

Deficits in memory of those with korsakoffs syndrome research paper samples

[Health & Medicine](#), [Alcoholism](#)



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Introduction

Korsakoff's Syndrome is reflected by impaired memory processes. Both existing memories and new formations are affected by this syndrome. Not all memory processes work in the same way. Throughout this paper, it shall be explored and discussed the different types of memory processes that are mostly affected by this syndrome. We will evaluate some researches done on Implicit, Explicit and Contextual memory recollections to find out which of these is the most affected by this syndrome. Furthermore, in order to understand how Korsakoff's Syndrome works we shall also look at the history of the condition starting with Wernicke's Encephalopathy which is the beginning stage of Korsakoff's.

The evaluation of past researches done on implicit and explicit processing of contextual information will provide information about which areas are more

affected and whether patients can recall episodic memory or self-identifying long term memories. The way to evaluate these is by looking at the types of learning used within the researches, in this way they will help identify which are the mostly impaired memory processes in patients diagnosed with KS. Ultimately this paper aims to further explain Korsakoff's Syndrome, learn about its causes, symptoms and consequences to understand why there are still underdiagnosed cases.

A brief history of Korsakoff Syndrome

Before Korsakoff syndrome was first discovered and diagnosed, a German doctor called Carl Wernicke, in 1881 had three patients who suffered from delirium, ataxia, impaired consciousness and ocular disturbances. Apart from that they had little in common except that two of them were alcoholic men. The autopsy showed that the three of them had similar brain lesions. This is how Wernicke disease was first diagnosed. He called it psychosis polyneuritica due to the disturbance of memory seen in the patients. After six years of the new disease, a Russian professor, S. S. Korsakoff discovered another disease also in alcoholic patients, although not all were alcoholics. The difference between the Wernicke's disease and Korsakoff's was that this one was chronic rather than simply acute. The main characteristic was amnesia (Brockington, 2006).

In 1910, a Japanese researcher, Umetaro Suzuki discovered thiamine that is a vitamin of the B complex. Today we know that lack of this vitamin can have severe consequences, including death. Deficiency of this vitamin is usually caused by malnutrition or by impaired nutritional status that are

normally related to chronic diseases such as alcoholism. Other gastrointestinal diseases, HIV-AIDS and persistent vomiting can also result in the lack of this vitamin (Brockington, 2006).

There are several syndromes related to this deficiency: Beriberi, Wernicke-Korsakoff syndrome, malabsorption and optic neuropathy. One of the main results is amnesia so the first reaction is to relate this deficiency to Alzheimer's disease. However there is no evidence that relates these two. There is evidence however, to support that thiamine-deficiency is related to Korsakoff's syndrome due to malnutrition and alcohol consumption (Pinel, 2014).

There were other experiments with thiamine-deficient rats who showed memory deficits and brain damage patterns similar to alcoholic people (Mumby, Cameli & Glenn, 1999 in Pinel, J. (2014) Biopsychology). The conclusion was that because alcoholics get most of their calories through alcohol, they still lack essential vitamins such as thiamine. While Alcoholism is not the cause of this syndrome but rather the lack of thiamine, alcoholism can however accelerate the process.

Thanks to the development in biopsychology and the diverse methods used in different researches done in patients suffering from Korsakoff's syndrome, we know today more about this illness. Currently there is more awareness of what alcoholism can cause which helps people suffering from this bad habit to do counselling to help them stop drinking while they are being treated with high doses of thiamine.

The brain damage caused by the lack of this vitamin cannot be reversed,

however treating patients with high doses can improve their condition for their future (Pinel, 2014).

The Epigenetic Etiology of Korsakoff's syndrome

As it has already been mentioned, both Wernicke's and Korsakoff's syndromes develop because of a deficiency in thiamine which is a vitamin of the complex B, more strictly speaking it is the Vitamin B1. Deficiency of this vitamin develops mainly because of malnutrition or malabsorption.

Malnutrition is evident in alcoholism (Pinel, 2014).

Most people with alcoholism receive most of their calories if not all, in the form of alcohol. Thus, most of the nutrients, including this vital vitamin B1 is absent. However, other chronic illnesses can also cause the same syndrome because ultimately it is not alcoholism itself that causes the syndrome but the lack of this vitamin (Pinel, 2014).

Some people who go through surgery due to obesity problems (bariatric) can also suffer from malabsorption and thus end up with this illness. Other gastrointestinal illnesses can also result in Korsakoff syndrome (Wernicke-Korsakoff Syndrome, n. d.).

While Wernicke's illness is acute, Korsakoff syndrome is chronic; the damage caused by the lack of vitamin 1 cannot be reversed. As Wernicke's symptoms disappear, Korsakoff's symptoms start to show. It could be said that Wernicke's syndrome is the beginning of Korsakoff. When this one starts, the person already has brain damages in lower parts of the brain, in the thalamus and the hypothalamus caused by Wernicke's syndrome. These areas that become permanently damaged are involved in memory processes

which end up causing psychosis and amnesia as the principal symptoms (Wernicke-Korsakoff Syndrome, n. d.).

Other conditions that can cause vitamin B1 deficiency are AIDS, cancers, extreme vomiting, long term treatments to heart failure done with diuretic therapy, long-term dialysis and extremely high thyroid hormonal levels called thyrotoxicosis (Wernicke-Korsakoff Syndrome, n. d.).

Some of these conditions have genetic causes such as cancer, diabetes and thyroids. Patients who develop Wernicke-Kornakoff syndrome have a genetic error in their metabolism that comes to play when their diet has inadequate levels of thiamine. What this means is that this syndrome is a recessive disorder (Blass & Gibson, 1977).

Symptoms and Treatments

Before Korsakoff appears, that is, before the damage is permanent, and the condition becomes chronic, Wernicke's symptoms are already present.

Patients show confusion and loss of normal mental activity. In some cases, these symptoms can develop up to the point of coma or death. Ataxia is also another common symptom: loss of muscle coordination causing tremor in the legs. Vision is also affected: abnormal eye movement, double vision and eyelid drooping (Wernicke-Korsakoff Syndrome, n. d.).

As the condition progresses these symptoms start to disappear, and those of Korsakoffs' start to show. Patients become unable to form new memories while they also lose their current memories. They start to make up stories, and in some cases they start to have hallucinations (Wernicke-Korsakoff Syndrome, n. d.).

Reflexes are also affected. They can have fast pulse, low blood pressure and body temperature. Because of the severe tissue mass loss, patients experience a general muscle weakness and atrophy affecting their walk and coordination. Thiamine Deficiency also causes transketolase activity in red blood cells. The brain damage caused by this syndrome cannot be reversed. However, treatments aim at controlling the symptoms and trying to prevent worse symptoms (Wernicke-Korsakoff Syndrome, n. d.).

Treatments include supplying patients with high doses of Vitamin B1. This one is supplied by injection or orally. Balancing the amount of Vitamin B1 in the brain can help improve symptoms of confusion and delirium, vision and eye movement symptoms, and it can help improve muscle coordination (Wernicke-Korsakoff Syndrome, n. d.).

In those cases where alcoholism was the cause of the syndrome, stopping alcohol use can prevent further brain damage and thus further memory loss. A treatment of Vitamin B1 intake plus a balanced diet is ideal to improve the symptoms, but it will not replace alcohol use if this one is not stopped. If these symptoms are not treated, the patient can end up dying (Wernicke-Korsakoff Syndrome, n. d.).

Wernicke-Korsakoff syndrome can be prevented by having a good balanced diet and by stopping alcohol consumption. If however the patient is unable to stop consuming alcohol, thiamine supplements and a balanced diet can help delay and reduce the damages; however the risk is will not disappear.

Research Evaluation

Kessels & Wester (2007) performed a study to evaluate the benefit of errorless learning in patients with amnesia. What are the mechanisms underlying errorless learning? Studies on the relation between errorless learning and implicit and explicit memory function show that the benefits of errorless learning are based on residual explicit memory rather than to intact implicit memory function. However, results are still inconclusive as further recent findings indicate that the implicit memory seem to be the reason behind errorless learning benefits in patients with moderate and severe amnesia. The question that arises then is whether the difference might be in the type of information being learned through errorless learning?

Route learning relies on multiple cognitive processes. Kessels & Wester, (2007) investigated the effects of errorless learning on route learning. They concluded that there is no clear benefit compared to trial-and-error learning. The conclusion they made was that implicit spatial memory tasks might be slightly benefited by errorless learning, but there is no clear evidence for this to benefit the procedural spatial learning as well. Thus, the benefit of errorless learning is dictated by the explicit or implicit nature of tasks.

Finally, it can be concluded that in terms of clinical applicability of errorless learning is limited. Kessels & Kopelman (2012) on the other hand evaluated the findings of patients with amnesia and context memory deficit. In particular, they focused on implicit learning of contextual information in patients with Korsakoff's syndrome.

Not all aspects of the memory are impaired in patients with Korsakoff syndrome. Implicit contextual learning is not affected as much, and however

working memory is heavily affected. Implicit learning of contextual information such as word associations in these patients is well-preserved. However, there are deficits in contextual working memory that is to maintain associative information. Whether it is encoding process impairment or a pure damage of the working memory is still unclear. But what Kessels & Kopelman (2012) conclude from their investigation is that patients with this syndrome do benefit from implicit contextual cues during learning, they are even able to learn a route in their own everyday environments. What these studies show is that explicit and implicit memory functions use distinct neural circuits.

If we look at the previous research of Kessels & Wester (2007) and apply it to the conclusions of Kessels & Kopelman (2012) we can see that patients with Korsakoff syndrome would benefit from an errorless learning as implicit learning of contextual information for associations is still preserved. Also, implicit contextual cues while learning can help them form routes in their own environment. Probably what Kessels & Wester (2007) found that there is no benefit on errorless learning in route learning might be because as seen in Kessels & Kopelman (2012), while implicit learning is intact, contextual working memory, that is an encoding process is impaired. What would be an interesting further study would be to evaluate if errorless learning for en route learning relies on the working memory.

Hayes et al. (2012) also investigated different findings of the extent memory damage and its processes on patients with KS. They focused on procedural learning and priming. They conclude that the extent of damage or intact of implicit memory depends on the specific task requested and the demands

such as timing between stimuli. This is because it is an integrate process of different cognitive functions. This last part supports the two previous papers that there are different processes involved and not all of them use the same neural circuit.

Hayes et al., (2012) conclude that procedural learning and priming in KS, which is the focus Kessels & Kopelman (2012) of their study reflect the complexity of the syndrome. This is because of the lack of Thiamine and the abuse of Alcohol that has profound effects in the brain affecting many areas and not simply one neural circuit. Hayes et al. (2012) also determined that those with KS have impairments in different cognitive functions such as executive functions, visuospatial and visuoperceptual abilities. These cognitive functions affect both procedural memory and priming.

We can conclude by saying that we cannot take a clear cut conclusion by isolating memory processes. This is because most memory tasks require the use of integrative cognitive functions and this syndrome affects most of the neural circuits related to memory. Thus, it is not clear which of those cognitive functions is the one impairing a particular memory function.

However, we can conclude based on the reviews we studied, on a general basis, working memory is clearly more affected than implicit memory learning.

Conclusion

The intention of this paper is to evaluate researches done on different memory processes in patients with KS to distinguish which of those processes are most affected. Not all memory processes work in the same

way, and the first conclusion we can make in this paper is that different processes rely on different cognitive neural systems. We also can say that some processes use differently cognitive functions each using different neural paths. Thus, we cannot isolate processes in this syndrome as in most cases the abuse of alcohol and the lack of thiamine performed severe brain damage affecting many of these neural circuits affecting many cognitive processes which in turn will affect several parts of particular memory processes being studied.

Having said this, we can conclude though that implicit memory is less affected than working memory. Thus, KS patients are able to learn through association while they find it harder to keep those newly formed memories as the process of encoding and decoding is highly impaired.

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