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PREVIEW

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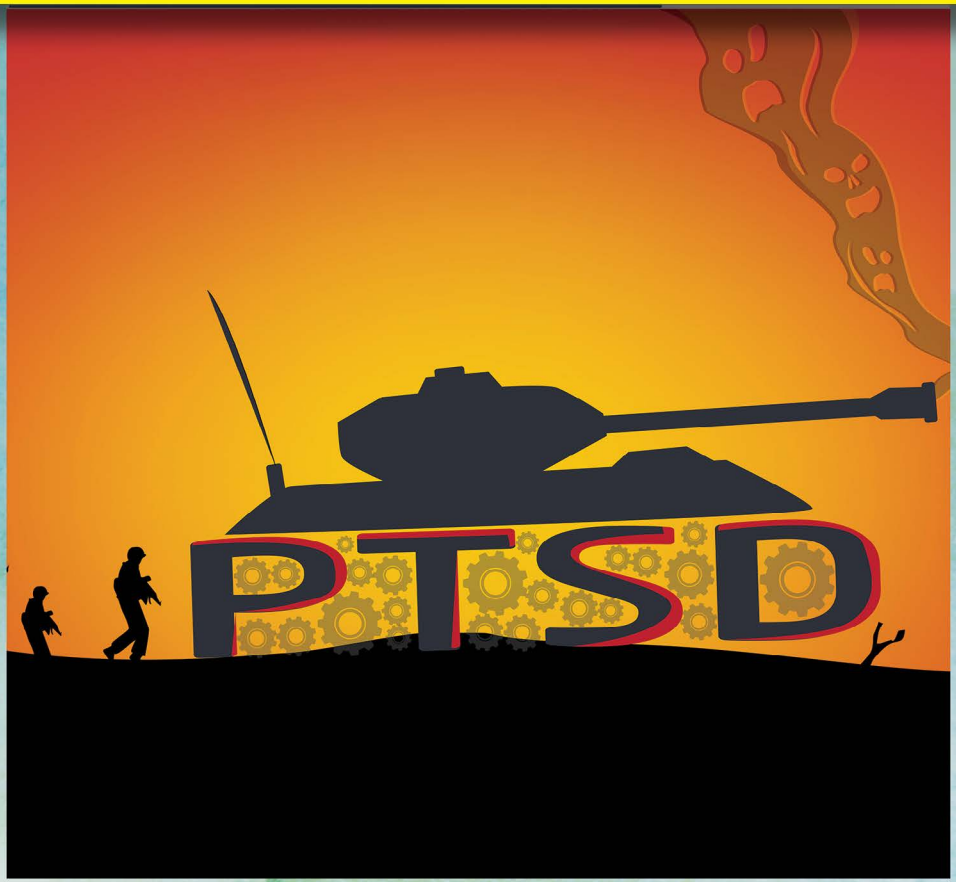
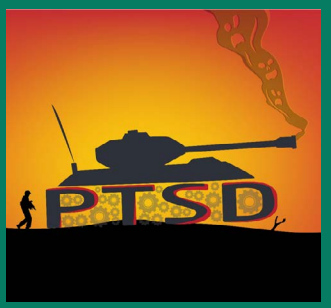


PTSD

Travis Dixon



Chapter 4
Post-Traumatic Stress Disorder

Introduction

During the Vietnam War a platoon of American soldiers raided the village of My Lai and killed many innocent men, women and children. Many gave the excuse that they were simply following orders, while others disobeyed orders and refused to kill. The lasting effects of this horrific event are unimaginable, for the Vietnamese civilians and for the soldiers. While the My Mai Massacre could be used as the basis of an investigation into the *origins* of extreme examples of violence, it's not introduced here as we're going to investigate the possible *effects* of being involved in such a traumatic event.

This example is just one of the countless acts of violence that have occurred during times of war. It's no surprise then that one of the most common causes of Post-Traumatic Stress Disorder (PTSD) is being exposed to situations of war. PTSD is the name given to the psychological disorder that occurs as a result of exposure to something extremely stressful. Individuals who develop symptoms of PTSD respond to the stressor (the traumatic event) with a feeling of fear, helplessness or horror. Many soldiers and civilians exposed to the horrors of war suffer from PTSD and early reports of this disorder came from soldiers fighting in World War One, when it was called "shell shock."

Experiences as a result of war make up just some of the common traumatic stressors that can cause the development of PTSD symptoms. Other examples include losing a loved one, experiences in natural or manmade disasters, car accidents, sexual abuse, physical assaults, or being diagnosed with a life-threatening illness. PTSD involves many symptoms, but individual symptomatologies can be different.

Generally speaking, the symptoms are grouped into three broad categories:

- Re-experiencing the traumatic event
- Avoidance and emotional numbing
- Increased anxiety and emotional arousal

There are many possible symptoms of PTSD and even more potential causes. In our limited time we can only skim the surface, so we'll focus primarily on symptoms related to memory and emotion. With this in mind, this chapter begins by explaining theories of how memories are formed. By first looking at these theories and the biological factors that influence memory, we will be able to develop a better understanding of how experiencing traumatic events might affect symptoms related to memory and emotion.

By understanding possible *causes* of PTSD, we can also investigate potential *treatments*. These will be explored later in the unit. But the study of psychological disorders raises numerous questions about ethics and diagnosis. How can we accurately diagnosis someone with PTSD? And do symptoms vary across cultures? What is the potential impact of diagnosing someone with such a disorder? These are some of the big questions we'll attempt to tackle towards the end of this unit

4.1 The Multi-Store Model

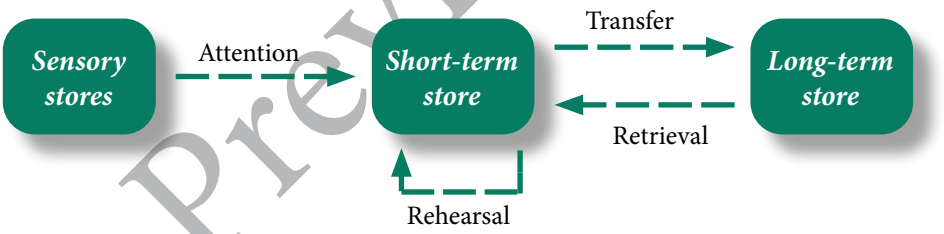
How are memories made?

(a) The Multi-Store Model of Memory (MSM)

Before we begin looking at the effects of PTSD on memory, it's important to explore just how memories are formed in the first place. The multi-store model of memory (MSM) is one of many ideas that came about during the cognitive revolution in psychology of the 1950s. Before this new wave of research into cognition, psychology was dominated by behaviourism. As the name suggests, this movement focused on observable behaviours as it was believed that internal mental processes could not be studied scientifically, so they shouldn't be the focus of psychological research. However, a new wave of research into cognitive processes began in the 1950s and the MSM is one product of this.

Like the dual process model of decision making attempts to explain how processing affects decision making, the multi-store model of memory attempts to illustrate how memories are formed through the interaction of memory stores and control processes.

Multi-Store Model of Memory



As the name suggests, Atkinson and Shiffrin's (1968) version of the multi-store model of memory posits that there are distinct "stores" for memory: sensory stores, a short-term store and the long-term store.

Control Process: a cognitive process that controls the flow of information from one store to another.

According to the MSM, information is first perceived and enters the sensory stores. These are modality specific stores, which means there are different stores for different modes of information. For instance, sounds would have an auditory store, and visual information a visual store (Eysenck and Keane, 2010).

The information is transferred through the stores by the control processes: attention, rehearsal and retrieval. For instance, information is transferred to the short-term store if we pay attention to it. This seems to make sense, as there's little chance we'd remember something if we weren't paying attention to it. If we rehearse information it will be transferred to our long-term store, where we can retrieve it and bring it back into our conscious memory when we need it.

Decay: information may be lost through the short-term store if it's not rehearsed. This is called decay.

The model also explains that there is a positive correlation between the amount of rehearsal of something and the strength of the memory trace created. If you only rehearse something a little, you might be able to retrieve it after a few minutes. After a couple of days it might be a faint memory. Whereas if you rehearse something

continually, you might be able to remember it for much longer because the memory trace is stronger.

Let's take a hypothetical example from a psychology class. Your teacher is explaining what normative social influence means and they say it's going to be on the test, so you pay attention. The information from their explanation goes into your sensory stores as you see the explanation on the board and listen to your teacher's voice. By paying attention to the information, it is transferred to your short-term store. You then write down the information in your notes, which is your rehearsal – you are going over the information again. About 20 minutes later in the lesson you are doing an activity and you have to help a classmate understand what the term means, so you retrieve the information from your long-term store and bring it back into your conscious memory, your short-term store. By explaining it you are rehearsing the information again. The more times you repeat this retrieval-rehearsal process, the stronger your memory trace will be, and the better you will remember the information.

The multi-store model of memory has significant empirical evidence to support the idea that our memory is made up of different stores and information flows between these stores through control processes. By this stage in your learning of the MSM you should be able to describe the model, including the different stores and the control processes.

Learning about the role of rehearsal and creating memory traces will help you to see the value in regular revision.

Memory Trace: a memory trace is a change in the brain's structure that facilitates memory storage. Neurons change their structure as a result of new learning (see NOVA's video "Memory Hackers" for this phenomenon being filmed).

Guiding Question:

How do control processes influence transfer of memory between the stores in the multi-store model of memory?

Abstraction Extension:
An Area of Uncertainty: To some extent the MSM seems to treat all information the same. It fails to address the role of emotion in memory. This will be explored more in later sections, but can you think how emotion might influence the control processes? Do you think all information, regardless of how emotional it is, would be rehearsed and/or retrieved in the same way, with similar effects? Can you provide examples?

If you're interested…

Crash Course has an introductory video to memory called: "How we make memories." The first half of this video is relevant for understanding the MSM, while the second half goes into details on working memory, which is the subject of the next topic.

(b) Duration and Capacity

As well as describing the existence of the three stores and explaining how memory travels between them through the control processes, another key aspect of the multi-store model of memory is the difference outlined between the three stores as they are different in terms of their duration and capacity.

Miller's 1956 article called "The Magical Number Seven, Plus or Minus Two" was instrumental in the cognitive revolution of the 1950s. It is also one of many pieces of research that led to the development of the multi-store model of memory.

Method	*Duration*	*Capacity*
Sensory Stores	A few seconds	Unlimited
Short-Term Store	About 20 seconds	7 units, plus or minus 2
Long-Term Store	Unlimited	Unlimited

The capacity refers to the amount of information that can be held in the store. According to the MSM, the sensory store has unlimited capacity, as does the long-term store. The short-term store, on the other hand, has a limited capacity. The magic number that researchers have deduced is seven units of information, plus or minus two. This basically means that a person can hold around five to nine pieces of information in their sensory stores. If people try to keep more than this in their sensory store they will make errors or forget parts.

The duration refers to how long information can stay in the store for. The sensory stores have a very short duration, with studies suggesting information can only last in our sensory store for a few seconds before it's lost. Interestingly, iconic memory (visual sensory details) seems to have a shorter duration in the sensory store than echoic memory (memory of sounds). The long-term store, not surprisingly, has a *potentially* unlimited duration as people may be able to remember things throughout their entire lives. But the short-term store has a duration of around 20 seconds (Eysenck and Keane, 2010).

If you are asked in an exam to explain a study that is "related to" a particular theory or model, you need to make it clear how the study is related. In many cases it will not demonstrate the complete theory but one particular aspect.

Peterson and Peterson (1959) found that unrehearsed short-term memory almost completely disappears after 18 seconds. They measured the duration of the short-term store by having participants remember trigrams - meaningless consonant triplets (e.g. XTB, MPT, PTR, etc.). Participants would be given one of these to remember but then they would be asked to count backwards in threes out loud as a form of distraction. For instance, a participant might be asked to remember TRM and then given the number 340. They would then have to say "337, 334, 331, 328…" After six seconds the ability to remember the three-letter stimuli (in the correct order) had about a 50% accuracy rate. When the time counting backwards was extended to 18 seconds, there was almost zero recollection of details.

By not giving participants a chance to rehearse the three-letter stimuli and giving them something distracting to do for various lengths of time, Peterson and Peterson could measure how long the information was able to be accurately held in the short-term store. (The lack of rehearsal prevents the information from being transferred to the long-term store.) By increasing the time counting backwards until there is 0% recollection of the stimuli, the researchers can provide approximations as to the duration of the short-term store.

Peterson and Peterson's work is another example of how studies can be used to demonstrate particular aspects of a theory. In this case their study using trigrams

can demonstrate the duration of the short-term memory store. It might be important to note that this experiment was conducted in 1959, *before* Atkinson and Shiffrin proposed their multi-store model of memory. Studies like Peterson and Peterson's were part of a body of research that lead to the development of the MSM.

Cognitive psychologists use true experiments to test the effects of variables on memory. Peterson and Peterson manipulated the length of the time delay before recall in order to measure short-term memory duration.

Guiding Question:

How can Peterson and Peterson's experimental results demonstrate the duration of the short-term store?

Abstraction Extension:

Mundane realism: Peterson and Peterson deliberately chose meaningless consonant triplets so participants would not be able to connect them to existing schema. Do you think this poses a problem in terms of the study's design? Could we generalize these findings to other situations, like learning something important? (I.e. think about mundane reality). What other factors may limit the extent to which this study can be used to demonstrate the MSM?

If you're interested…

Joshua Foer has a really interesting TED Talk about "Feats of memory anyone can do." In this video Foer describes memory competitions and his experiences in participating in one. You will be able to see some people with seemingly endless capacity for memory.

(c) Biological Evidence of the MSM

A model in psychology is a proposed illustration of how something occurs. While a theory attempts to explain relationships between variables and behaviour, a model attempts to represent the processes of a particular phenomenon.

A theory is only as strong as the evidence it has to support it. Therefore, when discussing and evaluating psychological theories and models it is important to examine their supporting evidence. In the previous section we looked at how one experiment can demonstrate the duration of the short-term store. This study has been critiqued, however, as the use of the same letters in multiple tests might have caused interference (Nairne et al., 1999).

Another way to investigate the validity of cognitive theories is to examine biological evidence that support their claims. There seems to be evidence to suggest that aspects of the MSM are biologically based. If the stores and control processes are different from one another (and memory is not just one big store in our brain), people who have damage to one aspect of memory may not experience problems with others. Case studies on patients with such abnormalities have shown just this and can be used as support for the MSM.

When evaluating models in essays you need to be able to describe the model in full and then explain how studies support (or contradict) particular aspects of the model.

Perhaps the most famous example of a patient with memory problems is that of Henry Molaison, known as HM while he was alive. He suffered from epilepsy, a disorder involving uncontrollable seizures and when he was 29 his seizures were becoming so bad that he and his family decided that he should have an operation. The operation proposed was radical and involved removing a part of his brain thought to be responsible for the seizures: his hippocampus. MRI scans carried out on HM much later (in the 90s) showed that other parts of his temporal lobe were also removed (Corkin et al., 1997). HM was first studied in the 1950s by Milner and Scoville (1957) after HM's doctor noticed that he suffered numerous problems with his memory as a result of the operation.

The hippocampus is in yellow in the above image. People who suffer from memory problems often have abnormalities in their hippocampi. It's also one of the first areas of the brain to be affected by Alzheimer's disease.

In one respect, the surgery was successful in that it resulted in HM suffering from fewer seizures. However, as a result of the surgery he was also suffering from anterograde amnesia: the inability to form new memories after the time of an accident. So while his memory of events from before the surgery was largely unchanged (i.e. his long term store was fine), he could not form new memories. An example described in the original article shows how his family moved houses after his surgery. Even though they only moved a few blocks from the previous address, HM could not remember the new house, and would walk to the old one instead. Milner also reports that he ate lunch in front of them, but 30 minutes later he could not remember eating. All other aspects of his character and personality remained perfectly unchanged. It was just that his ability to transfer information from his short-term store to his long-term store was damaged.

There's more information about HM available on our blog. You can also try an online version of a star tracing task.

Milner tested HM's short-term memory by giving him information to remember and then testing him on it later. In one example he was given a three digit number to remember for 15 minutes. If he continually focused on this number and kept paying attention to it, he was able to successfully remember the number. However, if he was

distracted he would forget it and the memory would not transfer to his long-term memory (Milner and Scoville, 1957; Squire and Wixted, 2011).

HM's short-term memory seemed to be fine provided that he was rehearsing the information and this rehearsal process seemed unaffected. And his long-term memory was mostly unaffected by the operation, meaning his long-term store was intact. However, the results suggest that there are biological components that influence the transfer of short-term memory to long-term memory. If our memory was just one big store with all the information in the same place and there was no *transfer* from store to store, patients like HM wouldn't experience such distinct abnormalities in their memory formation. Here we can see that case studies like HM's provide some evidence for the biological basis of different control processes that facilitate the transfer of information.

HM's study is another related to localization of brain function because it demonstrates the role of the hippocampus in the control process of transferring information from the short-term store to the long-term store.

However, one task carried out by Milner on HM suggests that not all types of memories were affected by the operation. Our long-term memory can be broadly categorized into two different types: declarative (explicit) memory and procedural (implicit) memory. HM's memory problems mainly affected his declarative memory, as he couldn't remember new events or remember new facts. His procedural memory, on the other hand, appeared to be fine. Milner tested HM's procedural memory by asking him to trace a star in a reflection. This is very challenging without practice and HM was slow and clumsy to begin with. But over successive trials he became more fluent, even though he couldn't remember having done the task before. The results of this star-tracing task show how the hippocampus may not be required for the formation of new procedural memories.

Declarative memory is also known as explicit memory. It includes memories of facts, events and experiences. Declarative memory can also be further divided into other types of memory, including semantic memory (facts and details) and episodic memory (events).

While HM's study can show that the hippocampus may be responsible for the transfer of some types of memory from the short-term to long-term store, providing biological evidence for the control processes in the MSM, it may not play a role in the formation of procedural memory. One major critique of the MSM is that it may not apply to procedural (implicit) memory.

Procedural memory is also known as implicit memory. It includes the ability to perform skills and tasks.

Guiding Question:

How does research on HM support claims of the MSM?

Abstraction Extension:

Contradictory Evidence: HM's study suggests that the hippocampus plays a role in the transfer of short-term memory to long-term memory. Throughout his life there weren't reports of improvements in these aspect of his cognition. Based on what you've learned about the brain, why might this be surprising? Does this lack of learning challenge other concepts you've learned about in this course? (Hint: neuroplasticity).

If you're interested…

You can see interviews with another amnesia patient in the UK called Clive Wearing. His wife, Deborah Wearing, wrote a book about Mr. Wearing's experiences called *Forever Today*. In the documentary "The Brain: Our Universe Within" you can also see how a promising young lawyer turned to an occupation of furniture making after a brain injury. Like HM, he lost his ability to form new long-term declarative memories, except his procedural memory was intact.

Relevant Topics

- Models of Memory
- Localization of Brain Function
- Techniques used to study the brain
- Cognitive processes
- Reliability of cognitive processes
- The brain and behaviour

Practice Exam Questions

- Explain one method used to investigate the reliability of cognitive processes.
- Evaluate the multi-store model of memory.
- Describe the multi-store model of memory.
- Outline one study related to one model of memory.
- Outline how one study demonstrates localization of brain function.

Research Methods

When investigating cognitive processes the true experiment (e.g. Peterson and Peterson) can be a valuable research method. Gathering empirical evidence is essential when testing theories of cognition. Early theories of cognitive processes relied on introspection, which involved psychologists reflecting on their own internal mental processes and using these to draw conclusions. Wilhelm Wundt, often credited as the father of Psychology, used introspection as a means of gathering evidence to test theories of cognition. This has obvious limitations in terms of objectivity and replicability – Wundt once claimed that studying the mind through introspection was like turning a light switch to try and study the darkness. By designing experiments that can test certain aspects of theories, researchers can describe their procedures in a way that would enable others to replicate their experiment and see if there is test-retest reliability. Case studies like HM's also allow researchers to study relationships between areas of the brain and behaviour.

Ethical Considerations

Case studies on patients with brain abnormalities come with many particular ethical considerations. HM agreed to donate his brain to science. After his death in 2008 his brain was sliced in 2,401 pieces to be preserved for study. Obviously, obtaining consent for such a procedure to take place is extremely important. However, HM's case isn't quite as simple as obtaining consent and comes with other interesting ethical dilemmas to consider. For example, can he really be said to have given consent if he would not be able to remember agreeing to the procedure? In this case, should family be consulted? In fact, there were questions raised over studies and procedures carried out on HM. You can read more about these in the NY Times article, "The Brain That Couldn't Remember."

4.4 Etiologies of PTSD

How might biology and cognition interact in PTSD?

(a) Genetics

In the previous topic you learned about how the amygdala plays an important role in fear conditioning. You also saw how prolonged experience of elevated levels of cortisol may have detrimental effects on the brain, including the hippocampus. While this topic is called "Etiologies of PTSD," the previous topic also gave you plenty of possible explanations for PTSD symptoms, including amnesia and memory impairments.

Etiology: the cause or set of causes of a disease or disorder.

When discussing origins and explanations of disorders in psychology, the term etiology is often used. Investigating the cause or origin of symptoms (their etiology) is at the heart of studying psychiatric disorders such as PTSD. One reason for this is because if we can discover underlying causes, we can develop better treatments.

You've already been introduced to some of the commonly cited biological etiologies of PTSD, including hippocampal atrophy and a hyper-responsive amygdala. But there's a question that needs to be asked of these studies that investigate the correlations between the brain and psychiatric disorders: are the brain abnormalities an *etiology* of the disorder, or are they a *symptom*? Bremner et al. (1995), for instance, showed that Veterans with PTSD had an 8% smaller hippocampus than the control group. But is this reduction a symptom of their experience in war, or was it a precondition that increased their likelihood of developing PTSD as a result of their experiences? It's a chicken-or-the-egg sort of dilemma.

It's difficult to determine if the brain abnormalities in PTSD patients are a cause or effect of the experience of trauma because patients only have their brain scans *after* they are exposed to trauma and are diagnosed with PTSD. This means there is often no data for before-and-after comparisons to be made. One way to overcome this would be to gather lots of data on people before a trauma and then compare this with what happened afterwards, but the practical limitation of this is that most people are not aware of when (or even if) they will experience trauma in the first place; it would also be expensive and time consuming.

You may be able to use the genetic origins of PTSD in Paper One or Paper Two.

As with genetics and violence, twin studies can elucidate the role of genetic factors on the development of PTSD. They can also provide an insight as to whether or not the difference in brain structure is a risk factor for PTSD, or a *symptom*. One way of overcoming the chicken-egg problem with PTSD symptoms is through the use of a case-control design, which can be done using identical twins.

In a case-control design the researchers gather two types of twins. The following is going to get rather tricky, so take your time to try to figure out how this design can determine if a biological variable like low hippocampus volume is likely to exist *before* trauma and increase the likelihood of developing PTSD, or *after* a traumatic event and is more likely a symptom of the PTSD.

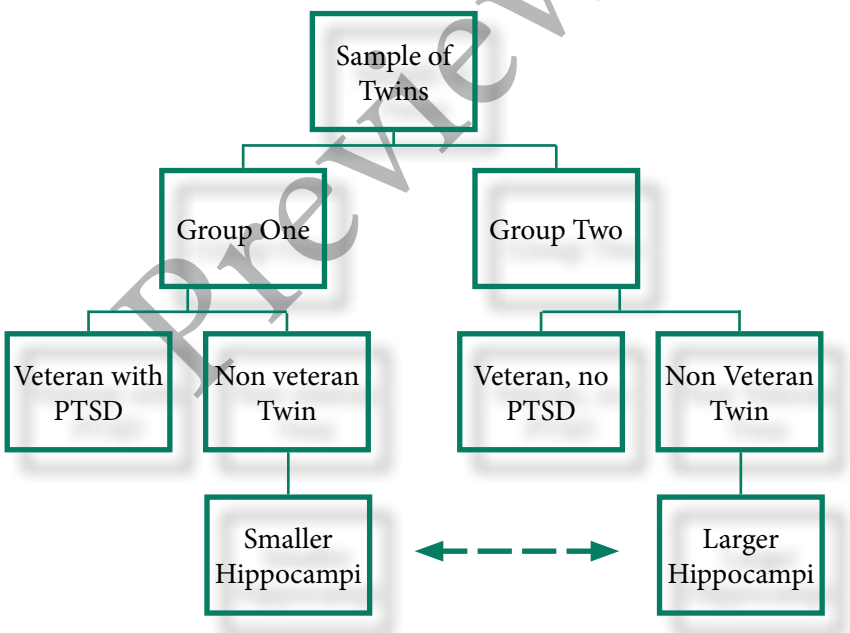
Gilbertson et al. (2002) conducted a case-control study on 34 sets of identical (monozygotic) twins. The design might seem complicated at first, but it is based on

some sound logic. Firstly, remember that monozygotic twins (MZT) have 100% of their DNA in common. If they've been raised in the same household they've probably had similar experiences. To use war veterans as an example, we can assume that before going off to war they had similar experiences and identical genes, so we could reasonably assume that their hippocampi would be the same in terms of structure and function.

Confused? It might help to draw a diagram or a mind map.

Gilbertson and colleagues used two different types of identical twins. One set of twins has one twin who has gone to war and developed PTSD (a trauma exposed twin), and the other twin who hasn't been to war and doesn't have PTSD (trauma unexposed). These twins are compared with a second set of twins. The second is the same as the first, except the twin that went away to war (trauma exposed) did not develop PTSD. The results of the study were that trauma unexposed twins of veterans with PTSD had smaller hippocampal volumes compared to unexposed twins of veterans without PTSD.

The important comparison to make here is between the two twins who didn't go away to war (trauma unexposed). The fact that the co-twin of the PTSD patient had a smaller hippocampi compared to the co-twin of the non-PTSD veteran, suggests that the low hippocampal volume is a factor that increases the vulnerability to developing PTSD. Therefore, the low hippocampal volume may be an existing factor in an individual that might increase their vulnerability to developing PTSD as a result of exposure to trauma.



A simplified diagram of how the case-control design works to see if a smaller hippocampus is a symptom or cause of PTSD.

Other MRI results from the study corroborate earlier studies that show the correlation between PTSD in veterans and hippocampal volume. Veterans with PTSD had an average hippocampal volume of 10% less than veterans without PTSD. There was also a negative correlation found between symptom severity and hippocampal volume: the lower the volume of the hippocampus the more severe the PTSD symptoms were.

This is further evidence that suggests genetics *and* hippocampal abnormality are possible etiologies and biological explanations of PTSD symptoms related to memory.

Guiding Question:

How does Gilbertson et al.'s study suggest that genetics could influence PTSD?

Abstraction Extension:
Population Validity: This sample deals with war veterans who are suffering from PTSD. The experiences of war are quite different to other traumatic experiences that could lead to the development of PTSD. In what way might war be different to other traumatic experiences, and how might this affect the validity of this study?

If you're interested…

If you're interested in the effects of stress on the brain and/or body I would highly recommend Sapolsky's *Why Zebras Don't Get Ulcers*. He also has a series of interesting documentaries that you may be interested in watching, including one called "Stress: Portrait of a Killer."

(b) Top-Down Processing and the PFC

So far in this chapter we've looked at two of the major potential biological etiologies of PTSD: a hyper-responsive amygdala and low volume in the hippocampus. You should be able to see how these areas of the brain may interact with our environment and cognitive processes to explain the possible development of some PTSD symptoms. There's one other important part of the brain that we haven't addressed yet: the prefrontal cortex (PFC). As you have already learned, the PFC plays a vital role in performing many cognitive processes, including processing and executive functions.

It's interesting to note that high levels of cortisol are not commonly found in people with PTSD. In fact, they tend to have slightly lower than normal levels of PTSD. This has also been a surprising finding for psychologists.

Numerous studies using MRI scans have shown that people with PTSD show decreased volumes in their prefrontal cortex. It's not only the structure that is affected, it might also be the function, as lower than normal activation of the prefrontal cortex can be seen in fMRI scans when people who have suffered from a traumatic experience are shown images related to their trauma (Shin et al., 2006). This reduced functioning of the PFC may play a role in an individual's ability to perform particular cognitive processes that may regulate emotional reactions. This could provide biological and cognitive explanations for some symptoms, including intrusive memories, increased arousal, emotional reactions and angry outbursts.

Bottom-up processing of emotion means that emotions are generated from a physiological response to environmental stimuli. Top-down processing of emotion means that emotions may begin with thoughts (originating in the prefrontal cortex) and these thoughts may affect other parts of the brain, including the amygdala, which activates the stress response. It has been a long-standing debate whether or not the experience of emotion is a bottom-up or top-down process. Research now suggests that both processes may play a role in the generation of emotion (Ochsner et al., 2010).

In criminology we focused on bottom-up processing. Now it's time to see how it can work both ways.

The PFC may affect PTSD symptoms through its role in top-down processing. You've seen extensive examples of how our amygdala receives input from our sensory organs, which is bottom-up processing, so let's now turn our attention to the relationship between top-down processing and symptoms of PTSD.

Top-down processing means thoughts and ideas can originate in our mind and

that can influence other areas of the brain responsible for processing sensory information, like our amygdala. In other words, instead of our stress response affecting our thoughts (bottom-up), our thoughts can influence our stress response (top-down). This means that negative emotions and the activation of the HPA axis may be generated from our negative thoughts (Taylor et al., 2010). As the PFC functions in executive cognitive control, including controlling our working memory, poor function in this area may explain such symptoms of PTSD, including intrusive memories: if we cannot control the regulation of sensory information in our short-term memory, we may experience unwanted (intrusive) memories that we can't "shake." These intrusive memories are one symptom of PTSD. This may lead to other symptoms, like depression and prolonged negative emotional states: if we can't get rid of our negative thoughts this is likely to get us down and keep us down, so to speak

How you think can influence your emotion. This is a powerful lesson to learn and has many implications for the study of disorders related to emotion.

Numerous studies have shown that the ability to regulate our emotions using top-down processing is an important function of the vmPFC (e.g. Koenigs and Grafman, 2009). Top-down processing by the ventromedial prefrontal cortex (vmPFC) also means that when our amygdala autonomically activates and triggers our stress response, a properly functioning vmPFC helps to regulate this reaction by enabling us to have cognitive control of our thoughts, feelings and emotions. It also results in a decrease in activation of the amygdala.

So while in the previous topic you saw how emotion might affect our cognition, the relationship also works in the other direction: our cognition can affect our emotion. Understanding the role of the vmPFC in top-down regulation of emotion will be important to fully understand how we might be able to treat PTSD, which is something we'll investigate later in this chapter.

You may be able to apply what you've learned in this topic to other topics in this course, such as the origins of violent crime.

Guiding Question:

How might reduced function in the vmPFC explain some symptoms of PTSD?

Abstraction Extension:

Making Connections: Think back to the Stanford Marshmallow Experiments. These studies showed that children who were able to exercise an ability to resist the temptation of one marshmallow and wait until they were given two, grew up to be more successful in other areas of life, such as in academics. Knowing what you do about the brain and behaviour, could you hypothesize why demonstrating an ability to perform cognitive control as a young child might lead to success later in life? Another question to consider is could top-down processing also be applied to the study of criminology?

If you're interested…

Psychology Today's online magazine has an article that discusses the debate between the processing of emotion called "Emotional Control: Top-Down or Bottom-Up?"

(c) Cognitive Reappraisal

In the previous section your understanding of the generation of emotions was enhanced by the introduction of a new idea: top-down processing. Our emotions aren't just a product of perception of environmental stimuli, they may also be generated from our thought processes. Understanding this process is important in order to fully comprehend etiologies and treatments of disorders such as PTSD.

The ability to diminish our fear through cognitive reappraisal and top-down processing is a cognitive ability that may be diminished in PTSD patients. Cognitive reappraisal is a strategy used to reduce emotional reactivity by considering the source of the emotion and thinking about it in a different way; the source of the emotion is reappraised, or re-evaluated. It's a type of top-down processing that involves our thought patterns reducing our emotional reaction generated in the amygdala. Understanding the role of cognition and thought patterns in etiologies and treatments of PTSD is just as important as understanding biological bases. In fact, it's hoped that you will be able to see how they're interrelated and can influence one another.

To test the role of the vmPFC in cognitive reappraisal, researchers use an experimental paradigm that involves participants being exposed to emotional stimuli while in an fMRI. They are then instructed to try to alter their emotional response to the stimuli through cognitive reappraisal. Urry et al. (2006) investigated the correlation between the vmPFC and the amygdala during a cognitive reappraisal task using fMRI scanners. In this study, 19 participants were exposed to a range of images while they were in the fMRI. The images were selected carefully so they ranged in ratings of emotional unpleasantness. The aim of the study was to expose participants to images that would induce an emotional response and then record their brain activity as they cognitively reappraised the images.

The reappraisal was manipulated as participants were flashed the images they were told to "increase," "decrease" or "attend." The participants received training in cognitive reappraisal, so they were able to follow the instructions. The strategies they were taught are included in the boxes below.

Increase

- Imagine someone you love experiencing the situation in the image (e.g. a car crash)
- Imagine a more intense version of the scene shown in the image (e.g. a vicious dog on a leash is shown in the image, and the participant imagines it breaking free)

Decrease

- Think about the situation as being fake or unreal
- Imagine the situation has a better outcome than the one shown (e.g. imagining that the people shown in a horrific car crash all survived and were fine)

Attend

- Maintain focus on the stimuli (control condition)

While the participants were performing these cognitive reappraisals, their brain activity was measured. The results showed that higher activation of the vmPFC during cognitive reappraisal to decrease the emotional effect of the stimuli led to greater reduction of the activity of the amygdala. This supports other similar studies that also demonstrate the role of the vmPFC in cognitive reappraisal and how this can reduce the activation of the amygdala during the processing of emotional stimuli (e.g. Delgado et al., 2008).

The research in this section provides you with another demonstration of the concept of localization of brain function.

Yet again we see the importance of understanding environmental, biological, cognitive and emotional factors contributing to behaviour. If a patient has abnormalities in their prefrontal cortex, they may not be able to perform cognitive reappraisal of affective (emotional) stimuli. This means that their emotional arousal generated in the amygdala and the accompanying negative thoughts will persist, keeping their stressful and emotional state high. An elevated sense of arousal and emotional affect (experiences of negative emotion) are symptoms of PTSD. This could explain some of the other emotional symptoms, such as increased startle responses and angry outbursts.

Cognitive reappraisal also applies to thinking differently about the traumatic event itself. If someone experiences intrusive thoughts or memories about their trauma, they may use cognitive reappraisal strategies they learn in therapy to reduce the emotional impact of these thoughts.

On the other hand, if they have developed thought patterns and constantly make negative appraisals of stimuli, they may be activating their stress response through top-down processes. For instance, someone who is experiencing nightmares or intrusive memories may be thinking, "I can't cope" or "I'll never recover." These thought patterns could be making the anxiety and stress response worse. Thus, cognitive *and* biological factors may explain the development and/or expression of symptoms of PTSD.

One category of symptoms in people with PTSD is also related to the avoidance of people, places and events that might remind them of the trauma. Perhaps through their conditioned fear of stimuli and their inability to regulate their emotional reaction because of their hypo-responsive vmPFC, patients with PTSD may find it easier to simply stay away from places that might trigger emotional reactions that they can't control. This may lead to feelings of isolation and depression.

Hypo-responsive is the opposite of hyper-responsive. If something is hyper it means it's overactive. A hypo-responsive area of the brain means it *lacks* reactivity to stimuli.

From what you've learned in the unit on meditation and mindfulness and their effects on the prefrontal cortex volume, and top-down control of the amygdala during

processing of emotional stimuli, you might be getting an idea of how treatments could be developed so that cognitive practice might lead to long-term structural changes in the brain. These treatments are the basis of a topic covered later in this chapter.

Guiding Question:

How might a hypo-responsive vmPFC influence emotional reactions to stimuli?

Abstraction Extension:
Alternative Explanations: So far we've looked at biological and cognitive etiologies of PTSD. Can you think of how cultural and/or sociocultural factors may be influential in the development of symptoms of PTSD? Think about what you've learned already about the environment and brain development.

If you're interested…

It helps to make learning about psychological disorders concrete by hearing real life stories about how people cope with this disorder. Devin Mitchell is a photographer who has produced a series of photos of people coping with PTSD and how they see themselves. The images are quite powerful and can provide some insights into the psychological effects of this disorder.

Relevant Topics

- Explanations for Disorders
- Etiology of Abnormal Psychology
- Cognitive Processing
- Emotion and Cognition

Practice Exam Questions

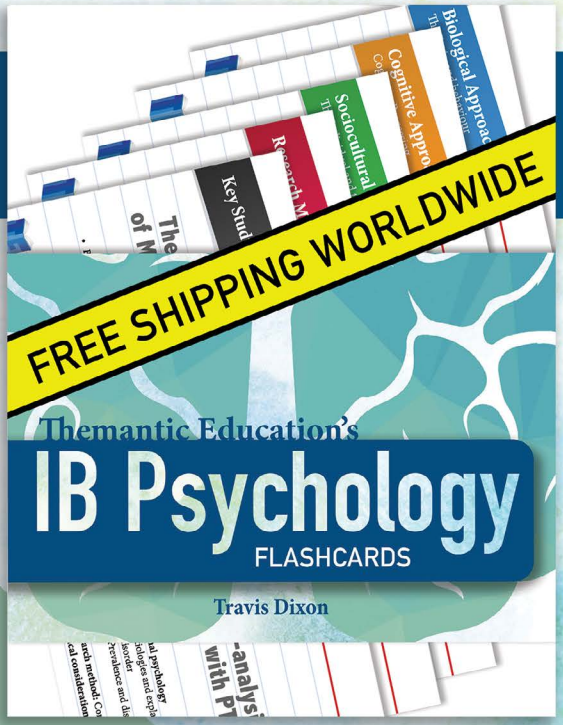
- Discuss two explanations for one or more disorders.
- To what extent can disorders be explained from the biological approach?
- Outline one study related to emotion and cognition.
- Discuss one ethical consideration relevant to the study of etiologies of abnormal psychology.

Research Methods

Urry et al.'s 2006 study is another example of the use of the true experiment when researching explanations of disorders. In this study, the researchers control the environment and extraneous variables. They also manipulate the independent variable, which is the type of cognitive appraisal of the stimuli that the participants are to perform. The dependent variable is the measure of the brain function during this particular task. Through the use of the experimental design you might think that researchers can draw a cause-and-effect relationship between the type of cognitive appraisal being practiced and the part of the area of the brain that is functioning. However, when using fMRIs and measuring brain activity, researchers need to be wary about drawing such cause-and-effect relationships. For example, do we know it's the cognitive appraisal that is causing the brain to function, or the function of the brain that is enabling the cognitive appraisal? This ambiguity means we can often only draw correlational conclusions when using fMRIs.

Ethical Considerations

Many of the studies you have seen involve exposing participants to stimuli that are designed to provoke an emotional reaction of some kind. This has obvious ethical implications. Informed consent would be important to ensure that the participants are not surprised by what they are asked to do while in such an experiment. The right to withdraw would also be important if the task became too stressful. This would be an especially important consideration if you were exposing victims of trauma to trauma-related stimuli.



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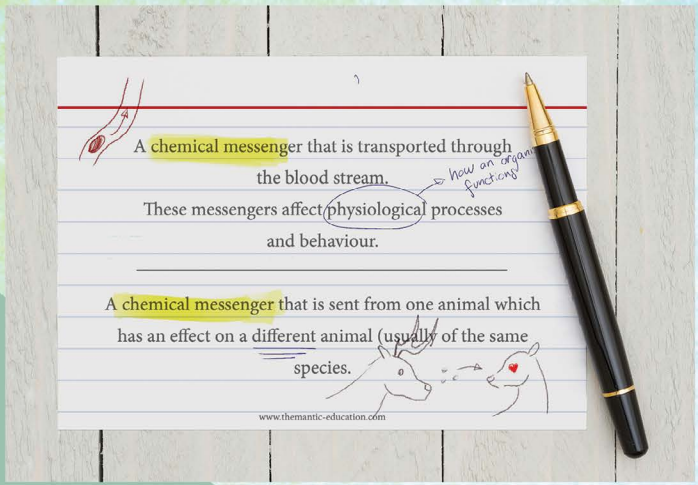

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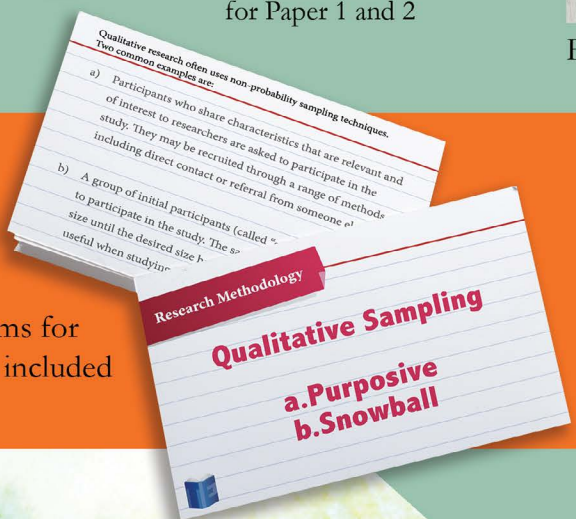

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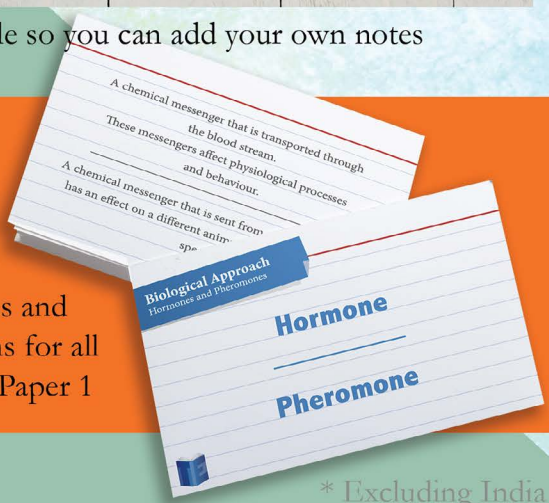

Key terms and definitions for all topics in Paper 1

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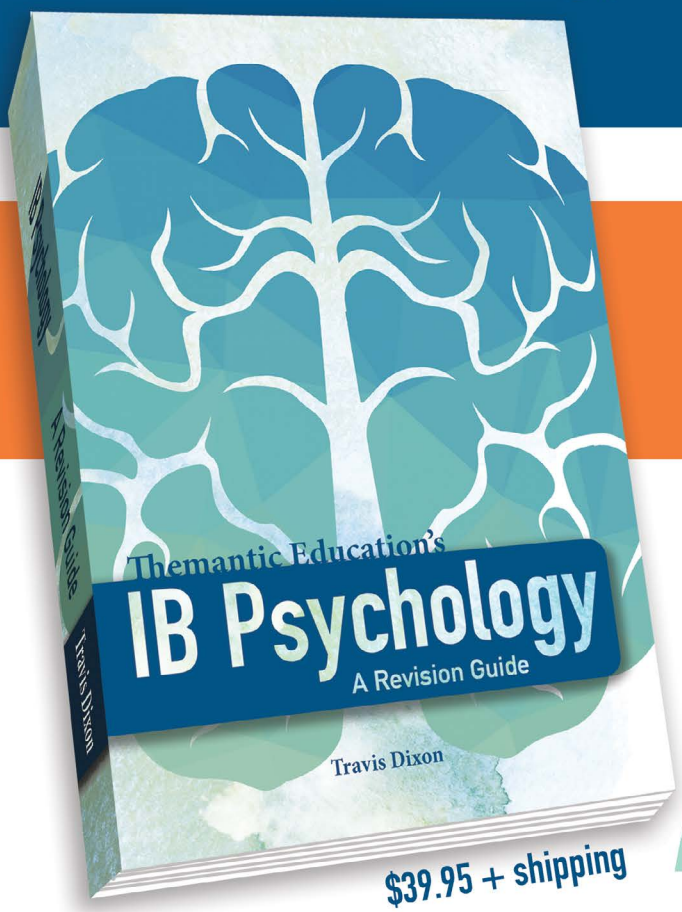

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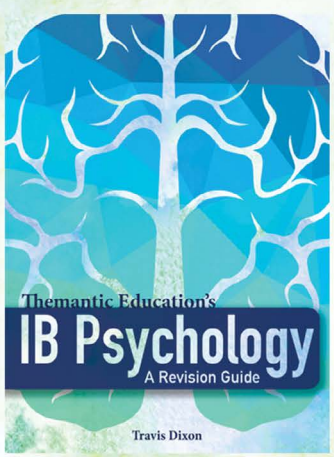

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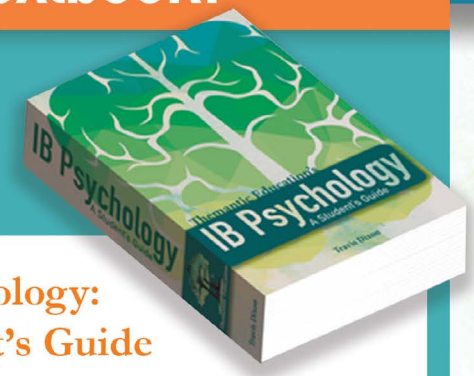

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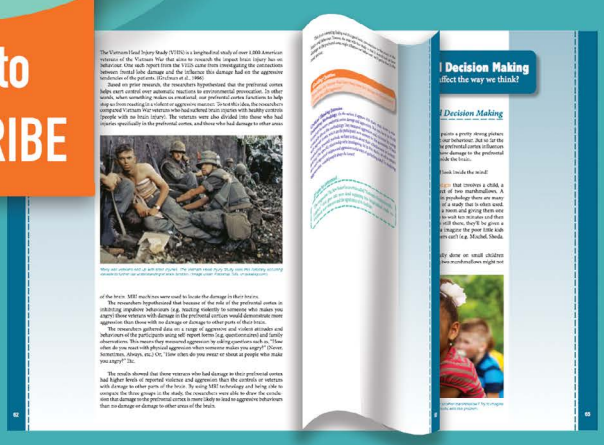

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