodiazepine-induced amnesia and organic amnesia may enable benzodiazepines to be used as another method of examining organic amnesia and may facilitate the understanding of the mechanisms underlying normal memory (Fang, Hinrichs, & Ghoneim, 1987). Benzodiazepines represent an invaluable methodology to explore amnesia and memory because they allow greater manipulation of experimental variables (eg: pre-amnesia performance, matching subjects for age, sex, education, IQ) than is possible with organic amnesia (Brown, Brown, & Bowes, 1989).

Benzodiazepines appear to act on benzodiazepine specific receptor sites located in the CNS, particularly in areas commonly associated with memory functions (Greenblatt, Shader, & Abernathy, 1983; Davies, 1985). Binding of a benzodiazepine to these specific receptor sites triggers a chain of neurochemical responses that combine to mediate neuronal inhibition (Ghoneim & Mewaldt, 1990). The presence of benzodiazepine enhances the action of GABA (gamma-aminobutyric acid), which is a major inhibitory neurotransmitter. GABA acts by opening up the neronal membrane for chlorine ions to enter and the chlorine ions alter the electrical potential of the neuron so that it less likely to excite, thus affecting memory (Davies, 1985).

Organic amnesia is associated with lesions to the neural circuit connecting the medial temporal lobes, diencephalon, and frontal lobes and involves relatively distinctive memory

deficits (Lezak, 1983; Walsh, 1987). Identifying the memory functions that are spared in amnesia plays an important role in understanding the mechanisms underlying memory itself (Brown et al., 1989). Perhaps the most widely accepted theoretical model of memory is the multistore model, which views memory functions as the product of distinct structural components (Atkinson & Shiffrin, 1968; Baddeley, 1990).

Visual, tactile, auditory, and olfactory Information enters a series of very brief sensory stores, where information either decays rapidly or is transferred into a temporary working memory (WM). WM represents the locus of control within the memory systems and is related to consciousness (Atkinson & Shiffrin, 1968; Baddeley, 1990). WM is responsible for holding information temporarily whilst it is acted upon by a wide range of cognitive processes, including reasoning, comprehension, and learning (Baddeley, 1990). WM is made up of at least two sub-systems, an articulatory loop and a visual-spatial scratch pad that are co-ordinate and supervised by a central executive (Baddeley & Hitch, 1974). The phonological loop processes and temporarily maintains speech-based information and contributes to verbal memory span (Morris & Jones, 1990). The visual-spatial scratch pad holds and processes visual image representations to perform a number of discrete visual and spatial functions. The central executive co-ordinates and supervises these sub-systems by either allowing well learned functions to occur relatively automatically or by interrupting and modifying on-going behaviour to deal with novel tasks or situations (Norman & Shallice, 1986). WM appears to remain

unaffected in amnesia, as indicated by normal performance on a variety of WM tasks, including digit span, Brown-Peterson task, and recency effect in free recall (Baddeley & Warrington, 1970; Milner, 1971).

WM also directs the flow of information into the relatively more permanent Long-Term Memory (LTM). LTM may be divided into episodic, semantic, and procedural components. Episodic memory is an autobiographical record of life events (Tulving, 1983). The primary deficit in amnesia is severely impaired delayed recall of newly presented information (Baddeley, 1983; Baddeley & Wilson, 1988). This deficit represents an impairment in episodic memory functioning. Semantic memory contains general knowledge about the world, concepts, rules and language (Ghoneim & Mewaldt, 1990). Semantic memory appears to be preserved in amnesia as indicated by preserved performance in verbal fluency tests and general intellectual functioning (Hirst, Johnson, Phelp, & Volpe, 1983). Procedural memory represents the learning of motor, visual, or cognitive skills (Tulving, 1985). The fact that amnesic patients are capable of learning a wide variety of visual-motor skills suggests procedural memory is spared (Brook & Baddeley, 1976; Cohen, 1984; Baddeley & Wilson, 1988). Perhaps another useful theoretical organisation of LTM is to distinguish between explicit and implicit memory functions (Graf & Schacter, 1985). Explicit memory is revealed when performance involves conscious or explicit recollection from LTM and is assumed to be a function of episodic and semantic memory (Ghoneim & Mewaldt, 1990). Implicit memory is revealed by performance that does not necessarily involve conscious recollection of information. It is assumed that

explicit memory functions are more affected than implicit memory functions in organic amnesia (Graf & Schacter, 1985; Tulving, 1991).

An alternative theoretical framework, the levels of processing approach, views memory as the product of cognitive processes rather than underlying structures (Craik & Lockhart, 1972). Memory reflects the depth to which information is processed. Deeper levels of processing involve encoding the semantic qualities of information and elaborating it with a context and associations and result in a longer lasting and stronger trace (Craik & Lockhart, 1972). The levels of processing approach adds to the multistore model by expanding upon the processes involved in LTM learning (Baddeley, 1993).

Benzodiazepines produce memory deficits that are similar to those found in organic amnesia. Firstly, the delayed recognition or recall of visual and verbal information, presented after benzodiazepine administration, is impaired profoundly (Brown & Lewis, 1981; Kliendienst-Vanderbeke, 1984) and the primacy effect in the serial position curve is reduced (Subhan & Hindmach, 1983). Secondly, WM tasks such as digit span (Brown & Lewis, 1983), the Brown-Peterson task (Ghoneim & Mewaldt, 1975), and the recency component in the serial position curve (Subhan & Hindmach, 1983) are unaffected. Thirdly, there is some evidence of preserved visual-motor skill learning (Lister & File, 1984; Ghoneim, Mewaldt, & Hinrichs, 1984). This pattern of memory performance is found with a wide variety of benzodiazepines including triazolam, flunitrazepam, clobazam, diazepam, midazolam, and lorazepam (for review see O'Boyle, 1988). However, the time of onset, duration, and potency of

these effects differ substantially according to the benzodiazepine derivative used, the mode of administration, and characteristics of the subject (Lister, 1985; Ghoneim & Mewaldt, 1990).

One memory function that represents a problem for the assumed similarity between organic and benzodiazepine-induced amnesia is priming. Priming is changed or enhanced performance in a processing task because of prior exposure to the information involved in the processing task (Brown et al., 1989). Priming is assumed primarily to be an implicit memory function (Schacter, 1986). A common task used to explore priming is word completion. This typically involves presenting subjects with a list of word for processing, for example subjects may be required to rate a word on how much they like it. Subsequently, and without reference to the previously presented words, subjects then are required to complete word fragments (A_c _u_t_n_) or word stems (mot_ _ _) with the first word that comes to mind. Priming is revealed by greater completion of stems that form previously presented words than stems that form new words.

It has been well established that priming is preserved in organic amnesia. Preserved priming effects have been found with pre-existing memory representations, such as words (Graf et al, 1984), and pre-existing associations between words, such as semantic associates (Tulving, Schacter, & Stark, 1982; Shimamura & Squire, 1984). Priming with new associations (eg: unrelated word pairs) has been demonstrated in mildly but not severely impaired amnesic patients (Graf & Schacter, 1986). Also, priming effects can extend beyond the modality in which

the information is processed and occur with completion tasks in another modality (Graf et al., 1985). Preserved priming effects in amnesic patients have been considered as strong evidence for the distinction between implicit and explicit memory. Amnesic patients are severely impaired on traditional tests of explicit memory, such as free recall, cued recall, and recognition but demonstrate preserved priming, which is assumed to be an implicit memory function.

It has not been demonstrated clearly that priming is preserved in benzodiazepine-induced amnesia. Priming effects may be dependent upon the benzodiazepine used. For example, priming appears to be preserved with diazepam but impaired with lorazepam. Fang, Hinrichs, and Ghoneim (1987) found subjects administered 0.3 mg/kg diazepam demonstrated preserved priming in a word completion task but were impaired in a free recall task. Danion, Zimmermann, Willard-Schroeder, Grange, and Singer (1989) found similar results with 0.2 mg/kg diazepam. In contrast, Brown, Brown, and Bowes (1989) found impaired priming with a word completion and cued-category task when subjects were administered 3 mg lorazepam and Danion, Peretti, Grange, Bilik, Imbs, and Singer (1992) found impaired word completion priming when subjects were orally administered 2.5 mg lorazepam. Sellal, Danion, Kaufmann-Muller, Grange, Imbs, Van Der Linden, and Singer (1992) directly compared the effects of diazepam and lorazepam on a word and picture completion priming task. Priming in the word and picture completion were impaired with administration of lorazepam but priming in the word completion task was preserved with administration of diazepam.

Currently there does not appear to be a satisfactory explanation for differential benzodiazepine effects. Sellal et al. (1992) found lorazepam impaired priming effects in word and picture completion tasks. This supports the notion that it is the underlying priming process, itself, which is impaired rather than verbal or visual components of the task. In that study subjects were presented with explicit instructions for the word completion task but implicit instructions for the picture completion task. It is, therefore, possible that the stronger impairment in the word completion task is the product of explicit memory operations not implicit ones. Also, the differential priming effects of lorazepam and diazepam do not appear to be explained by different doses. Lorazepam and diazepam produce comparable deficits in explicit memory but only lorazepam appears to impair priming effects (Danion et al., 1992; Sellal et al., 1992). An increase in lorazepam dose from 1.75 mg to 2.5 mg and diazepam from 15 mg to 20 mg produces the same pattern of results (Sellal et al., 1992). This suggests dose affects the magnitude but not the type of memory impairments. It is difficult, however, to be conclusive because there are few direct comparisons of dose effects between the two drugs, and some are not consistent (Dundee, McGowen, Lilburn, Mckay, & Hegarty, 1979; Brown et al., 1981; Brown & Lewis, 1983).

Brown et al. (1989) found that subjects administered lorazepam demonstrate preserved recognition performance but impaired priming. Brown et al. (1989) concludes these results indicate a "partial" double dissociation between lorazepam-induced and organic amnesia (Graf et al., 1984). Implicit memory

is preserved and explicit memory impaired in organic amnesia. In contrast, Brown et al. (1989) found implicit memory was impaired and explicit memory preserved in benzodiazepine-induced amnesia. This may limit or alter the type of contribution lorazepam, and benzodiazepines, could make to the understanding of organic amnesia. Brown et al. (1989) results, however, have not been replicated elsewhere, and, it appears recognition performance was partially impaired.

One possibility to consider is that the requirements of the word completion task explain, to some degree, preservation of priming effects. The studies on benzodiazepine-induced amnesia often differ from studies on organic amnesia in the degree of processing the words receive. In the majority of organic amnesia studies the words were presented twice in the processing task (Graf et al., 1984). In studies on benzodiazepine-induced amnesia the words were presented only once (Danion et al., 1989; Danion et al., 1992; Sellal et al., 1992). Further research is required to elucidate the status of priming in benzodiazepine-induced amnesia.

A secondary point of interest is the onset of delayed recall deficits with benzodiazepines. Delayed recall tasks have been used widely in amnesia research to demonstrate impaired explicit memory. The majority of benzodiazepine studies have observed impaired recall between 20 minutes and seven days after presentation (Ghoneim & Mewaldt, 1975; Brown, Lewis, Brown et al., 1981; Subhan & Hindmarch, 1983; Lister & File, 1984). The few studies that attempted to examine delayed recall within 10 minutes of presentation have observed impairments. Hennessy, Kirkby, and Montgomery (1990) found deficits in

delayed recall of a complex figure task by three minutes. Brown, Brown, and Bowes (1983) found impairments in recalling lists of 12 words with a filler task one and a half minutes after presentation. Further research is required to outline the time course of deficits in delayed free recall in the first five minutes after presentation.

In light of the above uncertainty as to the status of priming in benzodiazepine-induced amnesia, this experiment will investigate whether or not priming is preserved in lorazepam with task requirements of presenting the words twice. Preservation of priming effects with lorazepam, utilising similar task requirements as studies on organic amnesia, would suggest benzodiazepine-induced amnesia is functionally similar to the organic amnesic syndrome. Alternatively, impaired priming effects with lorazepam would suggest there are differential benzodiazepine effects and that lorazepam-induced amnesia and organic amnesia may have a different basis. A second aim of this research is to examine the time course of deficits in delayed free recall within the first five minutes after presentation.

METHOD

Subjects

Thirty five subjects participated in this experiment. The subjects were healthy volunteers who ranged in age from 19 to 34 (average age 23.2). Two subjects from the placebo group were not included in the analysis because of incomplete results. Subjects were recruited through advertisements at the

University of Tasmania and Department of Health and Community Services (Glenorchy office), and were offered food or drink after participating in the study.

Subjects were randomly allocated into placebo group (n=15) and lorazepam group (n=18) and tested by a double blind procedure. The age range of the placebo group was 19 - 33 with an average age of 22.8, and the lorazepam group was 19-34, with an average of 23. 5. The placebo group consisted of eight females and seven males, and the lorazepam group consisted of nine females and nine males.

All subjects were screened previous to testing and were excluded if there was a history of drug or alcohol abuse, brain-injury, psychiatric disorder, mental retardation, a significant medical condition, recent weight loss, or if they were currently pregnant or taking medication. Informed consent was obtained in writing from each subject prior to testing.

Materials

Sedation Rating Questionnaire: Subjects were presented with a questionnaire containing a rating scale and were requested to place a cross on the line that best described how tired they felt (eg., 'extremely tired' or 'not tired at all'). The questionnaire was presented at the beginning and at the end of the pre-drug and post-drug test sessions. This task was selected as a measure of sedative effects (Subhan & Hindmarch, 1982; Brown et al., 1989).

Digit Span Test: Subjects were presented the digit span task from the Wechsler Adult Intelligence Scale- Revised. Both parts,

digits forward and digits backward were administered. This test was selected as a measure of WM functioning (Squire, 1987).

Semantic orienting task. Subjects were presented 24 words, written on card, at one card per five seconds and requested to rate how much they liked the word (ranging from 'considerably dislike' to 'considerably like'). The list of 24 words were presented twice to increase the priming effect. The word lists were presented in a balanced order, with half the subjects receiving the words in one order and the other half of subject receiving it in the reverse order. The subjects were not informed that the purpose of this task was to test memory. All of the words fulfilled the following nine criteria

1. Two syllables.
2. Initial three letters could be used as a stem to complete at least ten other words
3. Initial three letters could not become a stem to complete another word already in the list
4. Four to nine letters.
5. Relatively concrete noun.
6. Not a proper noun
7. Between 50 and 300 per million of the Kucera-Francis scale for frequency (MRC Psycholinguistic Database, Colthart, 1981).
8. Between 500 and 700 on the familiarity scale (MRC Psycholinguistic Database, Colthart, 1981).
9. Between 500 and 700 on the concreteness scale (MRC Psycholinguistic Database, Colthart, 1981).

Immediate recall task. Subjects were requested to write down as many of the words that had just been presented in the semantic orienting task as they could recall in one minute.

***Word completion task*:** Subjects were presented with 30 three letter word stems on a sheet of paper and asked to use them to complete the first word that came into their mind. Eighteen of the word stems were target stems that could form the words presented in the semantic orienting task, and 12 of the stems could only form words that had not been presented previously . These 12 baseline stems could form words that had the same criteria as the 24 words in the semantic orienting task. The first six stems were baseline stems to provide practice examples for the task. The other six stems were randomly presented with the 18 target stems. If subjects asked if this task was related to the orienting task, the instruction to complete these stems with the first words that came to mind was repeated.

Delayed Free Recall Task: Subjects were presented verbally with four lists of 12 words, at a rate of one word per second and requested to recall the word list in any order immediately, one minute, three minutes, or five minutes after presentation. The order of recall delays was counterbalanced with a Latin square design. The presentation order of words within each list was determined randomly but each subject was presented the words in the same order. All the words fulfilled the same criteria as the semantic orienting tas, except that the words did not appear on any other of the delayed free recall word lists and did not appear in the semantic orienting task.

The delays between presentation and recall were filled by musical rating exercises, to prevent subjects rehearsing the words during the delay. Subjects were presented passages of music, running for one, three, or five minutes and requested to

rate how much they liked the passage (ranging from 'liked a lot' to 'disliked a lot') on a music passage rating questionnaire. Three music passages, of approximately one minute length were presented as pause fillers to provide an interval between recall of one list and presentation of the next list, in order to diminish interference effects.

Procedure

Subjects were randomly allocated into placebo and lorazepam group. The placebo group received 50 mg vitamin B6 (Pyridoxine, Vitaglow Pty. Ltd) and the drug group received 2 mg lorazepam (Ativan, Ayerst laboratories). All subjects were presented with a numbered envelope containing two tablets, either lorazepam or vitamin B6. Subjects were requested to abstain from caffeine, food or alcohol for at least four hours before testing.

Most subjects were tested in the evening but some were tested during the day on weekends. The procedure and possible effects of the lorazepam were explained to the subjects and then written informed consent was obtained. Subjects were requested to fill out a medical history questionnaire. Testing occurred prior to drug administration and 75 minutes after drug administration. Each testing session lasted approximately 30 minutes. The tasks were presented in the same order at both pre-drug and post-drug test sessions for all subjects.

Table 1. Test Session Presentation Order

TASK	TIME
1. Sedation Rating Questionnaire (Begin)	30 seconds
2. Priming Effect Task	8-10 minutes
Semantic orienting task	
Immediate free recall	
Word completion task	
3. Delayed Free Recall Task	18-20 minutes
4. Digit Span test	3-5 minutes
5. Sedation rating questionnaire (end)	30 seconds

RESULTS

The sedation rating questionnaire was scored by averaging the ratings made at the beginning and end of the session for the pre-drug and post-drug test sessions. The average score of the beginning and end of session ratings represents a sedation rating for the whole test session. The digit span test scores are the total of forward and backward span. Immediate recall in the priming effect task was the total number of words recalled correctly by the subject. Priming in the word completion task was measured from the percentage of primed words completed (stems that formed words from the semantic orienting task)

minus the percentage of baseline words completed (stems that could not form words presented previously). The delayed free recall task was scored by the total number of words recalled in each list.

Words were considered complete in the priming effect task despite appearing in the wrong tense, in plural, or with slight distortions of spelling. Words were counted in the delayed free recall task despite appearing in the wrong tense, in plural, or with slight distortions in spelling, or as a homonym.

Analysis of variance was used to compare pre-drug to post-drug performance for the placebo and lorazepam group in the priming effect task, delayed free recall task, digit span test, and sedation rating questionnaire. Summary tables for analyses conducted are presented in Appendix D.

Sedation Rating Questionnaire: There was an interaction between drug groups (placebo v lorazepam) and test session (pre-drug v post-drug) on the combined sedation rating questionnaire scores ($F(1,31) = 9.0$, $p=.0054$). Simple effects analysis (Keppell, 1982, p176) showed the lorazepam group rated themselves significantly more tired during the post-drug test session than during the pre-drug test session ($F(1,31) = 11.8$, $p=.002$). In contrast, the placebo group did not rate themselves significantly more tired during the post-drug test session than during the pre-drug test session. This suggests lorazepam produced sedative effects. Table 1 presents the sedation rating scores for the placebo and lorazepam groups in pre-drug and post-drug test sessions.

Table 1. Mean Scores on the sedation rating questionnaire for placebo and lorazepam groups at pre-drug and post-drug testing

	PRE-DRUG	POST-DRUG
PLACEBO GROUP	**6.6**	**7.0**
LORAZEPAM GROUP	**7.1**	**5.7**

SEDATION RATING KEY:

2 = Extremely tired
4 = Quite tired
6 = Tired
8 = Not really tired
10 = Not tired at all

Digit Span Test: There was no significant interaction between drug group (placebo v lorazepam) and test session (pre-drug v post-drug) on the combined forwards and backwards components of the digit span test. However there were significant main effects for Drug group ($F(1,31) = 4.63$, $p = .04$) and time ($F(1,31) = 4.40$, $p = .044$). Significant main effects for drug indicates the lorazepam group had a lower combined pre-drug and post-drug digit span score than the placebo group. Significant time effects indicate that the combined lorazepam and placebo group digit span scores were higher at the pre-drug session than the post-drug test session. As the interaction is not significant it cannot be concluded that lorazepam reduces digit span. Table 2 presents the mean scores for the digit span test for the placebo and lorazepam groups at pre-drug and post-drug test sessions.

Table 2. Mean Scores on the digit span test for placebo and lorazepam groups at pre-drug and post-drug testing

	PRE-DRUG	POST-DRUG
PLACEBO GROUP	**17.9**	**17.2**
LORAZEPAM GROUP	**16.1**	**15.2**

Immediate recall: There was an interaction between drug group (placebo v lorazepam) and test session (pre-drug v post-drug), ($F(1,31) = 11.7$, $p = .002$). This indicates the lorazepam group recalled significantly less of the semantic orienting task words than the placebo group in the post-drug test session ($F(1,30) = 11.2$, $p = .002$) but there was no significant difference between the lorazepam and placebo group at the pre-drug test session ($F(1,30) = 1.30$, $p = .263$). Figure 1. shows the mean number of words recalled by the placebo and lorazepam groups in the pre-drug and post-drug test sessions.

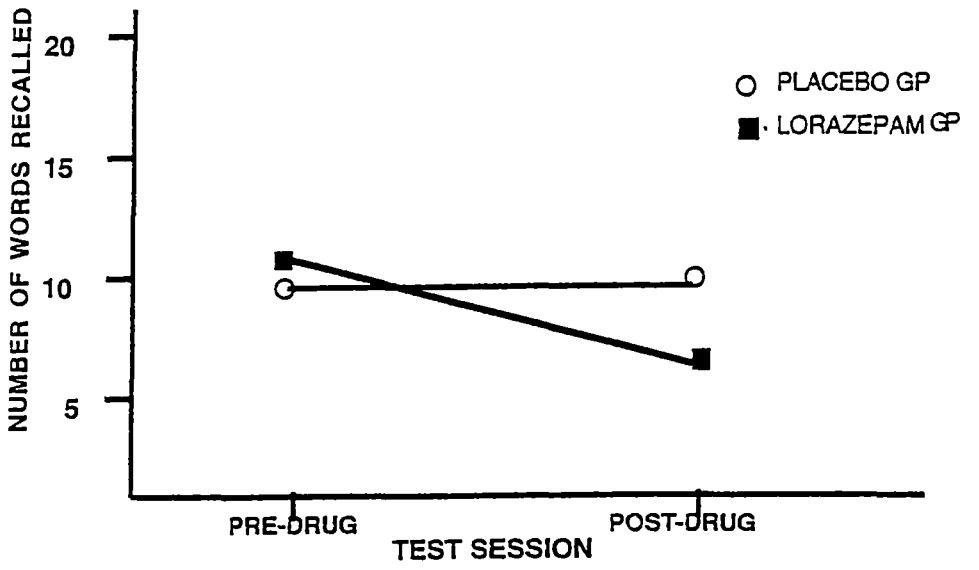

Figure 1: Words recalled in immediate recall task for placebo and lorazepam groups at post drug testing

Word completion task: Defining priming as the difference in the percentage of primed and non-primed words that were completed, an analysis of variance showed a significant interaction between drug group (placebo v lorazepam) and test session (pre-drug v post-drug), ($F(1,31) = 4.94$, $p = 0.03$). This indicates that at the post-drug test session the lorazepam group showed significantly less priming effect than the placebo group($F(1,31), = 6.2$, $p = 0.02$) but there was no significant difference between the lorazepam and placebo group at the pre-drug test session ($F(1,31), = 0.02$, $p = 0.8$). This suggests that lorazepam does impair priming effects. Figure 2 illustrates the percentage of priming effect for the placebo and lorazepam groups at the pre-drug and post-drug test sessions.

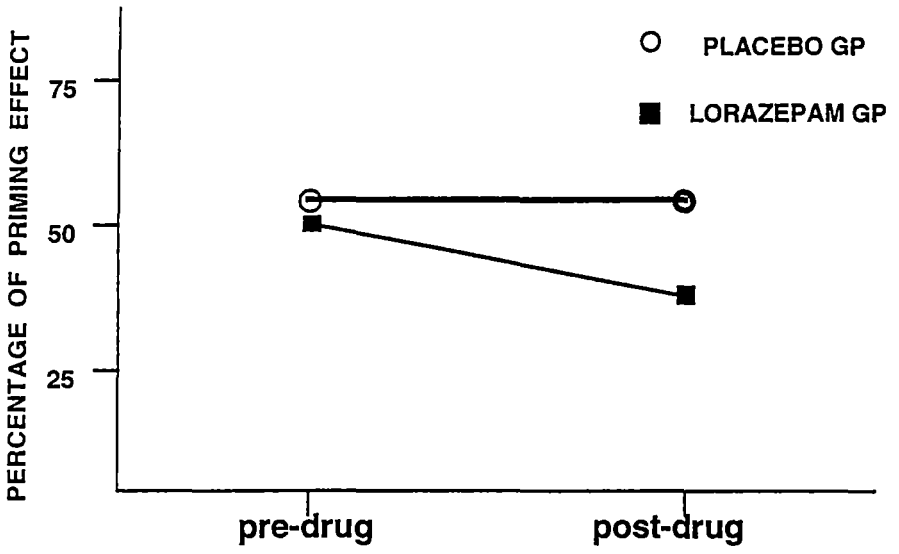

FIGURE 2. Mean percentage of primed words completed minus percentage of non-primed words completed for placebo and lorazepam groups at pre-drug and post-drug test sessions

Delayed Free Recall Task: There was a significant interaction effect between drug group (placebo v lorazepam), test session (pre-drug v post-drug), and delay intervals (immediate recall, one minute delay, three minute delay, five minute delay), ($F(1,31) = 3.703$, $p = .0144$). Analysis of pre-drug performance revealed no significant interaction between drug group (placebo v lorazepam) and delay interval, $F(1,31) = 2.092$, $p = .11$. Simple effects analysis indicated there was no significant difference between the placebo and lorazepam groups at immediate recall ($F(1,31) = .380$, $p = .542$), one minute delay ($F(1,31) = 3.7$, $p = .064$), three minute delay ($F(1,31) = .647$, $p = .427$), and five minute delay ($F(1, 31) = .440$, $p = .512$).

Figure 3 illustrates the percentage of words recalled by placebo and lorazepam groups at the post-drug test session with immediate recall, one minute, three minute, and five minute delay intervals. Analysis of post-drug performance revealed a significant interaction between drug group (placebo v lorazepam) and delay interval (immediate recall, one minute delay, three minute delay, 5 minute delay), ($F(1,31) = 4.865$, $p = .004$). Simple effects analysis showed that the lorazepam group recalled significantly less words than placebo group at the one minute delay ($F(1,31) = 7.080$, $p = .01$), three minute delay ($F(1,31) = 24.19$, $p < .001$), and five minute delay conditions ($F(1,31) = 1.903$, $p < .001$), but differences at immediate recall was not significant ($F(1,31) = .845$, $p = 0.36$).

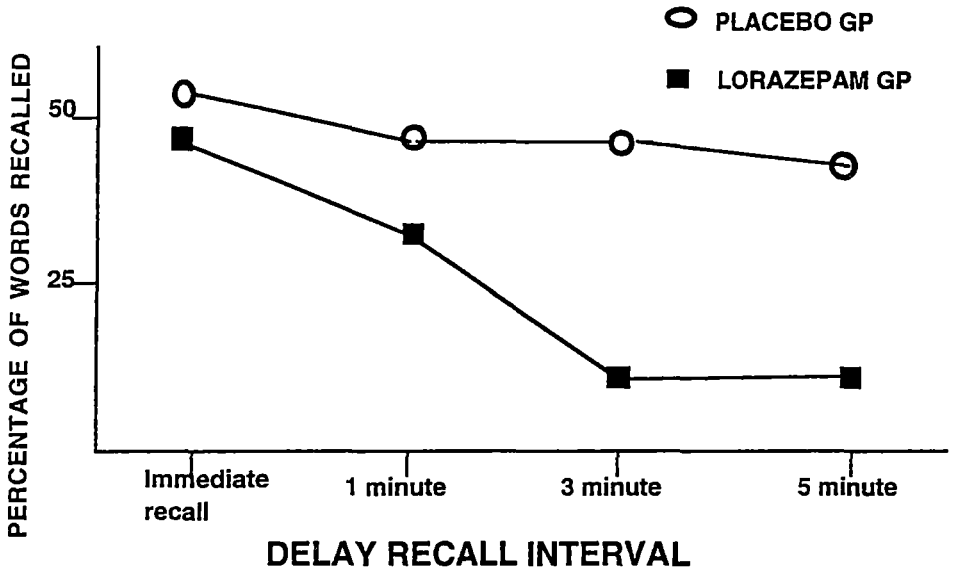

FIGURE 3: Mean percentage of words recalled by placebo and lorazepam group in the delayed free recall task at immediate recall, one minute delay, three minute delay, and five minute delay intervals in the post-drug sesion.

Sedation and priming effects: A correlational analysis was conducted between sedation and priming effect to determine if a relationship existed between them. At the pre-drug test session the correlation between sedation and priming effects was -0.14. At the post-drug test session the correlation between sedation and priming effects was -0.02. At the post-drug test session for the lorazepam group the correlation between sedation and priming effects was -0.03. None of these correlations were significant at the p= 0.05 level.

Sedation and free recall: A correlational analysis was conducted to determine if a relationship existed between sedation and free recall. At post-drug testing the correlation between sedation and immediate recall was .058, one minute delayed recall was -.156, three minute delayed recall was .225, and five minutes was .049. At post drug testing for the lorazepam group the

correlation between sedation and immediate recall was -0.24, one minute delay was 0.15, three minute delay was 0.35, and five minutes was 0.05. none of these correlations were significant at the p= 0.05 level.

Serial position curve: The serial position curve of the free recall task was investigated by dividing each free recall condition into three groups: words 1-4, words 5-8, words 9-12. The total number of words recalled in each group was added for subjects in the placebo group and lorazepam group. The scores were analysed with a drug (2) x position (3) repeated measures ANOVA for each delay condition. There was no significant interaction at the immediate recall ($F(1,31) = .81$, $p= .55$), three minute ($F(1,31) = 1.06$, $p= .35$), and five minute delay conditions ($F(1,31) = .725$, $p= .49$). There was a significant interaction at the one minute delay condition ($F(1,31) = 3.27$, $p=.04$). The serial position curves for the placebo and lorazepam group at post drug testing are illustrated in figure 4-7.

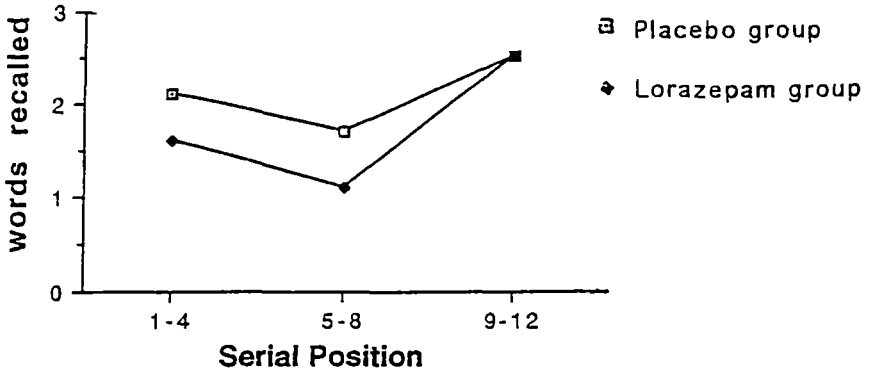

Figure 4: Serial position curve for placebo group and lorazepam group in the immediate recall condition at post drug testing.

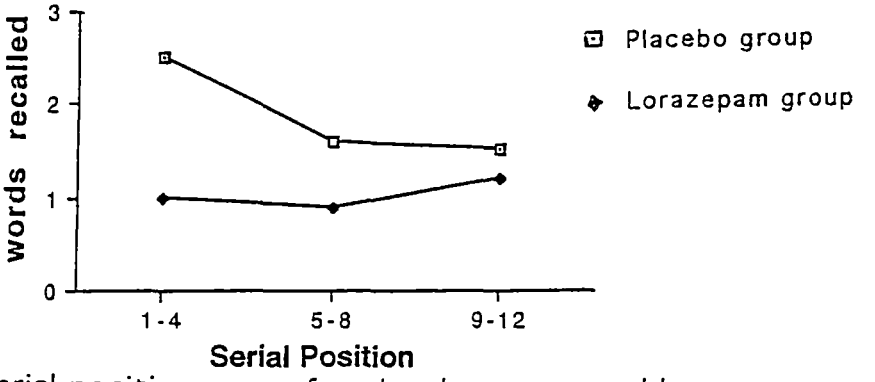

Figure 5: Serial position curve for placebo group and lorazepam group in the one minute delay recall condition at post drug testing.

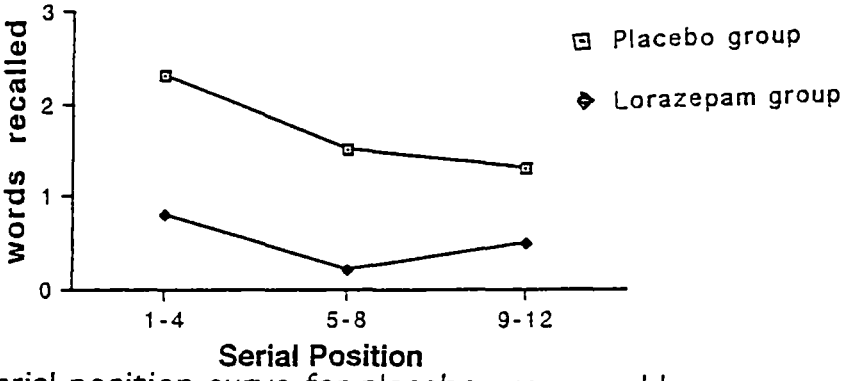

Figure 6: Serial position curve for placebo group and lorazepam group in the three minute delay recall condition at post drug testing.

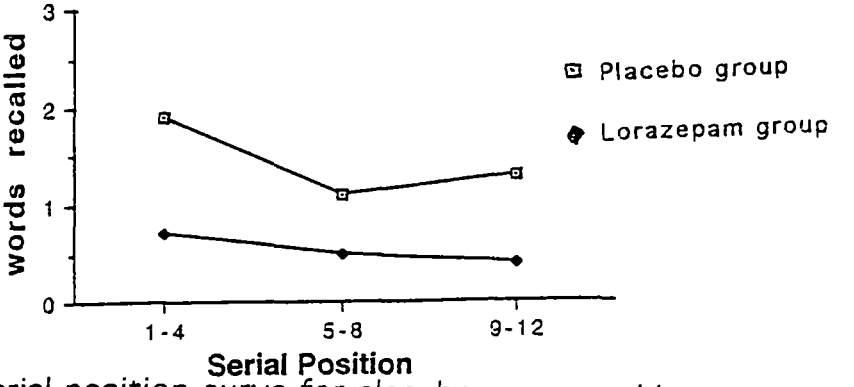

Figure 7: Serial position curve for placebo group and lorazepam group in the five minute delay recall condition at post drug testing.

DISCUSSION

In this experiment lorazepam produced sedative effects. Subjects in the lorazepam group rated themselves as more tired than the placebo group during the post-drug session. Considering that lorazepam produces sedative effects, the question is raised whether impaired priming and delayed free recall are attributable to deficits in more general cognitive processes or to specific memory impairments (File & Lister, 1982; Weingartner, Joyce, Sirocco, Adams, Eckardt, George, & Lister, 1993). A correlational analysis was conducted to explore the relationship between sedation and priming effects and delayed free recall. There was no significant correlation between the level of sedation and amount of priming effects. No significant correlation could be found between sedation and delayed recall one minute, three minutes, and five minutes after presentation. A failure to find a correlation between sedation and priming or delayed free recall performance suggests impaired priming and delayed recall are not explained directly by sedation impairing general cognitive processes. The fact that the impact of sedation, as rated by the subjects themselves, appears to be minor supports such an interpretation. Although the difference was significant, the placebo group rated themselves in between "A little tired" and "Not really tired" and lorazepam group rated themselves just below "A little tired".

Previous research also suggests that impaired priming effects and delayed free recall are not the direct result of sedative effects. Other drugs, like pentobarbital, are capable of producing similar, if not greater, sedative effects to

benzodiazepines but do not produce the same memory impairments (Roache & Griffiths, 1985). Lorazepam does not appear to impair the performance of a number of psychomotor or cognitive task which would be expected if it is the sedative effects that impair general cognitive functioning (Brown et al., 1982; Ghoneim & Mewaldt, 1984). Curran (1991) suggests that even though some studies have demonstrated a correlation between sedation and memory impairments (eg., Roth, Roehrs, Wittig, & Zorick, 1984) this does not mean that sedation produces the memory impairments because correlation does not imply causation, as both may involve individual sensitivity to the dosage.

Digit span has been shown to be a task that is relatively insensitive to the effects of benzodiazepines (Ghoneim & Mewaldt, 1975; Subhan, 1982; Brown et al., 1982). The decline of combined backwards and forwards digit span from pre drug test top post drug test of 0.9 items with lorazepam, compared with a decline of 0.7 for placebo is consistent with previous findings of little or no effect of benzodiazepines on digit span. However, there were main effects for both drug group and time. Main effects for drug indicates the lorazepam group had a lower combined pre-drug and post-drug test session score than the placebo group. Main effects for time indicate the combined lorazepam and placebo group digit span scores were higher at the post-drug than pre-drug test session. The difference between the lorazepam and placebo group at pre-drug testing may suggest the groups were not matched on digit span.

Lorazepam impaired the delayed recall of verbally presented word lists. The lorazepam group recalled significantly

less words than the placebo group one minute, three minutes, and five minutes after the words were presented. Immediate recall was not significantly impaired by lorazepam. These findings are consistent with the wide body of research that has found that the primary memory deficit produced by benzodiazepines is in the delayed recall of verbal information (Brown et al., 1983; Lister & File, 1984).

Subjects in the lorazepam group were significantly impaired in the free recall of word lists as early as one minute after the words were presented. The level of impairment increased and reached asymptote by three minutes after presentation. This is consistent with Hennessy, Kirkby, and Montgomery (1991) who found impairment in delayed recall as early as three minutes and Brown et al. (1983) who found impaired recall of word lists one and a half minutes after presentation.

The delayed free recall data from the current experiment are consistent with the notion that lorazepam-induced amnesia involves deficits in consolidation. According to the multistore model of memory information is consolidated into LTM from the limited capacity WM (Parkin, 1987; Baddeley, 1990). Consolidation occurs when information is elaboratively processed with contextual information and associative memories already in LTM (Bourne et. al, 1987; Baddeley, 1990). Impairment in delayed free recall at one, three, and five minutes suggests that most of the information is lost once it is no longer available in WM. Subjects were prevented from rehearsing the word lists , and therefore maintaining the words in WM, by a musical rating exercise.

The serial position curve showed that lorazepam does not significantly impair the pattern of free recall at immediate recall. The recency effect was spared in the lorazepam group. At the delayed recall conditions the pattern of recall appears to drop away at all three serial positions.

It was found in this experiment that compared to the placebo group, the lorazepam group completed significantly less stems that formed words that had been presented previously in the orienting task. This finding is consistent with previous research that has shown lorazepam impairs priming with similar doses to those used in this experiment (Brown et al., 1989; Danion et al., 1992; Sellal et al., 1992).

Although this experiment did not directly compare lorazepam and diazepam, the finding that lorazepam impaired priming in this experiment is also consistent with the suggestion of differential benzodiazepine effects, that is lorazepam impairs priming but diazepam preserves it (Danion et al., 1992; Sellal et al., 1992).

It is important to note that in this experiment the degree of impairment to the priming effect was not as substantial as has been found in previous research. Those studies that had reported impaired priming with lorazepam found the priming effect for the lorazepam group comparable to baseline levels, or slightly above it (Brown et al., 1989; Danion et al., 1992; Sellal, et al., 1992). Baseline level is defined as the number of stems that are completed to form words that were not presented previously. Completing stems that form previously unpresented words is assumed to represent chance levels because each stem can complete a minimum of ten proper words. This assumption is

supported by the finding of baseline levels approximating ten percent consistently when stems that can form at least 10 words are used (Graf, Squire, Mandler, 1984; Graf & Schacter, 1986; Danion et al., 1992). In this experiment the lorazepam group completed 47% of all the stems that formed previously presented words whereas the baseline level was 12.5%. This suggests that under the conditions of this experiment lorazepam only partially impairs the priming effect.

The task requirements in this experiment differed from the majority of other studies examining the effects of benzodiazepines on priming effects. The words were presented twice, rather than just once. These task requirements more closely resemble those of studies which have found preserved priming in organic amnesia (Graf et al., 1984: Graf & Schacter, 1985). One explanation of partially impaired priming with these task requirements may be based on an activation approach. Processing of the word is assumed to result in an automatic activation of its pre-existing memory representation (Shimamura & Squire, 1986). Two presentations of the words in the orienting task may result in greater, or stronger, activation of the temporary representation, which may enhance priming.

An alternative explanation of partially impaired priming with lorazepam is that some explicit or declarative strategies are invoked in the word completion task (Shimamura, 1986; Tulving, Hayman, & MacDonald, 1991). Squire, Shimamura, and Graf (1987) concluded normal subjects were capable of utilising explicit memory strategies to facilitate word completion. It is possible that the task requirements of this study facilitated some explicit memory strategies. Subjects may have become

aware of the memory component to the task and explicitly attempted to recall the words that had been presented previously. Awareness of the purpose of the task may have resulted from prior exposure to the task in the pre-drug testing and the close proximity in time between presentation of the words and completion of the stems. The attempts to camouflage the purpose of the word completion task may have been insufficient. Two presentations of the words may have improved the effectiveness of the explicit strategies that were utilised.

The results from the present experiment are unclear in regards to the distinction between explicit and implicit memory functions (Graf & Schacter, 1984). It has been assumed that a distinction exists between explicit and implicit memory functions because explicit memory functions are impaired but implicit memory functions are preserved in organic amnesia (Graf et al., 1984; Squire & Shimamura, 1986) . In the current experiment explicit memory, indexed by performance on the delayed conditions of the free recall task was impaired and implicit memory, indexed by the priming effect task was partially impaired.

Findings that lorazepam impairs priming supports the notion that there are some differences between organic amnesia and benzodiazepine-induced amnesia (Brown et al., 1989). Although, findings of partially impaired priming suggest a difference between lorazepam-induced amnesia and organic amnesia this does not provide clear support for Brown et al., (1989) proposition of a double-dissociation. In this experiment delayed recall was impaired and partially impaired priming does

not indicate clearly that lorazepam-induced amnesia and organic amnesia are dissociable in regards to priming.

The main implications of this experiment, in regards to delayed recall, is that impaired delayed recall appears to start as early as one minute after presentation and reaches asymptote by three minutes. Future research needs to compare the degree of impairment at three, five , and 10 minutes directly to determine if the deficit does reach its peak within the first five minutes. In regards to priming, lorazepam appears to impair priming effects but only partially when the task requirements more closely resemble those used in the majority of studies on priming in organic amnesia. A number of methodological weaknesses in this study need to be addressed to confirm this. In order to substantiate that the task requirements influence priming, rather than utilisation of explicit strategies, it would be necessary to ensure subjects are not aware of the purpose of the task and administer the recall component of the priming task after the word completion task. In addition, different task requirements would need to be directly compared to determine the effects of task requirements upon the levels of priming effects. Considering that lorazepam and diazepam appear to have differential effects it is suggested that the different tasks are used compared directly the effects of these and other benzodiazepines.

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APPENDICES

APPENDIX A: Statement of informed consent

APPENDIX B: Test protocols

APPENDIX C: Table of means

APPENDIX D: Table of anova summaries

APPENDIX E: Raw data

APPENDIX A:

Statement of informed consent

CONSENT TO PARTICIPATE IN A LORAZEPAM RESEARCH PROJECT

DATE _____

SUBJECTS NAME _____ PHONE _____

EXPERIMENTER _____ PHONE _____

I, _____

OF, _____

Have been invited to participate in a research project involving "AN EXAMINATION OF COGNITIVE FUNCTIONS UNDER THE EFFECTS OF LORAZEPAM"

The purpose of this experiment is to investigate the effects of the minor tranquilizer Lorazepam upon your performance in a variety of simple cognitive tasks. The drug Lorazepam is a sedative, very similar to valium, and is used widely for anxiety, sleep disturbances, and in medical procedures. One effect of Lorazepam is to cause a temporary reduction in memory of events yet allow the person to remain awake. We are examining the effects of Lorazepam upon cognitive functioning in detail, hoping to cast light on disease which are not fully understood. The research involves the use of simple cognitive ability tests to measure changes caused by Lorazepam.

I have been informed of the following points,

1) Approval has been given by the Human research ethics committee.

2) The procedure will involve a number of simple cognitive tasks before receiving a tablet and another series of simple cognitive tasks. The simple cognitive tasks involve a number of short exercises with word lists, ratings of music passages, and recalling short lists of numbers. Subjects will be randomly assigned to two groups one who recieves Lorazepam the other who receives a placebo.

3) I have been informed that I MAY find the test stressful.

4) Should I develop a problem which I might suspect have resulted from my involvement in the study, I am aware that I should contact Dr K. Kirkby on 354885.

5) The results of any test or information regarding my test results will not be published in any way that could reveal my identitiy.

6) I have been given adequate opportunity to ask questions about this project and my involvement, and I know if I have any other questions I may contact the researcher Mr M. Stanton on 251680

7) I will be given a copy of this form to keep.

8) I UNDERSTAND I MAY WITHDRAW FROM THE EXPERIMENT AT ANY POINT AND WILL BE OFFERED A BED TO REST IN OR TRANSPORT HOME !

After considering all of these points, I accept the invitation to participate in this project

SIGNATURE: _____ DATE:_____

WITNESS SIGNATURE:_____

WITNESS NAME:_____

ADDRESS:_____

STATEMENT BY RESEARCHER

I have explained this trial and the implications of participation in it to this participant, and believe that he/she understands it, and that this consent is based on adequate information.

SIGNITURE:_____ DATE:_____

APPENDIX B:

Test protocols

Sedation rating questionnaire procedure

Sedation rating questionnaire

Digit span test instructions

Digit span test form

Priming effects task instructions

Semantic orienting task word list 1

Semantic orienting task word list 2

Word completion task word list 1

Word completion task word list 2

Semantic orienting task answer form

Immediate recall answer form

Word completion answer form

Delayed free recall task instructions

Delayed free recall word list order 1

Delayed free recall word list order 2

Delayed free recall word list order 3

Delayed free recall word list order 4

Delayed free recall task word list 1

Delayed free recall task word list 2

Delayed free recall task word list 3

Delayed free recall task word list 4

Delayed free recall task word list 5

Delayed free recall task word list 6

Delayed free recall task word list 7

Delayed free recall task word list 8

Musical passage rating questionnaire

Sedation rating questionnaire procedure

Subjects at the beginning and and of pre and post drug testing will be presented with a simple sedation rating questionnaire. The sedation rating questionnaire is a simple 5 point rating scale ranging from 'not tired at all' to 'extremely tired' and subjects will be asked to place a cross at the point that best expresses their feelings at that time.

Extremely Tired	Very Tired	Tired	A little Tired	Not tired at all

Definitions

Extremely Tired: You are finding it hard to remain awake. All you want to do is to go to sleep.

Very tired: You are finding it hard to concentrate and you would like to lie down for a while.

Tired: You feel sleepy but can continue the task quite well.

A little tired: You feel just a little fatigued but it doesnt effect what you are doing at all.

Not tired at all: You are fully awake.

Instructions

" Here is a line (pointing to the sedation rating scale) that at 1 end has 'extremely tired' and at the other end 'not tired at all'. Please place a cross somewhere along this line that best expresses the way you feel at the momment. For example if a was a little tired but was quite capable of continuing on with a task a would place a cross here (place a cross on the 'a little tired' point. Is that clear? (if the subject understands then proceed). Please place a cross on the line that best describes the way you feel now."

Sedation rating questionnaire

LORAZEPAM INDUCED AMNESIA RESEARCH

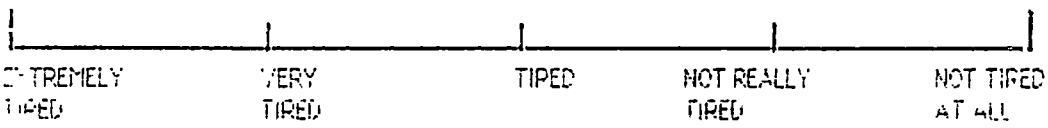
SEDATION RATING QUESTIONNAIRE

SUBJECT: _____

DATE: _____ SESSION: _____

TIME: _____

Please indicate how tired you feel at this moment, by placing a cross on the line.



DEFINITIONS

EXTREMELY TIRED: You are finding it hard to remain awake. All you want to do is go to sleep

VERY TIRED: You are finding it hard to concentrate and you would like to lie down for a while

TIRED: You feel fatigued

NOT REALLY TIRED: You feel a little fatigued but are still quite capable of continuing the task.

NOT TIRED AT ALL: You feel fully awake.

Digit span test instructions

The two parts of digit span- digits forward and digits backward- are administered seperately. Administer digits backward even if the subject scores 0 on digits forward. The digits should be given at the rate of one per second. Administer both trials of each item. Discontinue after failure on both trials of any item

Instructions

"I am going to say some numbers. Listen carefully, and when I am through say them right after me."

" Now I am going to say some more numbers, but this time when I stop I want you to say them backwards. For example, if I say 7-1-9, what would you say. (pause for subject to answer- if subject responds correctly say) thats right. (If the subject fails the example say) No, you would say 9-1-7. I said 7-1-9, so to say it backwards you would say 9-17. Now try these numbers. Remember, you are to say them backwards"

DIGIT SPAN TEST ANSWER FORM

SUBJECT ..... DATE: .....

SESSION

GIT SPAN	Discontinue after failure on BOTH TRIALS of any item. Administer BOTH TRIALS of each item, even if subject passes first trial.					
S FORWARD	Pass-Fail	Score 2, 1, or 0		DIGITS BACKWARD *	Pass-Fail	Score 2, 1, or 0
5 - 8 - 2			1.	2 - 4		
6 - 9 - 4				5 - 8		
6 - 4 - 3 - 9			2.	6 - 2 - 9		
7 - 2 - 8 - 6				4 - 1 - 5		
4 - 2 - 7 - 3 - 1			3.	3 - 2 - 7 - 9		
7 - 5 - 8 - 3 - 6				4 - 9 - 6 - 8		
6 - 1 - 9 - 4 - 7 - 3			4.	1 - 5 - 2 - 8 - 6		
3 - 9 - 2 - 4 - 8 - 7				6 - 1 - 8 - 4 - 3		
5 - 9 - 1 - 7 - 4 - 2 - 8			5.	5 - 3 - 9 - 4 - 1 - 8		
4 - 1 - 7 - 9 - 3 - 8 - 6				7 - 2 - 4 - 8 - 5 - 6		
5 - 8 - 1 - 9 - 2 - 6 - 4 - 7			6.	8 - 1 - 2 - 9 - 3 - 6 - 5		
3 - 8 - 2 - 9 - 5 - 1 - 7 - 4				4 - 7 - 3 - 9 - 1 - 2 - 8		
2 - 7 - 5 - 8 - 6 - 2 - 5 - 8 - 4			7.	9 - 4 - 3 - 7 - 6 - 2 - 5 - 8		
7 - 1 - 3 - 9 - 4 - 2 - 5 - 6 - 8				7 - 2 - 8 - 1 - 9 - 6 - 5 - 3		
Total Forward		Max=14		*Total Backward*		Max=14

DIGITS BACKWARD even if subject scores 0 on DIGITS FORWARD.

[ ] + [ ] = Max=28

Forward Backward Total

SESSION

IT SPAN	Discontinue after failure on BOTH TRIALS of any item. Administer BOTH TRIALS of each item, even if subject passes first trial.					
FORWARD	Pass-Fail	Score 2, 1, or 0		DIGITS BACKWARD *	Pass-Fail	Score 2, 1, or 0
5 - 8 - 2			1.	2 - 4		
6 - 9 - 4				5 - 8		
6 - 4 - 3 - 9			2.	6 - 2 - 9		
7 - 2 - 8 - 6				4 - 1 - 5		
4 - 2 - 7 - 3 - 1			3.	3 - 2 - 7 - 9		
7 - 5 - 8 - 3 - 6				4 - 9 - 6 - 8		
6 - 1 - 9 - 4 - 7 - 3			4.	1 - 5 - 2 - 8 - 6		
3 - 9 - 2 - 4 - 8 - 7				6 - 1 - 8 - 4 - 3		
5 - 9 - 1 - 7 - 4 - 2 - 8			5.	5 - 3 - 9 - 4 - 1 - 8		
4 - 1 - 7 - 9 - 3 - 8 - 6				7 - 2 - 4 - 8 - 5 - 6		
5 - 8 - 1 - 9 - 2 - 6 - 4 - 7			6.	8 - 1 - 2 - 9 - 3 - 6 - 5		
3 - 8 - 2 - 9 - 5 - 1 - 7 - 4				4 - 7 - 3 - 9 - 1 - 2 - 8		
2 - 7 - 5 - 8 - 6 - 2 - 5 - 8 - 4			7.	9 - 4 - 3 - 7 - 6 - 2 - 5 - 8		
7 - 1 - 3 - 9 - 4 - 2 - 5 - 6 - 8				7 - 2 - 8 - 1 - 9 - 6 - 5 - 3		
Total Forward		Max=14		*Total Backward*		Max=14

DIGITS BACKWARD even if subject scores 0 on DIGITS FORWARD

[ ] + [ ] = Max=28

Forward Backward Total

Priming effects task instructions

1. SEMANTIC ORIENTING TASK

Present the subjects the list of priming words, written on card, twice by placing the cards down at 1 per 5 seconds. Request the subjects to rate each word, on a 5 point scale on how much they liked the word on word rating questionnaire.

"I am going to place on the table a series of words, written on card, and I want you to rate each word, according to how much I you like or dislike the word, on the rating questionnaire for you. Rate the word by placing a cross on the line that best expresses your thoughts. Base your decision on any criteria, whether it be the sound of the word, the object is describes, or a memory that comes to mind. Do you understand? (If the subject understands proceed with the task)

Second presentation of priming list
" I am going to place the cards on the table as before. Please use the second rating questionnaire form and rate each word as you just have done. Are you ready? (If the subject is ready proceed with the task)

2 IMMEDIATE RECALL OF PRIMING LIST

immediately following the semantic orientation task request the subjects to write down as many of the words that were just presented to them as possible, in any order, on the Priming effect immediate free recall answer form..

" Please write down as many of the words from the words I just presented to you as you can, in any order. You have 1 minute, Go"

3. WORD COMPLETION TASK

Immediately following the Immediate free recall task request the subjects to complete 30 three letter word stems. The words will be presented 1 per 5 seconds, on card.

" I am now going to show you a series of 30 cards that has a word stem on them. A word stem is three letters that can be completed to form a word. Please complete the word stems by writing down the first word that comes to mind, whatever that may be. For example If I was to place this card (place the example card- MOT down) then I might write MOTEL or MOTHER. Remember to write the first word that comes to mind. Are you ready? (If the subject is ready then present the first card).

Semantic orienting task word list 1

WORD	LETTERS	KFF	CONCRETENESS
Mercy	5	20	239
Realm	5	19	303
Apple	5	09	611
Purse	5	14	572
Elbow	5	10	607
Panic	5	22	324
Laugh	5	28	433
Drama	5	43	375
Style	5	98	555
Frame	5	74	562
Money	6	265	574
Tiger	6	07	611
Circus	6	07	535
Deputy	6	17	455
Insect	6	14	593
Genius	6	23	342
Temple	6	38	565
Square	6	143	516
Capsule	7	05	540
Epitaph	7	04	449
Balloon	7	10	590
Tractor	7	24	590
Chicken	7	37	614
Picture	7	163	579
TOTAL		1093	12154
AVERAGE		**45.5**	**506.4**

NUMBER OF LETTERS PER WORD

LETTERS PER WORD	NUMBER OF WORDS
5	10
6	08
7	06

Semantic orienting task word list 2

WORD	LETTERS	KFF	CONCRETENESS
Quest	5	16	316
Array	5	11	371
Straw	5	15	603
Stove	5	15	591
Devil	5	25	274
Slave	5	30	539
Metal	5	61	582
Judge	5	77	506
Ankle	5	08	644
Plane	5	114	535
Glove	5	09	607
Brute	5	06	462
Value	5	200	260
Turtle	6	08	644
Summit	6	12	546
Carpet	6	13	581
Supper	6	37	563
Pencil	6	34	617
Friend	6	133	450
Monkey	6	09	566
Servant	7	19	515
Quarter	7	34	505
Balance	7	90	366
Captain	7	85	534
TOTAL		1066	12150
AVERAGE		**44.4**	**506.3**

NUMBER OF LETTERS PER WORD

LETTERS PER WORD	NUMBER OF WORDS
5	13
6	07
7	04

Word completion task word list 1

1. Nob... (Noble) (Baseline)
2. Stu... (Student) (Baseline)
3. Fin... (Finish) (Baseline)
4. Swe... (sweater) (Baseline)
5. Mel... (Melon) (Baseline)
6. Rec... (Record) (Baseline)
7. Dep... (Deputy)
8. Tem... (Temple)
9. Epi... (Epitaph)
10. App... (Apple)
11. Gal... (Galaxy) (Baseline)
12. Tra... (Tractor)
13. Pan.. (Panic)
14. Cir... (Circus)
15. Squ... (Square)
16. Dia... (Dialect) (Baseline)
17. Ins... (Insect)
18. Dra... (Drama)
19. Hon... (Honour) (Baseline)
20. Rea... (Realm)
21. Gen... (General)
22. Pic... (Picture)
23. Pur... (Purse)
24. Cas... (Casino) (Baseline)
25. Mod... (Modern) (Baseline)
26. Fra... (Frame)
27. Mer.. (Mercy)
28. Chi... (Chicken)
29. Sur... (surgeon) (Baseline)
30. Tig... (Tiger)

Word completion task word list 2

1. Bea... (Beard) (Baseline)
2. Bon... (Bonus) (Baseline)
3. Cho... (Chocolate) (Baseline)
4. Tab... (Table) (Baseline)
5. Aba... (Abate) (Baseline)
6. Che.. (Cheese) (Baseline)
7. Sup... (Supper)
8. Que... (Quest)
9. Dol... (Dolphin) (Baseline)
10. Ser... (Servant)
11. Enc... (Encore) (Baseline)
12. Swa... (Swamp) (Baseline)
13. Car... (Carpet)
14. Sum... (Summit)
15. Pen... (pencil)
16. Arr... (Array)
17. Pro... (provide) (Baseline)
18. Ton... (Tonic) (Baseline)
19. Glo... (Glove)
20. Sal... (Salad) (Baseline)
21. Jud... (Judge)
22. Ank... (Ankle)
23. Qua... (Quarter)
24. Met... (Metal)
25. Fri... (Friend)
26. Dev... (Devil)
27. Sla... (Slave)
28. Val... (Value)
29. Sto... (Stove)
30. Pla... (Plane)

Semantic orienting task answer form

LORAZEPAM INDUCED AMNESIA RESEARCH

WORD RATING QUESTIONNAIRE

SUBJECT: _____ DATE: _____

TIME: _____ SESSION: _____ WORD LIST: _____

Please rate the words presented infront of you on cards, according to how much you like it, by placing a cross on the line at the point that best describes your feelings

THE WORDS WILL BE PRESENTED EVERY 5 SECONDS SO MAKE YOUR RATING AS QUICK AS POSSIBLE

WORD 1 : _____

CONSIDERABLY DISLIKE	QUITE DISLIKE	LIKE	QUITE LIKE	CONSIDERABLY LIKE

WORD 2 : _____

CONSIDERABLY DISLIKE	QUITE DISLIKE	LIKE	QUITE LIKE	CONSIDERABLY LIKE

WORD 3 : _____

CONSIDERABLY DISLIKE	QUITE DISLIKE	LIKE	QUITE LIKE	CONSIDERABLY LIKE

WORD 4 : _____

CONSIDERABLY DISLIKE	QUITE DISLIKE	LIKE	QUITE LIKE	CONSIDERABLY LIKE

WORD 5 : _____

CONSIDERABLY DISLIKE	QUITE DISLIKE	LIKE	QUITE LIKE	CONSIDERABLY LIKE

WORD 6 : _____

CONSIDERABLY DISLIKE	QUITE DISLIKE	LIKE	QUITE LIKE	CONSIDERABLY LIKE

WORD 7 : _____

CONSIDERABLY DISLIKE	QUITE DISLIKE	LIKE	QUITE LIKE	CONSIDERABLY LIKE

WORD 8 : _____

CONSIDERABLY DISLIKE	QUITE DISLIKE	LIKE	QUITE LIKE	CONSIDERABLY LIKE

WORD 9 : _____

CONSIDERABLY DISLIKE	QUITE DISLIKE	LIKE	QUITE LIKE	CONSIDERABLY LIKE

WORD 10 : _____

CONSIDERABLY DISLIKE	QUITE DISLIKE	LIKE	QUITE LIKE	CONSIDERABLY LIKE

WORD 11 : _____

CONSIDERABLY DISLIKE	QUITE DISLIKE	LIKE	QUITE LIKE	CONSIDERABLY LIKE

WORD 12 : _____

CONSIDERABLY DISLIKE	QUITE DISLIKE	LIKE	QUITE LIKE	CONSIDERABLY LIKE

WORD 13 : _____

CONSIDERABLY DISLIKE	QUITE DISLIKE	LIKE	QUITE LIKE	CONSIDERABLY LIKE

WORD 14 : _____

CONSIDERABLY DISLIKE	QUITE DISLIKE	LIKE	QUITE LIKE	CONSIDERABLY LIKE

WORD 15 : _____

CONSIDERABLY DISLIKE	QUITE DISLIKE	LIKE	QUITE LIKE	CONSIDERABLY LIKE

WORD 16 : _____

CONSIDERABLY DISLIKE	QUITE DISLIKE	LIKE	QUITE LIKE	CONSIDERABLY LIKE

WORD 17 : _____

CONSIDERABLY DISLIKE	QUITE DISLIKE	LIKE	QUITE LIKE	CONSIDERABLY LIKE

WORD 18 : _____

CONSIDERABLY DISLIKE	QUITE DISLIKE	LIKE	QUITE LIKE	CONSIDERABLY LIKE

WORD 19 : _____

CONSIDERABLY DISLIKE	QUITE DISLIKE	LIKE	QUITE LIKE	CONSIDERABLY LIKE

WORD 20 : _____

CONSIDERABLY DISLIKE	QUITE DISLIKE	LIKE	QUITE LIKE	CONSIDERABLY LIKE

WORD 21 : _____

CONSIDERABLY DISLIKE	QUITE DISLIKE	LIKE	QUITE LIKE	CONSIDERABLY LIKE

WORD 22 : _____

CONSIDERABLY DISLIKE	QUITE DISLIKE	LIKE	QUITE LIKE	CONSIDERABLY LIKE

WORD 23 : _____

CONSIDERABLY DISLIKE	QUITE DISLIKE	LIKE	QUITE LIKE	CONSIDERABLY LIKE

WORD 24: _____

CONSIDERABLY DISLIKE	QUITE DISLIKE	LIKE	QUITE LIKE	CONSIDERABLY LIKE

PRIMING EFFECT TASK IMMEDIATE FREE RECALL ANSWER FORM

SUBJECT: _____ DATE _____

SESSION: _____ WORD LIST NUMBER .....

	correct
1.	_____
2.	_____
3.	_____
4.	_____
5.	_____
6.	_____
7.	_____
8.	_____
9.	_____
10.	_____
11.	_____
12.	_____
13.	_____
14.	_____
15.	_____
16.	_____
17.	_____
18.	_____
19.	_____
20.	_____
21.	_____
22.	_____
23.	_____
24.	_____

PRIMING WORD COMPLETION TASK

SUBJECT: _____ DATE _____

SESSION: _____ WORD LIST NUMBER .....

Please write down the completed word in the space provided, for each of the 3 letter stems that are presented.

1.	16. .....
2.	17. .....
3.	18. .....
4.	19. .....
5.	20. .....
6.	21. .....
7.	22. .....
8.	23. .....
9.	24. .....
10.	25. .....
11.	26. .....
12.	27. .....
13.	28. .....
14.	29. .....
15.	30. .....

Delayed free recall task instructions

Verbally present the subject 4 lists of 12 words, at a rate of 1 word per second, and request the subject to recall the word list, in any order immediately, 1 minute, 3 minutes, and 5 minutes after presentation. 4 word lists will be used used with parallel forms, and the order of recall delay was counterbalanced with subjects recalling in either one of the following four orders,

1. immediate recall, 1 minute delay, 3 minute delay, 5 minute delay
2. 1 minute delay, 3 minute delay, 5 minute delay, immediate recall
3. 3 minute delay, 5 minute delay, immediate recall, 1 minute delay
4. 5 minute delay. immediate recall, 1 minute delay, 3 minute delay

(see DELAYED RECALL WORD LIST PROTOCOLS)

Inform the subjects they are to be presented a number of musical passages and when they are finished to rate them according to how much they liked them. Inform them they will be presented with a number of simple cognitive tasks to do in-between the music passages.

Initial Instructions

" I am going to play a number of musical passages and the passage finishes I want you to rate each one on how much you liked it on this rating form (present musical rating questionnaire form). Rate the music passage by placing a circle around the phrase that best describes the way you feel about the passage. Please rate the passage within 2-3 seconds of the passage finishing. In-between the music passages I will give you a number of short cognitive tasks to do. As soon as you have finished the cognitive tasks I will play another piece of music for you to rate"

Word list presentation instructions

" I am now going to read you a list of words. Please concentrate on the list and tell me if I repeat any of the words"

Word list recall Instructions

" Please write down as many of the words in the last list of words I presented, as you can recall, in any order. You have 1 minute, Go !"

Delayed free recall word list order 1

ITEM	TIME
Present Word List 1	.30
Recall Word list1	1.00
Music Passage1	1.00
Present Word List 2	.30
Music Passage 2	1.00
Recall Word List 2	.30
Music Passage 3	1.00
Present Word List 3	.30
Music Passage 4	3.00
Recall word List 3	1.00
Music Passage 5	1.00
Present Word List 4	.30
Music Passage 6	5.00
Recall word List 4	1.00
TOTAL TIME	18.00

Delayed free recall word list order 2

ITEM	TIME
Present Word List 2	.30
Music Passage 2	1.00
Recall Word List 2	.30
Music Passage1	1.00
Present Word List 3	.30
Music Passage 4	3.00
Recall word List 3	1.00
Music Passage 5	1.00
Present Word List 4	.30
Music Passage 6	5.00
Recall word List 4	1.00
Music Passage 3	1.00
Present Word List 1	.30
Recall Word list1	1.00
TOTAL TIME	18.00 MINUTES

Delayed free recall word list order 3

ITEM	TIME
Present Word List 3	.30
Music Passage 4	3.00
Recall word List 3	1.00
Music Passage1	1.00
Present Word List 4	.30
Music Passage 6	5.00
Recall word List 4	1.00
Music Passage 3	1.00
Present Word List 1	.30
Recall Word list1	1.00
Music Passage 5	1.00
Present Word List 2	.30
Music Passage 2	1.00
Recall Word List 2	.30
TOTAL TIME	18.00 MINUTES

Delayed free recall word list order 4

ITEM	TIME
Present Word List 4	.30
Music Passage 6	5.00
Recall word List 4	1.00
Music Passage 1	1.00
Present Word List 1	.30
Recall Word list1	1.00
Music Passage 2	1.00
Present Word List 2	.30
Music Passage 3	1.00
Recall Word List 2	.30
Music Passage 5	1.00
Present Word List 3	.30
Music Passage 4	3.00
Recall word List 3	1.00
TOTAL TIME	18.00 MINUTES

Delayed free recall task word list 1

WORD	LETTERS	KFF	FAMILIARITY	CONCRETENESS
1. Husband	7	131	557	549
2. Wood	4	55	574	606
3. Column	6	71	519	520
4. Disease	7	53	580	505
5. Page	4	66	603	571
6. Hair	4	148	575	583
7. Ground	6	186	574	558
8. Train	5	82	584	592
9. Smile	5	58	594	514
10. Cattle	6	97	511	600
11. Bank	4	148	575	583
12. Spoke	5	87	532	526
TOTAL		1116	67759	6697
AVERAGE		**93.1**	**564.7**	**558.1**

Delayed free recall task word list 2

WORD	LETTERS	KFF	FAMILIARITY	CONCRETENESS
1. Camp	4	75	541	571
2. Circle	6	60	581	587
3. Father	6	183	591	594
4. Wine	4	72	570	621
5. Teacher	7	80	599	569
6. Step	4	131	578	508
7. Wind	4	63	592	552
8. Novel	5	59	530	529
9. Corner	6	115	556	553
10. Wheel	5	56	566	573
11. Horse	5	117	560	613
12. Concern	7	98	519	509
TOTAL		1109	678	6779
AVERAGE		**92.4**	**565.3**	**564.9**

Delayed free recall task word list 3

WORD	LETTERS	KFF	FAMILIARITY	CONCRETENESS
1. Foot	4	70	583	558
2. Island	6	167	507	596
3. Band	4	53	555	590
4. Dust	4	70	588	550
5. Engine	6	50	543	586
6. Phone	5	54	550	624
7. Spring	6	127	588	524
8. Lake	4	54	583	585
9. Officer	7	101	549	550
10. Mouth	5	103	572	568
11. Chest	5	53	543	580
12. Music	5	125	592	594
TOTAL		1117.9	6760	6823
AVERAGE		**93.2**	**563.3**	**568.6**

Delayed free recall task word list 4

WORD	LETTERS	KFF	FAMILIARITY	CONCRETENESS
1. Shore	5	61	531	574
2. Glass	5	99	611	635
3. Moon	4	60	585	581
4. Throat	6	51	548	578
5. Gold	4	52	550	576
6. Child	5	213	585	581
7. Village	7	72	524	576
8. Student	7	162	597	579
9. Test	4	119	566	520
10. Uncle	5	57	557	580
11. Bridge	6	98	561	623
12. Pool	4	111	541	573
TOTAL		1107	6722	6996
AVERAGE		**92.3**	**560.2**	**581.3**

Delayed free recall task word list 5

WORD	LETTERS	KFF	FAMILIARITY	CONCRETENESS
1. Machine	7	103	549	578
2. Desk	4	65	583	583
3. Record	6	137	609	558
4. Rock	4	75	583	600
5. Army	4	132	555	543
6. Staff	5	113	577	515
7. Picture	7	162	591	579
8. Motor	5	56	545	565
9. Block	5	66	544	558
10. Knife	5	76	573	612
11. Ship	4	83	553	615
12. Cousin	6	51	515	502
TOTAL		1118	6783	6808
AVERAGE		**93.2**	**565.3**	**567.3**

Delayed free recall task word list 6

WORD	LETTERS	KFF	FAMILIARITY	CONCRETENESS
1. Bedroom	7	52	646	615
2. Cross	5	55	525	514
3. Animal	6	68	620	587
4. Park	4	94	571	579
5. Battle	6	87	537	564
6. Pick	4	55	524	502
7. Grass	5	53	587	599
8. Mother	6	216	632	579
9. Neck	4	81	576	587
10. Brother	7	73	598	585
11. Dance	5	90	550	502
12. Club	4	145	533	509
TOTAL		1126	6899	6722
AVERAGE		93.8	574.9	560.2

Delayed free recall task word list 7

WORD	LETTERS	KFF	FAMILIARITY	CONCRETENESS
1. Cover	5	88	597	502
2. Blood	5	121	571	613
3. Doctor	6	100	573	575
4. Coast	5	61	541	562
5. Sign	4	94	543	520
6. Food	4	147	579	597
7. Market	6	155	518	551
8. Station	7	105	548	572
9. Artist	6	57	547	554
10. Beach	5	61	553	612
11. Nose	4	60	584	628
12. Roof	4	59	552	586
TOTAL		1108	6670	6872
AVERAGE		**92.4**	**558.8**	**572.7**

Delayed free recall task word list 8

WORD	LETTERS	KFF	FAMILIARITY	CONCRETENESS
1. Floor	5	158	551	559
2. Person	6	175	620	562
3. Baby	4	62	597	589
4. Valley	6	73	515	575
5. Crowd	5	53	523	546
6. Product	7	87	862	516
7. Ball	4	110	575	615
8. Dress	5	67	588	595
9. Column	6	71	519	520
10. Library	7	62	580	564
11 Lead	4	129	526	543
12. Lady	4	80	573	564
TOTAL		1126	6729	6748
AVERAGE		**93.8**	**560.7**	**562.3**

Musical passage rating questionnaire

lorazepam-induced amnesia research

MUSICAL PASSAGE RATING QUESTIONNAIRE

SUBJECT ..... DATE ..... SESSION .....

Please listen to the musical passsages presented to you. For each passage place a cross on the line that best describes the way you felt about the passage. Please spend only a few seconds making your decision.

Music passage 1 .....

liked a lot	liked a little	O.K.	disliked a little	disliked a lot

Music passage 2 .....

liked a lot	liked a little	O.K.	disliked a little	disliked a lot

Music passage 3 .....

liked a lot	liked a little	O.K.	disliked a little	disliked a lot

Music passage 4 .....

liked a lot	liked a little	O.K.	disliked a little	disliked a lot

Music passage 5 .....

liked a lot	liked a little	O.K.	disliked a little	disliked a lot

Music passage 6 .....

liked a lot	liked a little	O.K.	disliked a little	disliked a lot

APPENDIX C:

Table of means

TABLE: Means and standard deviations of scores in results section for placebo ad lorazepam group.

	Placebo group		Lorazepam group	
	M	sd	M	sd
Sedation				
pre- beginning	3.3	0.8	3.7	0.8
pre- end	3.2	0.8	3.4	0.8
pre- combined	6.5	1.5	7.1	1.3
post- beginning	3.2	0.9	3.1	1.3
post- end	3.6	0.9	2.8	1.4
post- combined	6.8	1.7	5.7	2.4
Digit span				
pre	17.9	4.2	16.1	3.2
post	17.2	3.3	15.2	2.8
Immediate recall				
Pre	10.5	2.5	11.3	2.8
post	9.7	2.5	6.2	2.6
Word Completion				
Pre-total completed	13.6	3.3	11.8	3.8
pre-primed completed	11.4	3.2	10.3	3.5
pre-baseline completed	2.2	1.3	1.4	2.1
pre-primed recalled	5.0	2.1	4.4	2.1
Post-total completed	13.1	3.2	9.7	3.5
post-primed completed	11.4	3.0	8.2	3.3
post-baseline completed	1.7	1.2	1.5	0.9
post-primed recalled	4.3	2.1	2.3	2.1
Delayed free recall				
pre-immediate	5.4	1.8	5.6	1.8
pre-1 minute	5.3	2.5	3.3	2.1
pre-3 minutes	5.1	2.4	4.5	2.8
pre-5 minutes	2.9	1.5	3.4	1.5
post-immediate	6.3	2.0	5.4	1.8
post-1 minute	5.3	2.9	3.2	1.8
post-3 minutes	5.1	2.6	1.6	1.4
post-5 minutes	4.4	2.0	1.8	1.9
Serial position curve				
Immediate-primacy	2.1	1.2	1.6	1.1
Immediate-asymptote	1.7	1.2	1.1	1.3
Immediate-recency	2.5	1.3	2.5	1.0
1 minute-primacy	2.5	1.3	1.0	1.0
1 minute-asymptote	1.6	1.3	0.9	0.8
1 minute-recency	1.5	1.0	1.2	1.0
3 minute-primacy	2.3	1.2	0.8	0.9
3 minute-asymptote	1.5	1.3	0.2	0.4
3 minute-recency	1.3	1.8	0.5	0.7
5 minute-primacy	1.9	1.4	0.7	1.0
5 minute-asymptote	1.1	1.9	0.5	0.9
5 minute-recency	1.3	0.9	0.4	0.6

APPENDIX D:

Table of anova summaries

Note
d = group (d1= lorazepam, d2= placebo)
t = time (t1= pretest, t2= post-test)

1. Anova summary table for sedation questionnaire

2. Anova simple effects summary table for sedation questionnaire

3. Anova summary table for digit span test

4. Anova summary table for immediate recall task (pre-drug test session)

5. Anova summary table for immediate recall task (post-drug test session)

6. Anova summary table for word completion task (pre-drug test session)

7. Anova summary table for word completion task

8. Anova simple effects summary table for word completion task

9. Anova summary table for Delayed free recall task

10. Anova summary table for Delayed free recall task (pre-drug test sesion)

11. Anova simple effects summary table for delayed free recall task (pre-drug test sesion)

12. Anova summary table for Delayed free recall task (post-drug test sesion)

13. Anova simple effects summary table for delayed free recall task (post-drug test sesion)

14. Anova summary table for serial position curve in the delayed free recall task

15. Anova simple effects summaruy table for serial position curve in delayed free recall task (post drug test session)

1. Anova summary table for sedation questionnaire

Source of Variation	df	Sum of Squares	Mean Squares	F	P	Epsilon Correction
d	1	2.480	2.840	.492	.4885	
Error	31	151.270	5.042			
t	1	3.223	3.223	2.375	.1338	
dt	1	12.223	12.223	9.007	.0054	
Error	31	40.714	1.357			1.00

2. Anova simple effects summary table for sedation questionnaire

Effect	Msn	dfn	dfe	Mse	F	p
d at t1	1.846	1	31	2.012	.917	.346
d at t2	12.858	1	31	4.387	2.931	.097
t at d1	1.286	1	31	1.357	.947	.338
t at d2	16.00	1	31	1.357	11.789	.002

3. Anova summary table for digit span test

Source of Variation	df	Sum of Squares	Mean Squares	F	P	Epsilon Correction
d	1	87.823	87.823	4.634	.0392	
Error	31	587	18.950			
t	1	12.449	12.449	4.402	.0441	
dt	1	2.691	2.691	.952	.3368	
Error	31	87.672	2.828			1.00

4. Anova summary table for immediate recall task (pre-drug test session)

Source of Variation	df	Sum of Squares	Mean Squares	F	P	Epsilon Correction
d	1	17.417	17.417	1.906	.1776	
error	31	247.067	9.136			
t	1	142.125	142.125	21.455	.0001	
dt	1	77.500	77.500	11.699	.0018	
error	31	198.734	6.624			1.00

5. Anova summary table for immediate recall task (post-drug test session)

Source of Variation	df	Sum of Squares	Mean Squares	F	P	Epsilon Correction
d	1	10.719	10.719	1.299	.2634	
error	31	247.5	8.25			
total	32	258.219				

6. Anova summary table for word completion task (pre-drug test session)

Source of Variation	df	Sum of Squares	Mean Squares	F	P	Epsilon Correction
d	1	84.198	84.198	11.211	.0022	
error	31	225.302		7.51		
total	32	309.5				

7. Anova summary table for word completion task

Source of Variation	df	Sum of Squares	Mean Squares	F	P	Epsilon Correction
d	1	34.934	34.934	2.206	.1476	
Error	31	491.006	15.839			
t	1	12.768	12.768	2.341	.1361	
dt	1	26.950	26.950	4.942	.0336	
Error	31		69.050	5.453		1.00

8. Anova simple effects summary table for word completion task

Effect	Msn	dfn	dfe	Mse	F	p
d at t1	.259	1	31	11.378	.023	.881
d at t2	61.625	1	31	9.914	6.216	.018
t at d1	1.200	1	31	5.453	.220	.642
t at d2	42.250	1	31	5.453	7.748	.009

9. Anova summary table for Delayed free recall task

Source of Variation	df	Sum of Squares	Mean Squares	F	P	Epsilon Correction
d	1	126.798	126.768	8.805	.0057	
Error	31	446.300	14.397			
t	1	17.285	17.285	4.457	.0429	
dt	1	38.194	38.194	9.849	.0037	
Error	31	120.22	3.878			1.00
i	3	223.497	74.499	28.842	.0000	
di	3	24.043	8.014	3.103	.0304	
Error	93	240.222	2.853			.86
ti	3	17.459	5.820	2.063	.1105	
dti	3	31.388	10.446	3.703	.0144	
error	93	262.367	2.821			.88

10. Anova summary table for Delayed free recall task (pre-drug test sesion)

Source of Variation	df	Sum of Squares	Mean Squares	F	P	Epsilon Correction
d	1	12.898	12.898	1.159	.2899	
Error	31	343.844	11.124			
i	3	120.021	40.007	14.169	.0000	
di	3	17.718	5.906	2.092	.1066	
Error	93	262.600	2.824			.96

11. Anova simple effects summary table for delayed free recall task (pre-drug test sesion)

Effect	Msn	dfn	dfe	Mse	F	p
d at i1	1.536	1	31	4.046	.380	.542
d at i2	23.645	1	31	6.395	3.698	.064
d at i3	4.400	1	31	6.804	.647	.427
d at i4	1.034	1	31	2.350	.440	.512
i at d1	28.106	3	93	2.824	9.945	.000
i at d2	16.778	3	93	2.824	5.942	.001

12. Anova summary table for Delayed free recall task (post-drug test sesion)

Source of Variation	df	Sum of Squares	Mean Squares	F	P	Epsilon Correction
d	1	152.065	152.065	21.265	.0001	
Error	31	221.678	7.151			
i	3	120.935	40.312	15.622	.0000	
di	3	37.663	12.554	4.865	.0035	
Error	93	239.989	2.581			.84

13. Anova simple effects summary table for delayed free recall task (post-drug test sesion)

Effect	Msn	dfn	dfe	Mse	F	p
d at i1	2.731	1	31	3.210	.851	.363
d at i2	28.171	1	31	3.979	7.080	.012
d at i3	97.698	1	31	4.039	24.188	.000
d at 14	61.127	1	31	3.665	16.681	.000
i at d1	4.911	3	93	2.581	1.903	.134
i at d2	52.259	3	93	2.581	20.251	.000

14. Anova summary tables for serial position curve in the delayed free recall task (post drug test session)

Immediate recall

Source of Variation	df	Sum of Squares	Mean Squares	F	P
d	1	3.712	3.712	2.98	.094
Error	31	38.611	1.246		
i	2	22.202	11.101	8.722	.0005
di	2	1.554	.777	.61	.5464
Error	62	78.911	1.273		

One minute recall

Source of Variation	df	Sum of Squares	Mean Squares	F	P
d	1	16.894	16.894	9.276	.0047
Error	31	56.459	1.821		
i	2	3.535	1.768	2.049	.1375
di	2	5.635	2.818	3.265	.0448
Error	62	53.496	.863		

Three minute recall

Source of Variation	df	Sum of Squares	Mean Squares	F	P
d	1	34.91	34.91	27.016	.0001
Error	31	40.059	1.292		
i	2	8.97	4.485	5.787	.005
di	2	1.644	.823	1.061	.3522
Error	62	48.052	.775		

Five minute recall

Source of Variation	df	Sum of Squares	Mean Squares	F	P
d	1	20.376	20.376	16.681	.0003
Error	31	37.867	1.222		
i	2	4.606	2.303	2.435	.0961
di	2	1.372	.686	.725	.4889
Error	62	58.689	.947		

15. Anova simple effects summaruy table for serial position curve in delayed free recall task (post drug test session)

Effect	Msn	df	Mse	F	p
Immediate recall					
primacy	40.178	1, 31	1.296	2.107	.1566
asymptote	43.111	1, 31	1.391	1.816	.1876
recency	34.233	1, 31	1.104	.008	.9283
One minute recall					
primacy	41.733	1, 31	1.346	13.073	.001
asymptote	35.378	1, 31	1.141	3.625	.0662
recency	32.844	1, 31	1.059	.747	.3939
Three minute recall					
primacy	33.433	1, 31	1.078	15.586	.0004
asymptote	26.844	1, 31	.866	16.242	.0003
recency	27.833	1, 31	.898	6.328	.0173
Five minute recall					
primacy	44.544	1, 31	1.437	8.352	.007
asymptote	34.233	1, 31	1.104	2.972	.0947
recency	17.778	1, 31	.573	11.273	.0021

APPENDIX E:

Raw data

SEDATION RATING QUESTIONNAIRE AND DIGIT SPAN TEST

CONTROL GROUP - MEMORY SPAN AND AWARENESS

SUBJECT NUMBER	DIGIT SPAN PRE-DRUG	DIGIT SPAN POST-DRUG	SEDATION RATING QUESTIONNAIRE PRE-BEGIN	PRE-END	POST-BEGIN	POST-END
6	22	19	3	2	4	4
8	21	20	3	3	3	3
9	18	17	3	3	3	3
11	23	21	2	2	2	2
12	17	19	4	4	4	5
15	20	21	2	2	2	3
16	11	13	4	3	4	5
17	16	17	2	3	2	3
18	11	13	3	4	4	4
22	18	18	4	4	4	4
29	23	21	4	4	3	4
31	16	17	4	4	4	4
34	23	18	4	4	3	4
35	12	11	4	4	4	4
24	19	13	3	3	2	2
TOTAL	270	258	50	50	50	56

EXPERIMENTAL GROUP - MEMORY SPAN AND AWARENESS

SUBJECT NUMBER	DIGIT SPAN PRE-DRUG	DIGIT SPAN POST-DRUG	SEDATION RATING QUESTIONNAIRE PRE-BEGIN	PRE-END	POST-BEGIN	POST-END
1	10	12	3	3	2	2
2	18	18	4	4	4	4
3	16	14	3	3	1	2
4	14	13	3	3	3	3
7	19	12	4	4	4	3
57	19	15	4	4	3	2
10	15	15	4	3	2	1
13	16	17	2	2	4	1
14	19	18	5	5	5	4
19	18	18	4	3	5	5
21	15	12	4	4	3	3
23	13	14	4	3	3	3
25	22	19	4	3	2	2
27	11	13	3	3	1	1
30	18	20	4	3	2	3
32	15	13	3	3	2	1
33			5	3	3	4
36			3	5	5	5
TOTAL	258	243	66	61	54	49

Priming effect task placebo group pre-drug session

	PRIMING EFFECT TASK												
												CONTROL GROUP	
SUBJECT NUMBER	WORD 1	WORD 2	WORD 3	WORD 4	WORD 5	WORD 6	WORD 7	WORD 8	WORD 9	WORD 10	WORD 11	WORD 12	WORD 13
6	0	0	0	0	0	1	0	1	1	0	1	0	0
8	1	1	0	1	0	0	1	0	0	1	0	0	1
9	1	0	1	0	0	0	1	1	0	1	0	1	0
11	0	1	1	1	1	0	1	0	0	0	0	1	0
12	1	0	0	1	0	1	1	1	0	0	1	1	1
15	0	0	0	1	0	1	0	1	0	0	1	0	0
16	1	1	1	0	1	0	0	1	1	1	1	0	0
17	1	1	3	0	1	1	0	1	0	0	1	0	0
18	1	0	0	1	0	0	1	0	0	0	1	0	0
22	1	0	1	1	0	0	0	1	0	1	1	1	1
29	0	0	1	0	0	0	0	0	0	0	0	1	0
31	0	0	0	1	1	0	0	0	0	1	0	0	0
34	0	0	0	1	1	0	0	1	0	1	1	0	0
35	0	0	0	0	0	1	1	1	0	1	0	1	1
24	0	0	0	0	0	1	1	1	0	1	0	0	1
TOTAL	7	4	6	8	5	6	7	10	2	8	8	6	5

PRE-DRUG TESTING												TOTAL WORDS
WORD 14	WORD 15	WORD 16	WORD 17	WORD 18	WORD 19	WORD 20	WORD 21	WORD 22	WORD 23	WORD 24	TOTAL	COMPLETED
1	0	0	0	0	0	1	0	1	1	0	8	13
0	0	0	1	1	1	1	0	0	1	1	12	12
0	0	1	0	0	1	1	0	0	0	1	10	13
0	1	0	1	1	1	0	0	0	0	1	11	17
0	0	0	1	1	0	1	0	0	1	1	13	9
1	0	1	0	1	1	1	0	0	0	1	10	19
0	0	1	0	0	1	0	1	1	1	0	13	14
0	0	0	0	1	0	1	0	0	1	0	10	14
0	0	0	1	1	0	1	0	0	1	0	8	14
1	0	1	0	0	0	1	0	0	1	1	13	10
0	1	0	1	1	0	0	0	0	0	1	6	18
0	0	0	1	0	1	1	0	0	1	1	8	16
1	1	1	0	1	1	1	1	1	1	1	15	13
1	1	0	0	0	0	0	0	0	1	1	10	8
1	1	1	1	1	1	1	1	1	1	1	18	5
6	5	6	7	9	8	11	3	4	11	11	165	195

TARGET WORDS COMPLETED	BASELINE WORDS COMPLETED	RECALLED TARGET WDS COMPLETED	NON - RECALLED TARGET WDS COMPLETED
9	4	2	7
11	1	6	5
11	2	7	4
14	3	5	9
8	1	4	4
18	1	8	10
13	1	4	9
13	1	7	6
10	4	5	5
9	1	6	3
14	4	1	13
13	3	5	8
11	2	7	4
5	3	2	3
4	1	4	0
163	32	73	90

Priming effect task lorazepam group pre-drug session

	PRIMING EFFECT TASK												
												EXPERIMENTAL GR	
SUBJECT NUMBER	WORD 1	WORD 2	WORD 3	WORD 4	WORD 5	WORD 6	WORD 7	WORD 8	WORD 9	WORD 10	WORD 11	WORD 12	WORD 13
1	0	1	1	1	0	0	0	0	0	0	1	0	0
2	1	1	1	0	0	0	0	1	0	0	1	1	1
4	0	0	0	1	0	0	1	0	0	1	1	1	1
5	0	0	0	0	0	0	1	0	0	0	1	0	0
7	0	0	0	1	1	0	0	0	1	1	1	0	0
10	0	0	0	0	0	1	1	1	1	0	1	1	1
13	0	0	0	0	0	0	1	0	0	1	0	0	0
14	1	1	1	1	1	0	0	1	0	1	1	1	1
19	1	1	1	0	0	0	1	1	1	0	1	1	1
21	0	0	0	0	0	0	0	0	0	1	1	0	0
23	0	0	0	1	0	1	1	1	0	1	1	0	1
25	0	0	0	0	0	0	1	0	0	0	1	1	0
27	1	0	1	0	0	0	1	0	0	0	1	0	0
30	1	0	0	0	0	0	1	1	0	1	1	0	1
32	0	0	1	0	1	1	0	0	0	1	0	1	0
33	1	0	0	0	1	1	1	1	0	1	0	1	1
36	1	0	0	1	0	0	0	1	0	1	1	1	1
TOTAL	7	4	6	6	4	4	10	8	3	10	14	9	9

UP - PRE-DRUG TESTING											TOTAL WORDS
WORD 14	WORD 15	WORD 16	WORD 17	WORD 18	WORD 19	WORD 20	WORD 21	WORD 22	WORD 23	WORD 24	TOTAL	COMPLETED
1	0	0	1	1	1	1	0	1	1	0	11	13
0	0	0	1	0	1	0	0	0	1	1	11	6
0	0	1	0	0	1	0	0	0	0	0	8	12
1	1	1	1	0	0	1	1	0	1	1	10	14
0	0	0	1	1	0	1	1	0	1	1	11	17
1	0	0	0	1	1	1	0	1	1	1	14	15
0	0	0	0	1	1	0	0	0	1	1	6	12
0	0	1	0	1	1	1	0	1	1	1	17	14
1	0	1	0	0	0	1	1	0	1	1	15	13
0	1	1	1	1	0	1	0	0	1	1	9	11
1	0	0	1	0	0	1	0	0	0	1	11	18
0	1	0	0	1	1	1	0	0	1	1	9	11
1	0	1	1	0	1	1	0	0	0	1	10	10
1	0	0	1	0	0	0	1	0	1	1	11	7
1	1	0	0	1	1	1	1	1	1	1	15	8
0	1	1	0	0	0	1	0	0	1	1	13	17
1	0	0	0	0	0	1	0	0	1	1	11	9
9	5	7	8	8	9	13	5	4	14	15	192	207

TARGET WORDS COMPLETED	BASELINE WORDS COMPLETED	RECALLED TARGET WDS COMPLETED	NON-RECALLED TARGET WDS COMPLETED
10	3	5	5
6	0	2	4
10	2	4	6
12	2	6	6
16	1	6	10
14	1	8	6
12	0	3	9
11	3	5	6
11	2	4	7
10	1	2	8
16	2	7	9
9	2	2	7
9	1	3	6
6	1	2	4
7	1	4	3
15	2	9	6
8	1	4	4
182	25	76	106

Priming effect task placebo group post-drug session

	PRIMING EFFECT TASK												
												CONTROL GROUP	
SUBJECT NUMBER	WORD 1	WORD 2	WORD 3	WORD 4	WORD 5	WORD 6	WORD 7	WORD 8	WORD 9	WORD 10	WORD 11	WORD 12	WORD 13
6	0	1	0	1	0	0	0	0	0	1	0	1	0
8	1	1	0	0	1	1	0	1	0	0	1	0	1
9	0	0	1	1	0	1	0	0	0	0	0	0	1
11	0	0	0	1	0	1	1	0	1	0	0	1	1
12	1	1	0	0	0	0	1	0	0	1	1	1	1
15	0	0	0	0	0	0	1	1	1	1	1	0	0
16	0	0	0	0	0	0	1	1	1	1	1	0	0
17	0	0	0	1	0	0	1	1	0	1	0	0	0
18	0	1	0	0	0	0	0	0	0	0	1	0	0
22	0	0	0	0	0	0	0	0	0	0	0	0	1
29	0	0	0	1	0	0	0	1	1	0	1	1	1
31	0	1	0	0	0	0	0	1	0	0	0	0	0
34	0	0	0	1	0	0	0	1	0	1	1	0	0
35	0	0	0	1	0	0	0	0	0	0	0	0	0
24	0	0	1	1	0	0	0	1	0	1	1	0	0
TOTAL	2	6	2	8	1	3	4	8	3	6	8	5	6

POST-DRUG TESTING												TOTAL WORDS
WORD 14	WORD 15	WORD 16	WORD 17	WORD 18	WORD 19	WORD 20	WORD 21	WORD 22	WORD 23	WORD 24	TOTAL	COMPLETED
0	1	1	0	0	0	1	0	0	0	1	8	16
1	1	1	1	0	0	1	0	0	1	1	14	13
1	0	0	1	1	0	0	0	1	1	1	10	13
0	1	1	0	0	0	1	1	0	1	1	12	13
1	1	0	1	0	0	1	0	0	0	0	11	7
1	0	1	1	0	1	1	0	0	1	0	11	18
1	0	1	1	0	1	1	0	0	1	0	11	18
0	1	1	1	0	0	1	1	0	1	1	11	15
1	0	0	0	0	0	1	1	0	0	0	5	9
0	0	0	1	1	1	0	1	1	1	0	7	12
1	1	0	0	0	1	1	0	0	1	1	12	12
1	1	1	0	0	1	1	1	0	0	1	9	10
0	0	0	1	0	1	1	0	0	1	1	9	15
0	1	1	0	0	1	0	0	0	1	0	6	12
0	1	0	1	1	0	1	1	0	1	1	12	4
8	9	7	9	5	5	12	7	3	10	11	138	184

TARGET WORDS COMPLETED	BASELINE WORDS COMPLETED	RECALLED TARGET WDS COMPLETED	NON-RECALLED TARGET WDS COMPLETED
12	4	4	8
10	3	6	4
11	2	4	7
12	1	6	6
6	1	1	5
17	1	7	10
17	1	7	10
13	2	5	8
9	0	2	7
9	3	2	7
11	1	5	6
10	0	5	5
12	3	5	7
10	2	1	9
3	1	0	3
157	27	55	102

Priming effect task lorazepam Group post-drug session

								PRIMING EFFECT TASK					
												EXPERIMENTAL GR	
SUBJECT NUMBER	WORD 1	WORD 2	WORD 3	WORD 4	WORD 5	WORD 6	WORD 7	WORD 8	WORD 9	WORD 10	WORD 11	WORD 12	WORD 13
1	0	0	1	0	0	0	0	1	1	0	1	0	0
2	1	1	1	0	0	0	0	1	0	0	0	0	0
4	0	0	0	1	0	1	0	0	0	0	1	0	0
5	0	0	1	0	0	0	0	0	1	1	1	0	1
7	0	0	1	0	0	0	0	0	0	0	0	0	0
10	1	0	1	0	0	1	0	0	0	0	1	1	1
13	1	1	0	0	0	0	0	1	0	1	1	0	0
14	0	0	0	0	0	0	0	1	1	0	1	0	0
19	0	0	0	0	0	0	0	0	0	0	0	0	0
21	1	0	0	0	1	1	0	0	0	1	0	1	0
23	0	0	0	0	1	0	1	1	0	0	0	0	0
25	0	0	1	0	0	0	1	0	0	1	0	0	0
27	0	1	0	0	0	0	0	0	0	0	0	1	0
30	0	0	0	0	0	0	0	0	0	0	0	1	0
32	0	0	0	0	0	0	0	1	0	0	0	0	0
33	0	0	0	0	0	0	0	0	0	0	0	0	0
36	0	0	0	0	0	0	0	0	0	0	1	0	0
TOTAL	4	3	6	1	2	3	2	6	3	4	7	4	2

UP - POST-DRUG TESTING												TOTAL WORDS
WORD 14	WORD 15	WORD 16	WORD 17	WORD 18	WORD 19	WORD 20	WORD 21	WORD 22	WORD 23	WORD 24	TOTAL	COMPLETED
0	0	1	0	0	0	1	0	1	0	0	7	13
1	0	0	0	0	0	1	0	0	0	0	6	9
1	0	1	1	0	1	1	0	0	0	0	8	10
0	1	0	0	0	1	1	0	0	1	0	9	8
0	0	1	0	0	0	0	0	0	1	1	3	11
1	0	0	1	0	0	1	1	1	1	0	12	20
0	0	0	0	0	0	0	0	0	1	1	7	12
0	1	0	1	0	0	1	0	1	0	0	7	12
0	0	0	1	0	0	0	0	1	1	0	3	11
1	0	1	0	0	0	0	0	1	0	1	9	9
0	1	0	0	1	0	0	0	0	0	0	5	10
1	0	1	0	0	0	0	0	0	1	1	7	7
0	1	0	0	0	0	0	0	0	1	1	5	5
0	0	0	0	0	0	0	1	0	1	0	3	6
0	1	0	1	1	1	0	0	1	0	1	7	9
0	0	0	0	1	0	1	0	0	1	0	3	10
0	0	0	0	0	1	1	0	0	0	0	3	8
5	5	5	5	4	4	7	2	6	8	6	104	170

TARGET WORDS COMPLETED	BASELINE WORDS COMPLETED	RECALLED TARGET WDS COMPLETED	NON-RECALLED TARGET WDS COMPLETED
11	2	1	10
8	1	2	6
9	1	4	5
7	1	2	5
8	3	2	6
17	3	9	8
11	1	2	9
11	1	2	9
9	2	1	8
8	1	5	3
8	2	3	5
4	3	1	3
4	1	1	3
5	1	1	4
9	0	3	5
8	2	0	8
8	1	2	6
145	26	41	104

Delayed free recall task placebo group pre-drug session

DELAYED FREE RECALL TASK

CONTROL

PRE-DRUG TESTING - IMMEDIATE RECALL

SUBJECT NUMBER	WORD 1	WORD 2	WORD 3	WORD 4	WORD 5	WORD 6	WORD 7	WORD 8	WORD 9	WORD 10	WORD 11	WORD 12	TOTAL
6	0	0	0	0	1	0	0	0	1	1	1	1	5
8	1	1	0	1	1	0	1	0	0	0	0	1	6
9	1	0	0	0	0	0	0	0	0	1	0	1	3
11	1	1	0	0	1	0	0	0	0	1	0	0	4
12	1	0	1	0	0	0	0	1	1	0	1	1	6
15	0	0	0	0	0	0	0	1	0	1	0	1	11
16	1	1	1	1	0	0	0	1	0	1	1	1	8
17	1	1	1	0	0	0	0	0	0	0	1	1	5
18	1	0	0	0	0	0	1	0	0	1	0	1	4
22	1	1	1	1	0	0	0	1	0	0	0	0	5
29	1	1	1	1	1	0	0	0	0	0	0	0	5
31	1	1	1	1	0	0	0	1	1	0	0	1	7
34	1	1	0	1	0	0	0	0	0	1	0	0	4
35	1	1	1	1	0	0	0	0	1	1	0	1	7
24	1	1	1	1	0	1	0	1	0	1	1	1	9
TOTAL	14	11	9	9	5	2	3	6	5	8	6	11	

PRE-DRUG TESTING - 1 MINUTE DELAY

SUBJECT NUMBER	WORD 1	WORD 2	WORD 3	WORD 4	WORD 5	WORD 6	WORD 7	WORD 8	WORD 9	WORD 10	WORD 11	WORD 12	TOTAL
6	0	1	1	1	0	0	0	1	0	1	0	1	6
8	1	0	0	0	0	0	1	1	0	1	0	0	4
9	0	1	1	0	0	0	0	0	0	0	0	1	4
11	1	0	0	0	0	0	1	0	0	0	0	0	2
12	1	0	0	0	0	0	0	1	0	1	0	0	3
15	1	0	0	1	1	0	1	1	1	0	0	0	12
16	1	0	0	0	0	0	0	1	0	1	0	0	3
17	1	1	1	1	0	1	0	0	0	0	0	0	5
18	1	0	1	0	1	0	0	0	0	0	0	0	3
22	1	0	1	0	0	0	0	1	0	1	0	1	5
24	1	0	1	1	0	1	1	1	1	1	1	0	9
29	1	1	1	0	0	0	0	0	0	0	0	0	3
31	1	1	1	0	1	1	0	0	0	1	0	0	6
34	1	0	1	0	0	0	0	1	1	1	1	1	7
35	1	1	1	1	1	1	1	1	1	1	1	0	11
TOTAL	13	7	11	5	4	5	4	10	4	10	5	6	

PRE-DRUG TESTING - 3 MINUTE DELAY

SUBJECT NUMBER	WORD 1	WORD 2	WORD 3	WORD 4	WORD 5	WORD 6	WORD 7	WORD 8	WORD 9	WORD 10	WORD 11	WORD 12	TOTAL
6	0	1	1	0	0	0	0	0	0	1	0	0	3
8	1	0	1	0	0	0	1	0	0	1	0	0	4
9	1	0	0	0	0	0	0	0	0	0	0	0	1
11	1	0	0	0	0	0	0	0	0	1	0	0	2
12	0	1	0	0	0	0	1	0	1	0	1	1	5
15	1	1	0	1	1	1	1	1	1	1	1	0	10
16	0	1	1	1	0	0	0	0	0	1	0	0	4
17	1	0	1	0	0	1	1	0	1	0	0	1	6
18	1	0	0	0	0	0	1	0	0	0	1	0	3
22	1	0	1	1	0	0	0	0	0	1	1	1	6
24	1	1	1	1	1	0	1	1	0	1	0	0	8
29	1	1	0	0	0	0	0	0	1	0	1	1	5
31	1	1	0	0	0	0	1	1	1	1	0	1	7
34	0	1	1	1	1	0	0	0	0	0	1	0	5
35	1	1	1	1	0	0	1	0	1	1	0	0	7
TOTAL	11	9	8	6	3	2	8	3	6	9	6	5	

PRE-DRUG TESTING - 5 MINUTE DELAY

SUBJECT NUMBER	WORD 1	WORD 2	WORD 3	WORD 4	WORD 5	WORD 6	WORD 7	WORD 8	WORD 9	WORD 10	WORD 11	WORD 12	TOTAL
6	0	0	1	1	0	0	0	0	0	0	0	0	2
8	1	0	0	0	0	0	0	0	1	0	0	0	2
9	0	0	0	0	0	1	0	1	0	1	0	1	4
11	0	0	0	1	0	1	0	0	0	0	1	1	4
12	1	0	0	0	0	0	0	0	0	0	0	1	2
15	1	1	1	1	1	1	0	0	0	0	0	0	6
16	0	0	0	0	0	0	1	0	1	1	0	1	4
17	0	0	0	0	0	1	0	1	0	1	0	0	3
18	0	1	1	0	1	1	0	0	0	0	0	0	4
22	0	0	0	0	0	0	0	0	0	0	0	1	1
24	1	1	1	1	0	0	0	0	0	0	0	0	4
29	1	0	0	0	0	0	0	0	0	0	0	0	1
31	0	0	0	0	0	0	0	0	1	0	0	0	1
34	0	0	0	1	0	0	0	0	0	0	0	1	2
35	1	1	1	0	0	0	0	0	0	0	0	0	3
TOTAL	6	4	5	5	2	5	1	2	3	3	1	5	

Delayed free recall task lorazepam group pre-drug session

PRE-DRUG TESTING - IMMEDIATE RECALL

SUBJECT NUMBER	WORD 1	WORD 2	WORD 3	WORD 4	WORD 5	WORD 6	WORD 7	WORD 8	WORD 9	WORD 10	WORD 11	WORD 12	TOTAL
1	1	0	0	0	0	1	0	1	0	1	1	1	6
2	1	1	1	0	0	0	0	1	0	1	1	1	7
3	0	0	0	0	1	1	0	1	1	1	0	1	6
4	1	0	1	0	1	0	0	0	0	1	0	0	4
5	1	0	0	0	0	0	0	0	0	1	0	1	3
7	0	0	0	0	0	1	1	1	0	1	0	1	5
10	1	0	0	1	0	0	0	1	1	0	0	1	5
13	1	0	0	0	0	1	1	0	1	1	1	1	7
14	1	1	0	1	0	1	1	1	1	0	0	1	8
19	0	1	1	0	0	1	1	0	1	1	1	1	8
21	1	0	0	0	0	0	0	0	0	1	0	1	3
23	1	1	1	0	0	1	0	1	0	0	0	1	6
25	1	0	0	1	0	1	1	1	0	0	0	0	5
27	0	1	0	0	0	0	0	0	1	0	1	1	4
30	1	1	1	0	0	1	0	1	1	1	0	1	8
32	1	1	0	1	0	1	0	0	1	1	1	1	8
33	1	0	0	0	0	0	0	0	0	1	0	1	3
36	1	0	0	0	0	0	0	0	0	0	1	1	3
TOTAL	14	7	5	4	2	10	5	9	8	11	7	16	99

PRE-DRUG TESTING - 1 MINUTE DELAY

SUBJECT NUMBER	WORD 1	WORD 2	WORD 3	WORD 4	WORD 5	WORD 6	WORD 7	WORD 8	WORD 9	WORD 10	WORD 11	WORD 12	TOTAL
1	1	0	1	1	0	0	0	0	1	1	0	0	5
2	0	1	1	1	0	0	0	0	0	0	0	1	4
3	0	1	1	0	1	0	0	0	0	0	0	1	4
4	0	0	0	1	1	0	1	0	0	1	1	1	6
5	0	0	0	0	1	1	0	0	1	1	0	0	4
7	1	0	1	1	0	0	1	0	1	0	0	0	5
10	1	0	0	0	0	0	0	0	0	1	0	1	3
13	0	0	0	1	0	0	0	0	0	0	0	0	1
14	1	0	0	0	0	0	1	1	0	1	1	1	6
19	0	1	1	0	0	0	0	0	0	0	1	0	3
21	0	1	0	0	0	0	0	0	0	0	0	0	1
23	0	0	1	0	0	0	0	0	0	0	0	0	1
25	0	0	1	0	0	0	0	0	0	0	0	0	1
27	0	1	1	0	1	1	0	1	0	1	0	0	6
30	0	0	0	1	0	0	1	1	0	1	0	0	8
32	0	0	0	1	0	0	1	1	0	1	0	0	4
33	1	0	0	0	0	0	0	1	0	0	1	1	4
36	1	1	0	1	0	0	0	0	0	1	0	0	3
TOTAL	7	6	9	8	4	2	4	5	4	9	5	6	69

PRE-DRUG TESTING - 3 MINUTE DELAY

SUBJECT NUMBER	WORD 1	WORD 2	WORD 3	WORD 4	WORD 5	WORD 6	WORD 7	WORD 8	WORD 9	WORD 10	WORD 11	WORD 12	TOTAL
1	1	0	1	1	0	1	1	1	0	1	1	0	8
2	1	0	0	0	1	1	0	1	1	0	1	1	7
3	1	0	0	1	0	0	0	1	1	1	0	0	5
4	0	0	1	0	0	0	0	0	0	0	1	0	2
5	0	0	0	0	0	0	1	0	1	0	1	0	3
7	1	1	1	1	1	0	1	0	0	0	0	0	6
10	1	0	1	0	0	1	0	1	0	1	0	1	6
13	0	0	0	0	0	0	0	0	1	1	0	0	2
14	0	1	1	0	0	0	0	0	0	0	0	1	3
19	0	1	0	0	0	0	0	1	0	0	0	1	3
21	1	0	0	0	0	0	0	0	1	1	0	1	4
23	1	0	0	0	0	0	0	0	0	0	0	0	1
25	1	0	0	0	0	0	0	0	0	1	0	1	3
27	1	1	0	0	0	0	0	0	0	0	0	1	3
30	1	1	1	0	1	1	1	1	1	1	1	1	11
32	1	1	0	1	0	1	0	0	1	1	1	1	8
33	0	0	1	0	0	0	0	0	0	0	0	0	1
36	0	1	0	1	0	0	0	0	0	0	0	0	2
TOTAL	11	7	7	5	3	5	4	6	7	8	6	9	78

PRE-DRUG TESTING - 5 MINUTE DELAY

SUBJECT NUMBER	WORD 1	WORD 2	WORD 3	WORD 4	WORD 5	WORD 6	WORD 7	WORD 8	WORD 9	WORD 10	WORD 11	WORD 12	TOTAL
1	1	0	0	0	0	0	0	0	0	1	1	1	4
2	1	0	0	1	0	1	0	1	1	1	0	0	6
3	0	0	1	1	0	1	0	1	0	0	0	0	4
4	0	0	1	0	0	0	0	0	1	0	0	1	3
5	0	0	0	0	0	1	0	0	0	1	0	1	3
7	1	1	0	0	0	0	0	1	0	1	0	0	4
10	0	1	1	1	1	1	1	1	0	0	0	0	6
13	0	1	1	0	0	0	0	1	1	0	0	0	4
14	0	0	0	1	0	1	1	0	0	0	0	1	4
19	0	0	0	0	0	0	0	0	0	0	0	0	0
21	0	0	0	0	0	1	0	1	0	0	0	0	2
23	0	0	0	0	0	0	0	0	1	0	0	0	2
25	0	0	0	1	1	1	0	0	0	0	0	0	3
27	1	0	1	1	1	0	0	0	0	0	0	0	4
30	0	1	1	1	0	0	0	1	0	0	0	0	4
32	0	0	1	1	0	0	0	0	0	0	0	1	3
33	1	0	0	0	0	0	0	0	0	0	0	0	1
36	1	0	0	0	0	0	0	0	0	0	0	0	1
TOTAL	6	4	7	8	2	8	2	7	4	4	1	5	58

Delayed free recall task placebo group post-drug session

DELAYED FREE RECALL TASK — CONTROL GROUP

POST-DRUG TESTING - IMMEDIATE RECALL

SUBJECT NUMBER	WORD 1	WORD 2	WORD 3	WORD 4	WORD 5	WORD 6	WORD 7	WORD 8	WORD 9	WORD 10	WORD 11	WORD 12	TOTAL
6	1	0	0	0	0	1	1	1	0	1	0	1	6
8	1	1	0	1	0	0	0	1	0	1	0	1	6
9	0	0	0	0	1	0	0	1	1	0	1	1	5
11	0	0	0	1	0	0	0	0	1	1	1	1	5
12	1	0	1	0	0	1	0	0	0	1	1	1	6
15	1	1	1	1	1	1	1	1	1	0	1	1	3
16	1	1	1	1	0	0	0	1	0	1	1	1	8
17	1	0	0	0	1	1	1	1	1	0	0	0	6
18	0	1	0	1	0	0	0	0	0	0	0	0	2
22	1	0	0	0	1	1	0	0	1	0	1	1	6
24	1	1	0	0	1	0	0	0	0	1	1	1	6
29	1	1	1	0	0	1	1	0	0	1	1	0	7
31	1	0	1	1	0	0	1	1	1	1	1	1	9
34	1	1	0	1	0	0	1	0	0	1	1	1	7
35	1	0	1	0	0	1	0	0	1	0	0	1	5
TOTAL	11	6	5	6	4	6	5	7	6	10	9	12	

POST-DRUG TESTING - 1 MINUTE DELAY

SUBJECT NUMBER	WORD 1	WORD 2	WORD 3	WORD 4	WORD 5	WORD 6	WORD 7	WORD 8	WORD 9	WORD 10	WORD 11	WORD 12	TOTAL
6	0	0	0	0	0	0	0	0	0	1	0	1	2
8	1	0	1	1	0	0	0	0	0	0	0	1	4
9	1	0	0	0	0	0	0	1	0	1	0	0	3
11	0	1	0	0	1	1	0	1	0	1	0	0	5
12	1	0	1	1	0	0	0	1	0	0	0	1	4
15	1	1	1	1	1	1	1	1	1	1	1	1	6
16	1	1	0	0	0	0	0	0	0	0	1	0	3
17	1	1	1	1	0	1	0	1	1	1	0	0	8
18	1	0	1	0	1	0	0	1	0	1	0	0	5
22	1	0	1	0	1	1	1	0	0	0	0	1	6
24	1	0	1	1	0	0	0	0	0	0	0	0	3
29	1	1	1	1	0	0	0	1	1	0	0	0	6
31	1	1	0	1	1	0	0	1	1	1	1	0	8
34	0	0	1	0	1	0	0	1	1	0	0	0	4
35	1	1	1	1	1	1	1	0	1	0	1	1	10
TOTAL	12	6	9	8	7	4	3	9	6	6	3	4	

POST-DRUG TESTING - 3 MINUTE DELAY

SUBJECT NUMBER	WORD 1	WORD 2	WORD 3	WORD 4	WORD 5	WORD 6	WORD 7	WORD 8	WORD 9	WORD 10	WORD 11	WORD 12	TOTAL
6	0	0	0	0	0	1	1	0	1	0	1	0	4
8	1	1	0	0	0	0	0	1	0	0	0	0	3
9	1	0	0	0	0	1	0	0	0	0	0	1	3
11	1	1	1	0	0	0	0	0	1	0	0	0	4
12	1	0	0	0	1	0	0	1	1	0	1	0	5
15	1	1	1	1	1	1	1	1	1	1	0	0	10
16	1	1	1	1	0	1	0	0	1	0	1	0	7
17	1	1	1	0	0	1	1	1	0	1	0	0	7
18	1	1	0	0	0	0	0	0	0	0	0	0	2
22	1	1	0	0	0	0	0	0	0	0	0	0	2
24	1	0	1	1	0	0	0	0	1	0	0	0	3
29	1	1	1	0	1	0	1	1	0	1	0	0	6
31	1	1	0	0	0	0	0	1	0	0	0	0	3
34	1	0	0	0	1	0	1	1	1	1	1	1	8
35	1	1	1	0	1	0	1	1	1	1	1	0	9
TOTAL	14	10	7	3	5	5	5	8	8	5	5	2	

POST-DRUG TESTING - 5 MINUTE DELAY

SUBJECT NUMBER	WORD 1	WORD 2	WORD 3	WORD 4	WORD 5	WORD 6	WORD 7	WORD 8	WORD 9	WORD 10	WORD 11	WORD 12	TOTAL
6	1	0	0	1	0	1	0	0	0	1	0	0	4
8	1	0	0	0	0	0	0	0	0	0	0	1	2
9	0	0	1	0	0	0	0	0	0	0	0	1	2
11	1	0	1	0	0	0	1	1	0	0	0	1	5
12	0	0	0	0	0	0	0	1	1	0	0	0	2
15	1	1	1	1	1	1	0	1	1	0	0	0	7
16	0	0	0	1	0	0	0	1	1	1	0	0	4
17	1	0	1	1	0	0	0	1	0	0	0	1	5
18	0	1	1	0	0	0	0	1	1	1	1	0	6
22	0	0	0	0	0	1	0	0	1	0	0	0	2
24	1	1	0	0	0	0	0	0	0	0	0	0	2
29	1	0	1	1	0	0	0	0	1	1	0	0	5
31	1	1	1	1	0	0	0	0	0	1	1	0	6
34	0	0	0	0	1	1	1	1	0	1	0	0	5
35	1	1	1	1	1	0	0	1	0	0	0	0	7

Delayed free recall task lorazepam group post-drug session

POST-DRUG TESTING - IMMEDIATE RECALL

SUBJECT NUMBER	WORD 1	WORD 2	WORD 3	WORD 4	WORD 5	WORD 6	WORD 7	WORD 8	WORD 9	WORD 10	WORD 11	WORD 12	TOTAL
1	1	0	1	0	0	0	0	0	0	1	1	1	5
2	1	1	1	1	0	0	0	0	0	1	1	1	7
3	1	1	0	0	1	1	1	1	1	0	1	0	8
4	1	0	0	0	0	0	0	0	0	1	0	1	3
5	1	1	1	0	0	0	0	1	0	1	1	1	7
7	1	0	0	0	1	0	1	0	0	0	1	0	5
10	1	1	0	0	0	0	0	0	0	1	1	1	6
13	0	0	1	0	0	0	0	0	0	1	0	1	3
14	1	0	0	0	0	0	1	0	0	1	0	1	4
19	0	0	0	0	1	0	0	0	1	1	1	1	5
21	1	1	0	0	1	0	0	0	1	0	1	1	6
23	0	1	0	0	1	0	1	0	0	1	0	0	4
25	0	1	1	1	1	0	0	0	1	0	1	1	7
27	0	0	0	1	0	0	1	0	0	0	0	1	3
30	1	1	0	0	1	1	0	1	1	1	1	1	9
32	0	0	0	0	1	0	1	0	1	1	1	1	6
33	1	1	0	0	0	0	0	1	1	0	0	0	4
36	0	0	0	0	0	0	0	0	0	1	1	1	3
TOTAL	11	9	5	3	7	2	6	4	7	12	12	14	94

POST-DRUG TESTING - 1 MINUTE DELAY

SUBJECT NUMBER	WORD 1	WORD 2	WORD 3	WORD 4	WORD 5	WORD 6	WORD 7	WORD 8	WORD 9	WORD 10	WORD 11	WORD 12	TOTAL
1	1	1	0	0	0	1	1	1	0	1	0	0	6
2	1	0	0	1	0	0	1	0	0	1	1	0	5
3	0	0	0	0	0	0	0	1	1	1	0	0	3
4	0	0	0	0	1	1	0	0	1	1	0	0	4
5	1	0	0	0	0	0	1	0	0	0	0	0	3
7	0	0	0	0	0	0	0	1	0	0	0	0	1
10	0	0	1	0	0	1	0	0	0	0	0	1	3
13	0	0	0	0	0	0	0	0	0	0	0	0	0
14	0	0	0	0	0	0	0	0	0	0	0	1	1
19	0	0	1	0	0	0	0	0	0	1	0	0	2
21	1	0	1	0	0	0	0	0	0	1	0	0	3
23	0	0	0	0	0	0	0	1	0	1	0	1	3
25	0	0	0	0	0	0	0	0	1	0	0	0	2
27	0	0	0	0	0	0	0	1	1	0	1	1	4
30	1	1	1	1	0	0	0	1	1	0	1	1	7
32	1	1	0	0	0	0	0	1	1	0	0	1	5
33	1	0	0	0	1	1	0	0	0	0	0	0	3
36	1	1	0	0	0	0	0	0	0	0	0	0	4
TOTAL	8	4	5	1	2	5	3	9	5	9	2	6	59

POST-DRUG TESTING - 3 MINUTE DELAY

SUBJECT NUMBER	WORD 1	WORD 2	WORD 3	WORD 4	WORD 5	WORD 6	WORD 7	WORD 8	WORD 9	WORD 10	WORD 11	WORD 12	TOTAL
1	1	1	0	0	0	0	0	0	0	0	0	0	3
2	0	1	1	0	0	0	0	0	1	0	0	0	3
3	0	0	0	0	0	0	0	0	0	1	0	0	1
4	0	0	0	0	0	0	1	0	1	0	1	0	3
5	0	1	1	0	0	0	0	0	0	0	0	0	2
7	0	0	0	0	0	0	0	0	0	0	0	0	0
10	0	0	0	0	0	0	0	0	0	1	0	0	1
13	0	0	0	0	0	0	0	1	0	1	0	0	2
14	0	0	0	0	0	0	0	0	0	0	0	0	0
19	0	0	1	0	0	0	0	0	0	0	0	0	1
21	0	1	0	0	0	0	1	0	0	0	0	0	2
23	0	0	0	0	0	0	0	0	0	0	0	0	0
25	0	0	0	0	0	0	0	0	0	0	0	0	0
27	0	0	0	0	0	0	0	0	0	0	0	1	1
30	1	0	0	0	0	0	0	0	0	0	0	0	1
32	1	1	0	0	0	0	0	1	1	0	0	1	5
33	1	1	0	0	0	0	0	0	0	0	0	0	2
36	1	1	0	0	0	0	0	0	0	0	0	0	2
TOTAL	5	7	4	0	0	0	2	2	3	3	1	2	29

POST-DRUG TESTING - 5 MINUTE DELAY

SUBJECT NUMBER	WORD 1	WORD 2	WORD 3	WORD 4	WORD 5	WORD 6	WORD 7	WORD 8	WORD 9	WORD 10	WORD 11	WORD 12	TOTAL
1	0	1	1	0	0	0	0	0	0	0	0	1	3
2	0	0	0	1	1	0	0	0	0	0	0	1	3
3	0	0	0	0	0	0	0	0	0	0	0	0	0
4	0	0	0	0	0	1	0	0	0	1	0	1	3
5	0	0	0	0	0	0	0	0	0	0	0	0	0
7	0	0	0	0	0	0	0	0	0	0	0	0	0
10	0	1	0	0	0	0	0	0	0	0	0	0	1
13	0	0	0	0	0	0	0	0	0	0	0	0	0
14	1	0	0	0	1	0	0	1	0	0	0	0	3
19	0	0	0	0	0	0	0	0	0	0	0	0	0
21	0	0	0	0	0	0	0	0	0	0	1	1	1
23	0	0	0	0	0	0	0	0	0	0	0	0	0
25	0	0	0	0	0	0	0	0	0	0	1	1	1
27	0	1	1	0	0	0	0	0	0	0	0	0	2
30	1	0	0	0	1	0	1	1	1	0	0	0	5
32	1	1	0	1	0	1	1	0	1	0	0	0	5
33	1	0	1	0	0	0	0	0	0	0	0	0	2
36	0	0	0	0	0	0	0	0	0	0	0	0	0