Symposium on Memory Enhancement: Program
April 21+22, 2012 – Max Planck Institute of Psychiatry, Munich

Saturday

09:00 Registration opens

09:30 Martin Dresler (Max Planck Institute of Psychiatry, Munich)
Opening remarks

09:40 Barbara J. Sahakian (University of Cambridge)
Pharmacological cognitive enhancement of memory and other forms of cognition

10:40 Dimitris Repantis (Charité University Hospital, Berlin)
Pharmacological memory enhancement: acetylcholinesterase inhibitors and memantine

11:20 Kamilla Miskowiak (Copenhagen University Hospital)
Memory enhancement by Erythropoietin (Epo)

12:00 Lunch

13:00 Shira Knafo (University of Madrid)
The role of AMPA receptors in memory enhancement

13:40 Lisa Genzel (University of Edinburgh)
Schemas and memory enhancement

14:20 Carsten Wotjak (Max Planck Institute of Psychiatry, Munich)
Pros and cons of memory enhancement in the therapy of anxiety disorders

15:00 Coffee brake

15:15 Benno Roozendaal (University Medical Center Groningen)
Stress hormones, amygdala activation, and memory of emotionally arousing experiences

15:55 Oliver Wolf (Ruhr University Bochum)
Stress and memory in humans: enhancing and impairing effects

16:35 Coffee break

16:50 Amir Homayoun Javadi (Technical University Dresden)
Memory modulation by transcranial direct current stimulation (tDCS)

17:30 Simone Kühn (Max Planck Institute for Human Development, Berlin)
Memory enhancement by computer training

18:10 Carlos Trenado (University of Maryland, College Park)
Computational models of memory enhancement

19:30 Bavarian Dinner
Sunday

09:00 Boris Nikolai Konrad (Max Planck Institute of Psychiatry, Munich)
Memory enhancement by mnemonic techniques

09:50 Steffen Gais (Ludwig Maximilian University of Munich)
Memory enhancement by sleep

10:30 Coffee break

10:40 Björn Rasch (University of Zurich)
Strengthening memories by reactivation during sleep

11:20 Thomas Metzinger (University of Mainz)
Neuroethics of pharmaceutical cognitive enhancement: The first ten years

12:00 Lunch

13:00 Anders Sandberg (University of Oxford)
The ethics of memory enhancement

13:40 Laura Cabrera (University of Basel)
Issues not to be forgotten when evaluating memory enhancement

14:20 Christoph Bublitz (University of Hamburg)
A duty to remember, a right to forget? Legal issues in memory enhancement and modification

15:00 Coffee break

15:20 Veljko Dubljevic (University of Tübingen)
A theoretical investigation of principles of justice as criteria for assessing cognitive enhancement

16:00 Felix Krause (University of Münster)
Neuro-Enhancement as part of human freedom and personal responsibility:
A theological-philosophical reflection

16:40 General discussion

17:00 Closing remarks and farewell

Address

Max Planck Institute of Psychiatry
Kraepelinroom, 3rd floor Kraepelinbuilding
Kraepelinstr. 2-10
80804 Munich

Googlemaps: http://map.mpip.de

Phone: +49-176-34171915 (Martin Dresler)
Directions

By public transportation

From the station or city center:
From the main railway station (Hauptbahnhof) take subway U2 headed for Feldmoching. Get off at Scheidplatz (a 7-minute ride).
OR: From city center Marienplatz take U-Bahn U3 headed for Moosach. Get off at Scheidplatz (a 9-minute ride). Take the exit Bummstraße. At the end of Bummstraße turn left onto Kraepelinstraße.

From the airport:
Take the regional train line S8 or S1 to the main station (Hauptbahnhof) and transfer to the subway line U2 headed for Feldmoching.
OR: Take the S8 to Marienplatz and transfer to subway line U3 headed for Olympiazentrum. For further directions, see above.

By car

From the north: Approaching Munich on highway A9 from Nürnberg, get off at exit M-Schwabing/West. Take Schenkendorfstr./Petuelring into the Petueltunnel and the next exit Milbertshofen. Turn left onto Belgradstraße and after 300 m take the first left onto Bummstraße, which will pass into Kraepelinstraße.

From the south: Approaching on highway A95 from Weilheim/Garmisch, get off at exit Garmischer Straße (Mittlerer Ring). Take the Mittlerer Ring (Landshüter Allee, Georg-Brauchle Ring and Petuelring) to exit Milbertshofen. Turn right onto Belgradstraße and proceed as above.
OR: Approaching on highway A8 from Rosenheim, get off at exit M-Ramersdorf (Mittlerer Ring). Take the Mittlerer Ring to M-Steinhausen, then Prinzregentenstraße and Von-der-Tann-Straße. Turn right onto Ludwigstraße and follow the Leopoldstraße. Turn left onto Hohenzollernstraße, then right onto Belgradstraße. Pass Scheidplatz, turn right onto Bummstraße and approaching Kraepelinstraße to the left.

From the east: Approaching on highway A94 from Forstinning (direction from Linz), get off at the exit M-Steinhausen. Then proceed as under “From the south/A8”.

From the west: Approaching on highway A96 from Lindau, get off at the exit Garmischer Straße (Mittlerer Ring). Then proceed as under “From the south/A95”.

Bavarian dinner:

Wirtshaus Zur Brez’n
Leopoldstraße 72
80802 Munich

From Scheidplatz take the subway U3 south, get off at Münchener Freiheit.
Psychiatric disorders are disorders of neurocognition. Many psychiatric disorders, such as schizophrenia and attention deficit hyperactivity disorder, are of neurodevelopmental origin with an onset or prodromal stage in childhood or adolescence. It is now recognised that cognitive problems may impact performance of everyday functioning of patients with mental health disorders and prove to be the biggest barrier to rehabilitation and return to paid employment. For example, poor episodic memory has been linked to impairments in functionality in both Alzheimer’s disease and schizophrenia.

Therefore biomarkers, including cognitive, genetic, and neuroimaging ones are needed for prevention, early detection and for assessing efficacy of treatments. In addition, cognitive enhancing drugs are needed to treat cognitive impairment associated with debilitating neuropsychiatric disorders. Indeed, recent neuroscience and mental health policy has emphasized the importance of novel approaches for drug development for psychiatric disorders. For instance, targets for treatment may become closely related to genetics and neurobiology (e.g. episodic memory, impulsivity) rather than diagnostic categories (e.g. schizophrenia, ADHD). A treatment which improves episodic memory problems might prove useful for improving cognition and functional outcome in both mild Alzheimer’s disease and first episode schizophrenia. Working memory problems are found in a variety of psychiatric disorders and therefore improving working memory could be of benefit to many groups, including patients with ADHD or those with first episode psychosis and schizophrenia.

These cognitive enhancing drugs are able to improve forms of cognition, such as working memory, not only in people with neuropsychiatric disorders but also in healthy people. This raises important societal neuroethical issues due to increasing lifestyle use of cognitive enhancing drugs by healthy people.
The term neuroenhancement refers to improvement in the cognitive, emotional and motivational functions of healthy individuals through, inter alia, the use of drugs. Of known interventions, psychopharmacology provides readily available options, such as the anti-dementia drugs, e.g. acetylcholinesterase inhibitors (donepezil, galantamine, rivastigmine) and memantine. Based on a systematic review we found that expectations about the potential of these drugs exceed their actual effects, as has been demonstrated in randomised controlled trials. Both single and repeated dose trials were included in the systematic review, however repeated dose trials have only been conducted for donepezil. In six small trials lasting 14–42 days, the following results emerged: donepezil improved the retention of training on complex aviation tasks and verbal memory for semantically processed words. In one study episodic memory was improved, whereas in others it remained unaffected by donepezil. In a sleep deprivation trial, donepezil reduced the memory and attention deficits resulting from 24 h of sleep deprivation. Two studies reported even transient negative effects. Regarding the safety profile of donepezil, these studies found that it was rather well tolerated. In any case, since large longitudinal studies are not available no conclusions can be drawn. Seven small studies about the effects of a single dose of memantine, and one study with a single dose of rivastigmine have been reported. Again, these studies are not adequate to answer our research question. If, as here and elsewhere suggested, the concept of pharmaceutical neuroenhancement is not to be rejected in principle, the decision of healthy individuals to take drugs for the purpose of neuroenhancement should be based on exhaustive information. At the moment, the research that would support or oppose the use of acetylcholinesterase inhibitors and memantine for neuroenhancement by healthy individuals has not yet been performed.
Dr. Kamilla Miskowiak (Copenhagen University Hospital)

Memory enhancement by Erythropoetin (Epo)

Current pharmacological treatments for mood disorders fail to reverse cognitive dysfunction. Erythropoetin (EPO) has neuroprotective and neurotrophic actions and improves memory function in animal models of acute and chronic neurodegenerative conditions and in patients with cognitive decline. We therefore investigated the effects of EPO administration to healthy volunteers and patients with clinical depression to clarify 1) if EPO has direct actions on neurocognitive function independent of effects on hematocrit 2) and if so, whether such actions would indicate memory enhancement and increased neural plasticity. We used functional magnetic resonance imaging to explore the effects of a single dose of EPO (40,000 IU) versus saline on neural and cognitive response during hippocampus-dependent picture memory tests in healthy volunteers 3 or 7 days after administration and in depressed patients 3 days after administration. Blood tests were taken before and after EPO/placebo administration. We demonstrated that a single dose of EPO directly enhances memory-relevant hippocampal response in healthy volunteers one week, but not three days, after administration consistent with increased downstream neurotrophic signaling. EPO also modulated memory-relevant hippocampal response and improved memory specificity in depressed patients. These effects occurred in the absence of changes in hematocrit pointing to a direct neurobiological action. Our findings highlight EPO as a candidate agent for management of memory dysfunction in patients with mood disorder. Larger-scale clinical trials of EPO as a treatment for neurocognitive symptoms in these patients are therefore warranted.
Cell adhesion molecules and growth factor-dependent signalling are critical for brain development and synaptic plasticity, and they have been linked to cognitive function in adult animals. We have previously shown that a mimetic peptide (FGL) from the neural cell adhesion molecule (NCAM) triggers fibroblast growth factor receptor (FGFR) activation and enhances spatial learning in young rats. We investigated the cellular and molecular basis of this cognitive enhancement using biochemical, morphological, electrophysiological, and behavioral analyses. FGL triggered a long-lasting enhancement of synaptic transmission, recorded from hippocampal CA1 neurons. This effect was mediated by facilitated synaptic delivery of AMPA receptors, accompanied by enhanced NMDA receptor-dependent long-term potentiation. Both synaptic potentiation and cognitive enhancement were mediated by rapid FGL-induced activation of the protein kinase C pathway, followed by slow but long-lasting activation of Ca2+/calmodulin-dependent protein kinase II. These results suggest that transient pharmacological activation of the NCAM-FGFR pathway persistently facilitates AMPA receptor synaptic insertion and plasticity, leading to enhanced hippocampal-dependent learning.
In the past research has largely ignored the impact of previous knowledge on memory encoding and consolidation. More recent studies could show the importance of existing memory schemas in humans and rodents. By utilizing pre-existing schemas memory encoding becomes more efficient and consolidation occurs at a much faster rate. For example when rodents can incorporate new flavour-location paired-associates into an existing framework, the retrieval becomes hippocampus independent in mere 48 hours instead of previously assumed weeks to months. The concept of schemas is an important factor in memory enhancement and challenges current models of memory consolidation.
Pros and cons of memory enhancement in the therapy of anxiety disorders

Generalized avoidance belongs to the core symptoms of a variety of anxiety disorders such as Panic disorder or Posttraumatic stress disorder. However, therapy for avoidance behavior still bears many obstacles. Even though exposure-based approaches are the method of choice, they suffer from inferior patient compliance. This can be ascribed to patients’ inability to stand the high emotional load experienced during the therapeutic sessions. The situation could be much improved if learning about the safety of a feared situation could be enforced. This would allow for the number/duration of the exposure sessions to be restricted to a minimum and at the same time, the emotional load of the therapeutic sessions could be dampened, with direct consequences on compliance rates. So far, however, most of the treatments with anxiolytic capabilities (e.g. benzodiazepines) lead to state-dependency or amnesia, with the consequence that safety learning is attenuated, if not completely blocked. I will introduce the endocannabinoid system of the brain as an interesting new target for the pharmacoenhancement of exposure-based therapies. I will provide evidence that endocannabinoid signaling via cannabinoid CB1 receptors is essential for acute fear relief and safety learning. Moreover, I will demonstrate that inhibition of endocannabinoid uptake (and, thus, enhancement of endocannabinoid signaling) promotes safety learning and attenuates the risk of relapse.
Extensive evidence indicates that stress hormones released from the adrenal glands are critically involved in memory consolidation of emotionally arousing experiences. Epinephrine or glucocorticoids administered after exposure to emotionally arousing experiences enhance the consolidation of long-term memories of these experiences. Our findings indicate that adrenal stress hormones influence memory consolidation via interactions with arousal-induced activation of noradrenergic mechanisms within the basolateral complex of the amygdala (BLA). In turn, the BLA regulates memory consolidation via its efferent projections to many other brain regions. In contrast to the enhancing effects on consolidation, high circulating levels of stress hormones impair memory retrieval and working memory. Such effects also require noradrenergic activation of the amygdala and interactions with other brain regions. These apparently dual effects of glucocorticoids on memory consolidation versus memory retrieval and working memory appear to be related in terms of function and neurobiological substrate. The BLA is a key structure in a memory-modulatory system that regulates, in concert with other brain regions, stress and glucocorticoid effects on these different memory functions.
Stress is known to influence memory. Research over the last decades has revealed that glucocorticoids released from the adrenal cortex are important mediators in this respect. Stress can have enhancing as well as impairing effects on episodic long-term memory. For example we may remember a certain embarrassing moment for our entire life. In contrast we may forget an appointment, because we are stressed at work. The former is an example of a stress-induced memory consolidation enhancement while the latter is an example of a stress-induced retrieval impairment. The talk will give an overview of experimental studies conducted in my laboratory using behavioral as well as imaging techniques. A better understanding of the modulatory effects of stress on human memory will enhance our understanding of stress associated mental disorders and could provide the fundament for improved treatment approaches.
Memory modulation by transcranial direct current stimulation (tDCS)

Transcranial electrical brain stimulation has been used since more than 200 years ago in clinical applications such as treatment of depression. This method, however, has only been recently reintroduced as a noninvasive technique to alter cortical activity in humans. This method has been used in different tasks to modulate behaviour, perception and cognition. Transcranial direct current stimulation (tDCS), as a specific type of transcranial electrical brain stimulation, has been successfully applied in modulation of different types of memories, e.g. working and declarative memories. It has been shown that it can be effective for both patients and healthy participants. It can improve, as well as impair memory capability based on the application protocol. In this talk I am going to review advances during the last decade in the modulation of memory using tDCS.
Saturday, 17:30
Dr. Simone Kühn (Max Planck Institute for Human Development, Berlin)

Memory enhancement by computer training

The rapid growth of computer game popularity in adolescents has generated concern among practitioners, parents, scholars and politicians, however besides detrimental effects of violent computer games in the social domain, also favorable effects of frequent computer game playing have been observed. Computer games allow repeated, sometimes rewarding, training of various mental tasks with variation and interactivity. While improved performance on the tasks inside the games is unsurprising, they may also be able to transfer their effects to other cognitive domains or enhance general cognitive abilities. In particular computer training of working memory tasks has been proposed to show transfer effects.

On the neural level, increases in striatal activity have been suggested to mediate training-related improvements in working-memory ability. We investigated the temporal dynamics of changes in task-related brain activity following training of working memory. Participants in an experimental group and an active control group, trained on easier tasks of a constant difficulty in shorter sessions than the experimental group, were measured before, after about one week, and after more than 50 days of training. In the experimental group an initial increase of working-memory related activity in the functionally defined right striatum and anatomically defined right and left putamen was followed by decreases, resulting in an inverted u-shape function that relates activity to training over time. Activity increases in the striatum developed slower in the active control group, observed at the second posttest after more than 50 days of training. In the functionally defined left striatum, initial activity increases were maintained after more extensive training and the pattern was similar for the two groups. These results shed new light on the relation between activity in the striatum (especially the putamen) and the effects of working memory training, and illustrate the importance of multiple measurements for interpreting effects of training on regional brain activity.
A critical issue in the area of psychopharmacology and neuroscience pertains to the development and use of drugs to enhance cognitive capacities as in the case of memory. At the neural level, memory enhancement has been attributed to the activity of a larger number of synapses or neurons and higher level of neurogenesis. Thus drugs aimed at stimulating relevant neurotransmitters at the synapses connecting neurons, which is also believed to improve the efficiency of brain circuits and strength of memory consolidation in the pathway between the hippocampus and prefrontal cortex, have been advocated. In order to gain insight into the neural mechanisms of memory enhancement and to test hypotheses about drugs aimed to its improvement, a computational modeling approach that considers synaptic and neural ensembles embedded into a cortical patch, is proposed. It is concluded that modeling represents a useful approach to overcome limitations inherent in experiments.
Memories enhance by mnemonic techniques

Individuals with superior memory abilities are interesting for research. Memory athletes are of particular interest due to their extreme performances demonstrated at regular memory championships. Records of human memory performances have gone up a lot in the last years. We have studied a group of memory athletes, who all ranked within the Top 50 of the World ranking list, using various tests and methods including fMRI and sleep EEG. All of them have in common that they studied and heavily practiced specific mnemonic techniques. It has also been shown that training in mnemonic techniques allows normal subjects to improve memory capacity; however the necessary volume of training to achieve outstanding results has been named as a limiting factor for research. We therefore addressed the question whether or not a superior memory can be achieved in limited time in a group training setting. We provided a group of students with a structured training in mnemonic techniques including the phonetic memory and the method of loci over the course of just a weekend and compared their performance in a digit-span task and a self-paced digit memorisation task with a control group. The resulting data shows that the subjects could significantly increase their digit-span. In another study subjects had to practice the techniques for 20 hours over a period of six weeks following the initial training. All of them further increased their results in digit memorization tasks – the best participants even reached results comparable to average memory athletes. These findings suggest that memory can be improved by mnemonic techniques faster and further than usually anticipated. The use of mnemonic training allows for further studies on the basic principles of superior memory performances.
Memories, after they are encoded, are not carved in stone. Their brain representations and their content can change. On the one hand, forgetting leads to a diminution of the memory trace and reduced recall. On the other hand, consolidation processes work on the systems and synaptic levels of the brain to strengthen memories. Furthermore, there is evidence that the brain also reprocesses the content of memories, which can lead to changes in what we remember. Sleep is a state that is particularly suited to process stored information. During sleep, changes in neurotransmitter activity switch the brain into a different mode of functioning and shut it off from external input. Recently acquired memories can then be reactivated or “replayed”. This is supposed to provide additional practice of the material, leading to stronger memory. In addition, it is assumed that (hippocampal) episodic memory becomes integrated into (neocortical) semantic networks during sleep. It has also been shown that memories are changing over sleep, e.g. with regard to the emotionality of memory content, that sleep helps selecting which information will be retained, and that ongoing processing during sleep can lead to solutions for problems that were placed in memory before sleep. Together, research has shown that sleep does not shut down processing in the brain, but, on the contrary, it is a period actively transforming and strengthening our memories.
According to a widely held concept, the formation of long-term memories relies on a reactivation and redistribution of newly acquired memory representations from temporary storage to neuronal networks supporting long-term storage. This process of system consolidation takes place preferentially during sleep as an “off-line” period during which memories are spontaneously reactivated and redistributed in the absence of interfering external inputs. I will present data showing that these sleep-dependent memory formation processes can be enhanced by triggering memory reactivation during sleep using reminder cues, leading to a strengthening of memory traces. In addition, I will compare reactivation during sleep with memory reactivation during wakefulness, which leads to a transient destabilization of memory traces. I will propose that reactivation serves distinct functions in the maintenance of long-term memories depending on the brain state, allowing for updating of existing memories during wakefulness and integration of newly acquired knowledge in pre-existing knowledge networks during sleep.
Sunday, 11:20
Prof. Dr. Thomas Metzinger (University of Mainz)

**Neuroethics of pharmaceutical cognitive enhancement: The first ten years**

An evaluating survey of the development of the neuroethics of pharmaceutical cognitive enhancement (PCE) during the last decade, focussing on the situation in Germany, has been undertaken. The talk presents the most important conceptual problems, current substances and central ethical and legal issues. Very first guidelines and recommendations for policy-makers are formulated.
Sunday, 13:00
Dr. Anders Sandberg (University of Oxford)

The ethics of memory enhancement

Human memory is fallible, and since time immemorial people have both complained about it and tried to enhance it. Merely amplifying capability does not imply an enhancement: to be valuable memory must fit in with the ability of the person to live a good life. Memory enhancement can be instrumentally good (in order to enhance the ability to do important tasks or to make proper moral judgements) or be seen as a goal in itself (because personal memory itself might be valuable). In addition other considerations also matter ethically: what kinds of interventions are acceptable, what the impact on individuals and society might be, and the proper management of the practice. This talk will give an overview of these issues.
The human brain is in great part what it is because of the functional and structural properties of the 100 billion interconnected neurons that form it. These make it the body’s most complex organ, and the one we most associate with concepts of selfhood and identity. The assumption held by many supporters of human enhancement, transhumanism, and technological posthumanity seems to be that the human brain can be continuously improved, as if it were another one of our machines. In this talk, I focus on some of the ethical issues that we should keep in mind when thinking about memory enhancement interventions. I start with an overview of one of the most precious capacities of the brain, namely memory. Then I analyze the different kinds of memory interventions that exist or are under research. Finally, I point out the ethical issues that we should not forget when we consider enhancing our memories. In this regard, my argument is not against memory enhancement interventions; rather, it concentrates on the need to “keep in mind” what kind of enhancements we want. We should consider whether we want the kind of “enhancements” that will end up making us lose synapse connections, or the kind that promote more use of them.
Sunday, 14:20
Christoph Bublitz (University of Hamburg)

A duty to remember, a right to forget?
Legal issues in memory enhancement and modification

Many neurotechnologies raise legal questions about the scope and limits of cognitive liberty or individual self-determination over one’s mental states. While the inward working of one’s mind should, by large, be considered off-limits for states and governments, the preservation of memories and access to them might be an exception. At least for purposes of security and law-enforcement, there has always been a heightened interest in memories (e.g. of witnesses), and there may be further social interests calling for legal regulations of what people remember or forget (think about soldiers blunting memories). This talk will sketch some of the new legal issues and provide some ideas for discussion.
A theoretical investigation of principles of justice as criteria for assessing cognitive enhancement

The author conducts a brief conceptual analysis of the debate on psycho-pharmacological cognitive enhancement from the point of view of public reason as it is construed in contemporary political philosophy. The research points toward the conclusion that the strong reasonable disagreement that marks utility, authenticity, posthumanist and “playing God” arguments stems from their presuppositions in religious, ethical or metaphysical comprehensive doctrines. On the other hand, the principles of justice could be the basis for an “overlapping consensus” in the context of regulating use of cognition enhancement drugs for non-therapeutic purposes.

Using the method of reflective equilibrium, the author starts from the considered judgment of many citizens that treatments are obligatory and permissible while enhancements are not, and with the application of general principles of justice explains why this is the case. Cognitive enhancement is not an issue of providing basic necessities for those who are lacking, benefiting the least advantaged or restoring citizens to a position of equal opportunity, while therapy certainly is. Furthermore, if widespread, the use of enhancements by healthy adults would more likely maintain or increase than reduce social inequality. Moreover, cognition enhancement drugs are already used as means for obtaining undeserved positional advantage and with the unknown long-term side-effects and/or through coercion could create additional disadvantages and needs for citizens lacking basic necessities.

The author further analyzes four reasons that some influential authors in the field of neuroethics might have for downplaying the importance of justice: 1. the issue of justice allegedly leads to a reasonable disagreement as well as it is loaded with political ideology; 2. justice applies only to public funds and state action - not to individual choice or corporate actors; 3. “performance enhancement” does imply questions of justice, while “performance maintenance” does not; and 4. there is no sufficient difference between psycho-pharmacological enhancement and other technologies to warrant the importance of justice. The liberal, socialist and conservative principles of social justice in a democratic society are elaborated, and the basic content of justice for the idea of public reason is clarified in order to refute the first challenge. The other challenges are refuted by taking into account the difference between consumption and tool-use, and the influence of socioeconomic pressure for widespread use that existing drugs could have on the basic structure of society and equal autonomy of citizens.

The author concludes by claiming that discouraging the use of psycho-pharmacological enhancements with the imposition of taxes, fees and requirements of additional insurance would be the most legitimate public policy. Such regulations would make the use and indirect coercion to use less profitable and less widespread, while additional funds thus created could be allocated to meet basic medical needs of citizens and/or to finance education.
Neuro-enhancement as part of human freedom and personal responsibility: A theological-philosophical reflection

It is currently possible to enhance the human body or mind beyond a level of “health” by means of pharmaceutical intervention. Enhancement beyond actual human capabilities by means of technological intervention is within reach in the foreseeable future – a potential exemplified by a single patient therapeutic trial of deep brain stimulation deploying, for the first time, 24 electrodes in 3 brain areas in a patient with a treatment-resistant major depressive disorder (a trial evaluated by the author in his masters’s thesis). Ethical discourse has voiced several reservations about Neuro-Enhancement – reservations about perfecting humans, change in personality, social disparity et cetera. Theological ethics in particular has argued that we should accept the limitations of human beings and that man should not be his own Creator. However, it is the author’s proposition that Memory Enhancement, when in accordance with certain ethical principles, may actually merit support from a theological-philosophical perspective – particularly where it can help increase the quality of life. From a theological point of view, humans obtained the talent to lead their life freely and responsibly. It rest on humans to deal responsibly with the use of Memory Enhancement. Evolutionary theory tells us that humans have always developed and enhanced themselves in a co-evolution of nature and culture in order to meet life’s challenges autonomously. The use of Memory Enhancement is a part of this development and shows that it can help to lead a responsibly human life.