Not Extended, but Enhanced:

Internal Improvements to Cognition and the Maintenance of Cognitive Agency[1]

Abstract:

This chapter will address the axiological objection to cognitive enhancement, which is that the use of cognitive enhancers reduces the value of cognitive achievement. In a recent defense of cognitive enhancement, Carter and Pritchard (2019) utilize the extended mind hypothesis to argue that cognitive enhancers do not compromise knowledge acquisition. In this chapter, it will be demonstrated that the reliance on the extended mind hypothesis leaves some cognitive enhancers vulnerable to the axiological objection. To expand the scope of the argument, it will be shown that criteria for cognitive integration are applicable even to enhancers that cause changes to cognition internal to the human organism. This chapter will begin with a description of several cognitive enhancers and with the identification of the type of cognitive process they purportedly improve. It will then be demonstrated that even when those improvements are internal, they need not affect cognitive character and do not compromise cognitive agency.

Introduction

Cognitive enhancement refers to the variety of medical and technological means of improving cognition. Some of the most familiar cognitive enhancers are pharmacological, including

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methylphenidate and dextroamphetamine, both of which have medical uses for the treatment of attention deficit and hyperactivity disorder (ADHD), but have also shown, modest and often variable, efficacy in improving cognition in typically functioning individuals, i.e., individuals not diagnosed with any psychiatric, neurological, or medical conditions known to cause cognitive impairment (Elliott et al., 1997; Izquierdo et al., 2008; Mehta et al., 2000). Additional means of cognitive enhancement have been considered in terms of their potential to improve human cognition, including genetic modification (Persson & Savulescu, 2008) or the use of brain technology, such as deep brain stimulation (DBS).

There are a variety of ethical considerations related to the use of purported cognitive enhancers, including the very important questions related to safety and efficacy of the use of cognitive enhancers as well as the use just allocation of resources (Farah et al., 2004). A more recent debate related to cognitive enhancers is concerning their impact on cognitive character and the acquisition of knowledge (Carter & Pritchard, 2019; Gordon & Dunn, 2021; Wang, 2021). This current debate centers on the question of whether use of cognitive enhancers threatens the maintenance of cognitive character in a way that would compromise the epistemological justification of beliefs. In order to respond to the this objection, Carter and Pritchard (2019) propose that the extended mind hypothesis, an argument originally proposed by Clark and Chalmers (1998), can be used to argue that at least some types of cognitive enhancers can become properly incorporated into our cognition and therefore would not undermine cognitive character. The extended mind hypothesis is the argument that cognition

extends past the biological boundaries of the human organism to incorporate external tools, e.g., notepads and smartphones, into human cognitive processes.

In this chapter, I argue mostly in agreement with Carter and Pritchard (2019) that the use of cognitive enhancers need not undermine cognitive character. However, I will demonstrate that at least some cognitive enhancers cannot be likened to external tools, i.e., they are not akin to notepads or smart phones. I will then argue that in order to expand the argument that cognitive enhancers do not undermine the epistemological integrity of knowledge to most cognitive enhancers, we need to show that the argument for cognitive integration does not require the extended mind hypothesis. In this chapter, I demonstrate that the use of cognitive enhancers that cause changes to cognition within the boundaries of the human organism can be properly incorporated into cognition and do not violate cognitive character or undermine knowledge acquisition.

1. Varieties of Cognitive Enhancers

Carter and Pritchard (2019) introduce Google Glass, among others, as a potential cognitive enhancer. Smart glasses, as smart phones, makes easily available stores of information to an individual using this wearable technology. This technology is not inner to the human body, as it is not within the confines of the skull; instead, it is part of the environment that becomes incorporated into an individual's cognitive processes. Google Glass, for example, can utilize facial recognition technology to identify any individual one walks past on the street and immediately make all the publicly available information about that individual accessible to the

Google Glass user. This information can then be used to modify behavior, e.g., either to start a conversation or to cross the street. But many purported cognitive enhancers, including pharmacological enhancers, are not akin to Google Glass because they do not modify the knowers environment; rather, they directly modify the individual's endogenous cognitive abilities.

Before I describe some of those cognitive enhancers and the data that supports their efficacy, I wish to note that biomedical enhancement has been used to designate improvements in typically functioning individuals. This can be contrasted with the use of medicine for the restoration of typical function in people with cognitive deficits due to psychiatric, neurological or medical disease. This designation of certain medical or technological means as enhancers has often been made utilizing a distinction between uses of medicine for the treatment of disease, i.e., restoration of typical function, and its uses for enhancement, i.e., improvements of function for typically functioning individuals. The treatment and enhancement distinction has been controversial. Some of those who challenge the distinction point out that normality, often used to designate typical functioning, is a normative term that cannot be defined using purely biological means (Daniels, 2000), and others argue that all medicine is aimed at enhancing human functioning (Synofzik, 2009). Despite this controversy, I will maintain the usual use of cognitive enhancers to designate the medical or technological means of improving cognition in typically functioning individuals. In what follows, I will describe the array of potential cognitive enhancers that modulate cognitive capacities by relying on different mechanisms and that vary in their level of invasiveness. Some require the taking of medication that alter cognition by

modulating the biological underpinning of cognitive faculties, such as memory, attention, or learning. Other potential enhancers are more invasive, and might require the use of a wire lead inserted into a particular area of the brain in order to provide deep brain stimulation (DBS) to alter aspects of neurological function. I will primarily present evidence for the claim that these medical interventions have an effect on the cognitive functioning of individuals without diagnosed neurological or psychiatric conditions, i.e., that they have the potential for being used as cognitive enhancers.

Many purported cognitive enhancers have established medical uses for the treatment of a number of conditions that might improve cognitive function. Some of those enhancers are pharmacological. For example, two often studied enhancers are methylphenidate and dextroamphetamine. Both of these drugs are approved by the Food and Drug Administration (FDA) for the treatment of ADHD, but there have been a number of studies testing the effectiveness of these agents in improving aspects of cognition in normal individuals. Elliot et al. (1997) studied the effects of methylphenidate in 28 healthy volunteers. The participants were given a battery of tests, including tests for spatial working memory, planning, verbal fluency, and attention. Use of methylphenidate was shown to improve spatial working memory on some tasks, although the improvements were only seen when the task was novel. In repeated performances of this task, the stimulant seemed to be detrimental to performance, and individuals who did not take the drug performed better on the task of spatial working memory. The drug had no effect on verbal fluency or attention (Elliot et al. 1997). An additional study by Mehta et al. (2000) showed methylphenidate to produce improvements in working memory.

Those were most prominent for individuals who started with a lower baseline of working memory prior to the administration of the drug. Izquierdo et al. (2008), investigated the effects of methylphenidate on memory decline using two types of memory tasks: one was focused on retention of incidental information, such as details of a recently viewed movie, and the other was a formal memory test where participants were asked to learn and recall new information. The study investigated memory in human volunteers ages 16-82. Younger individuals between ages 16-30 did not experience significant memory loss even 7 days after the initial event. However, individuals older than 41 demonstrated a significant decline in recall on both memory tests. Methylphenidate was shown to mitigated that loss in older participants. In particular, methylphenidate was studied in individuals older than 35 and was shown effective in mitigating memory loss on the formal memory task in that group.

A further class of drugs with the potential for use as cognitive enhancers is acetylcholinesterase inhibitors, including donepezil, galantamine, and rivastigmine. Out of these three the one most studied for its enhancing properties in normal individuals is donepezil, which is FDA approved for the treatment of Alzheimer's disease (AD). Yesavage et al. (2002) performed a randomized, double-blind study of 18 licensed pilots ranging from 30 to 70 years of age to determine the effects of donepezil on the long-term retention of skills required for aviation after training in a flight simulator. The study showed that pilots treated with donepezil retained their ability to perform the set of complex flight related tasks even a month after training while pilots treated with placebo experienced deterioration in performance (Yesavage et al. 2002).

A yet another potential neuroenhancer is modafinil, which has been FDA approved for the treatment of narcolepsy, but has been prescribed off label for a variety of sleep disorders, including sleep apnea. Two studies on normal healthy adults showed that modafinil could be successfully used to abate the negative effects of sleep deprivation. In a study by Grady et al. (2010), healthy patients underwent a protocol in which the period of sleep-wakefulness was significantly different from their usual. The participants remained awake for longer and slept fewer hours. The study was a randomized double-blind, placebo-controlled study. The participants who received modafinil for the duration of the experiment were better able to remain awake and alert. Modafinil was particularly efficacious in improving cognitive psychomotor speed and attention. Furthermore, a study of sleep-deprived physicians showed that modafinil was successful in diminishing the cognitive deterioration associated with sleep deprivation (Sugden et al., 2012).

In addition to these pharmacological means of cognitive improvement, Savulescu and Person (2008) explore the potential of genetic modification as a mode of cognitive enhancement. For example, they had suggested that genetic memory enhancement, currently demonstrated only in animal models, might have translational application and result in human genetic modification to improve memory formation and retention.

Recently, there has been research on the underlying circuitry of learning and memory that has enabled research to identify potential areas of the brain (the fornix or nucleus basalis of Meynert) that if targeted by deep brain stimulation (DBS) could have potential therapeutic

benefits in improving memory. The data from animal models demonstrates that the use of DBS can induce neuroanatomical, neurophysiological, and neurochemical changes within the memory circuits (Lozano et al., 2019). These changes in turn have sometimes resulted in improvements in memory and amelioration of memory loss in rodent models (Aldehri et al., 2018).

There have been clinical studies investigating the effectiveness of DBS in delaying and restoring cognitive decline in patients with AD. The use of DBS far patients with Parkinson's has been associated with cognitive decline, although it is not clear whether that decline was due to DBS or because patients treated with DBS no longer take dopaminergic drugs used for the treatment of Parkinson's which can support certain aspects of cognitive functions. However, there have also been small and early studies of DBS for the treatment of AD. In these studies, some patients experienced changed at the neurological level, such as increase in size of the hippocampus, but evidence of improvements at the clinical level is still lacking (Aldehri et al., 2018).

2. The Extended Mind Hypothesis

The use of cognitive enhancers has been controversial. For example, Kass (2003), Sandel (2004), and Harris (2011) have argued that cognitive enhancement removes the obstacles required for cognitive achievement in a way that renders the acquisition of knowledge trivial. Carter and Pritchard (2019) call this the axiological objection. Based on the axiological objection, cognitive enhancers diminish cognitive achievement by improving our biological ability to remember or

by supplementing memory through the use of smart technology and thereby removing the difficulty inherent in mastering any given subject.

The basis for the argument in defense of cognitive enhancers has emerged from a strain of epistemology which identifies cognitive agency as a way of justifying true beliefs. Based on this view, an individual has cognitive agency only when the acquisition of true belief can be properly credited to that knower. To clarify the notion of cognitive agency one can liken it to moral agency. In order for an individual to be credited with a good deed, they should be primarily or mostly responsible for it. For example, if I attempt to buy a lottery ticket but inadvertently donate to charity, most would argue that I should not be credited with a good deed. Similarly, if I come by a true belief without properly utilizing any of my cognitive abilities to acquire it, I should not be credited with knowledge.

An example of this view is proposed by Pritchard (2010), who argues that knowledge is a product of a reliable cognitive process appropriately integrated into an individual's cognitive character. A reliable cognitive process is one that regularly leads to true beliefs, such as perception, memory, or problem solving. Furthermore, a cognitive process is appropriately integrated into the person's cognitive character when the beliefs it generates can be credited to that individual's agency, i.e., when the individual is responsible for obtaining that belief. The notion of cognitive character is akin to the notion of psychological or moral character, where an individual develops a stable cognitive character as they might develop a stable moral character.

The emphasis on cognitive agency aims to avoid attributing knowledge to individuals who for internal or external reasons cannot take responsibility for their beliefs. An individual might acquire a true belief due to environmental luck, which is when features of their environment are favorable to acquiring justified true belief even if the individual is not gaining them by utilizing their cognitive abilities. For example, this could be because every time you look at a broken clock unbeknownst to you a stealth helper has adjusted the clock to reflect the correct time in your part of the world. Or, it could be, due to factors internal to the agent that obviate the need for the agent to apply their cognitive abilities. For example, an individual who is unaware that they have a brain implant that automatically generates accurate belief about external temperature. In both those cases, the individual, according to Pritchard (2010), has true beliefs, but they do not have knowledge because the belief forming process does not exploit the individual's cognitive agency.

According to both Pritchard (2010) and Carter and Pritchard (2019), this view of knowledge does not exclude the possibility that at least some types of enhancers could become incorporated into an individual's character. To argue that cognitive enhancers can becomes incorporated into cognitive character, they rely on the extend mind hypothesis, which is the view that some external tools, such as notebooks, smart phones, or Google Glass, when properly incorporated into our cognition can be said to become part of cognition. If that is the case, then cognitive enhancers can become incorporated into an individual's cognitive process and therefore become part of their cognitive character.

The extended mind hypothesis was originally proposed by Clark and Chalmers (1998) who argue for a thesis called active externalism, which is the view that the environment plays a role in cognitive processes. This view is not merely that elements of the environment may provide data for cognitive processes. Instead, it is the view that parts of the environment become incorporated into our cognition--the mind becomes extended to include parts of the environment. To support this claim, Clark and Chalmers (1998) describe the example of Otto, who because of his waning memory compensates for this loss by writing down into a notebook information he can no longer remember. When he needs to, Otto utilizes his notebook to gain access to facts that aid him in making decisions regarding what to do next, such as how to get to the grocery store. Clark and Chalmers argue that Otto's notebook becomes part of his cognition.

Based on the criteria proposed by Clark (2008, p. 79), an environmental resource can become incorporated into an individual's cognition if it fulfills the following criteria:

1. "That the resource be reliably available and typically invoked."
2. "That any information thus retrieved be more-or-less automatically endorsed. It should not usually be subject to critical scrutiny. [...] It should be deemed about as trustworthy as something retrieved clearly from biological memory."
3. "That information contained in the resource should be easily accessible as and when required."
4. "That the information in the notebook has been consciously endorsed…"

Otto's notebook fits all criteria. Otto carries his notebook with him and has reliable access to it, i.e., he can look up information without much difficulty. And he uses it in his daily life whenever he needs to reach the grocery store or to purchase the right items when in the store. Otto endorses the information in the notebook and does not, for the most part, question the veracity of the information contained within it. Hence, Otto's notebook has become a reliable part of Otto's cognitive process as it has assumed the functional role of Otto's biological memory. This particular thesis of cognitive integration is used to argue for the view that tools external to the person's biological organism can become incorporate into their cognitive function. This argument can then be used to argue that certain types of cognitive enhancers can become incorporated into an individual's cognitive character, rebuffing the view that use of enhancers in some way undermines cognitive agency. But it can be also be used to expand the boundaries of cognitive enhancers to include things like smart phones or smart glasses.

3. Internal Enhancement

In section II, I described a variety of potential cognitive enhancers, including pharmacological enhancers, genetic enhancers, DBS, and Google Glass. The axiological objection, in particular as endorsed by Kass (2003), is an objection to the use of medicine for human improvement and would apply to the use of any cognitive enhancers and not just to enhancers that modulate our environment. The extend mind hypothesis provides a direct defense only for the type of cognitive enhancers that would extend our cognition beyond the anatomical confines of the skull. The active externalism thesis leaves some cognitive enhancers open to the attack from the axiological objection. For example, pharmacological enhancements and genetic enhancers

aim to improve cognitions by changing our biology not by changing any aspect of our environment. These enhancers would not fit into the framework of the extended mind hypothesis as they are changes internal to the organism.

Clark and Chalmers (1998) take it for granted that something akin to DBS, i.e., a brain implant, is part of Otto's cognition because it is within the skull. They use this example to identify a prejudice in favor of internalist views of cognition. They argue that most of us would not hesitate to accept that a brain implant would become part of Otto's cognition, but some of us would object to the claim that Otto's notebook would be incorporated into his cognition. So, perhaps, those using the extended mind hypothesis in order to reject the axiological objection take it for granted that internal modification to cognition, i.e., modifications made within the boundaries of the biological human organism need not be defended against the axiological objection. But some of the proponents of this objection, such as Kass (2003), would not distinguish between enhancers based on the internalist/externalist distinction; instead, they would distinguish the permissible use of medicine and technology based on the treatment and enhancement distinction. Which would mean that Otto using DBS to improve his memory because of the advancing of AD would be justified, but the use of DBS to improve cognition, if Otto were healthy and his memory were intact, would not be permissible.

One could put this in terms of preserving cognitive character in the following way: neurological disease changes an individual's cognitive character, e.g., by affecting their memory, and use of medicine is permissible in such cases because it contributes to the restoration of the original

cognitive character. Use of medicine and technology to change cognitive character, however, is not permissible. And this is true whether such changes are internal or external to the individual. Thus, in order to argue against the axiological objection, one needs to show how cognitive integration can be used to justify cognitive improvements *tout court*. This means that one needs to show that use of cognitive enhancers would not affect cognitive agency even when enhancement causes internal changes to cognition. Luckily, this argument could be made by relying on Pritchard's view of cognitive agency.

Pritchard (2010) defines cognitive agency thusly: "S knows that *p* iff S's true belief that *p* is the product of a reliable belief-forming process which is appropriately integrated within S's cognitive character such that her cognitive success is to a significant degree creditable to her cognitive agency" (Pritchard 2010 224).[2] To assess, which of the cognitive enhancers fit Pritchard's criteria and can be used without violating cognitive character, let's imagine a detective who is contemplating the use of cognitive enhancers in order to resolve a backlog of cold cases.

[2] Pritchard distinguishes between weak and strong versions of the cognitive agency thesis. The one cited in this chapter is the weak version of the definition, which is the one he thinks can accommodate the extended mind thesis. The distinction between the two definitions is regarding the degree to which knowledge can be credited to an individual's agency. The weak version requires that it be significantly creditable and the strong that it be primarily or mostly creditable to the individual's cognitive agency. The distinction does not have particular bearing on my argument, so I will just adopt the week version of it as this is the version Pritchard seems to favor.

Let us begin by imagining that the detective decides to use modafinil first. Modafinil has been shown to be effective in diminishing the deleterious effects of fatigue on cognition. Hence, the use modafinil would not improve the detective's cognition in any particular way; it would merely maintain the detective's usual cognitive function even if she decides to work through the night in order to solve a case. The detective would be able to rely on her usual mode of solving cases: she would rely on the usual belief-forming processes and would be able to provide an explanation for her endorsement of certain beliefs related to the case. Modafinil would enable her to maintain her cognitive character for longer. Thus, the use of modafinil would not violate cognitive integration. Modafinil would also not introduce external influences that would violate the detective's cognitive agency because none of the environmental facts within which the detective is working would be altered. She would not be given the medication surreptitiously, as she is choosing to take it herself, so the maintenance of her cognitive character despite sleep-deprivation could be credited entirely to her agency.

The other two potential pharmacological cognitive enhancers that the detective could use are the stimulants dextroamphetamine and methylphenidate. The effects of dextroamphetamine include improvements in spatial working memory, planning, verbal fluency, attention, and long-term memory. The use of these stimulants for cognitive enhancement would similarly not violate cognitive character or cognitive agency because they do not alter the repertoire of cognitive processes that would be brough to bear in order to solve a case or to accomplish a particular task. If spatial working memory is required in order for the detective to survey a scene of the crime and to derive clues from it, the use of stimulants would merely improve this

ability in a way that might increase the likelihood that the detective would identify a clue that helps resolve the murder. This, on its own, would not violate any cognitive habits the detective has. She would still rely on the same belief forming processes and would still be able to justify why a particular item in the scene of the crime would count as a clue and how that clue, e.g., a piece of ripped fabric, would help identify an individual with a torn jacket as the murderer. Again, as is the case with modafinil, the changes directly caused by the cognitive enhancer would be internal to the detective's biological organism and would not be due mostly to changes in the environment.

In addition, the data about the benefits of stimulants on cognition does not indicate an outsized improvement in cognitive ability. Much of the studies on stimulants were placebo-controlled, which means that the improvement of performance was compared to a control group that had not received stimulants. Although the differences in performance between the two groups were statistically significant, they did not show prodigious improvements in performance. The benefits that each individual might experience from the use of stimulants can vary, but most would not experience an extreme increase in performance that would violate cognitive character. For example, if we imagine that the detective has bad episodic memory and with the help of medication becomes able to remember everything that ever happened to her, an increase in performance might cause a change in cognitive character. For one, the detective might be suspicious of this sudden endowment, and not lend automatic endorsement to beliefs formed based on her episodic memories. In addition, being able to remember everything might affect the detective's usual way of obtaining knowledge, she might stop relying on memory

aids, e.g., taking notes. In addition, she might adopt a different cognitive style where more of her problem-solving would become reliant on her episodic memory. But the use of dextroamphetamine and methylphenidate regrettably does not result in such large improvement. But even if they did, this would not be a determinative argument that use of stimulants changes cognitive character in a way that is different from the likely changes that might occur naturally overtime. For example, as the detective becomes more experienced solving cases, her cognitive character-- e.g., the way she approaches case, what types of clues she identifies--might adapt and change overtime.

Beyond pharmacological enhancers, the detective will run out of options for currently available cognitive enhancers. But I will consider whether some proposed, but not yet available, enhancers would violate cognitive integration. Person and Savulescu (2008) cite genetic modification to improve memory. Assuming that these changes would occur by modifying the genome either at the embryonic stage or early in childhood, these changes would be present at the early stages of development of cognitive character. They would be part of a genetically modified individual's cognitive development and would be at least partially determinative of the individual's cognitive character from the outset of their life. One could, of course, object to genetic modification for reasons unrelated to epistemic integrity of beliefs, including concerns related to the risks associated with genome modification and the opposition to the modification of the human genome especially when it might cause heritable changes. Such objections, however, are outside of the scope of this chapter.

I will now turn to improvements in cognitive function that requires direct modification of neurological function, such as DBS. DBS requires the insertion of a wire lead with electrodes into a specific area of the brain, the lead is connected to a neurostimulator that is implanted near the patient's collarbone. It is this neurostimulator that sends electrical signals to the implanted lead. When used to treat Parkinson's disease, the neurostimulator has to be adjusted by medical professionals in order to obtain optimal stimulation for symptom relief. The patient can, however, turn the stimulator on or off. Thus, the actual neurostimulator could be characterized as being external to the individual both because it is not contained within the skull and because the locus of control is external. In addition, there is some evidence that, at least phenomenologically, the externality of the stimulation affects the individual using DBS as they report feeling as though they are remotely controlled (Agid et al., 2006) and that they do not feeling quite like themselves (Agid et al., 2006). These facts about DBS could support a disanalogy between Otto with the notebook, who has control over this cognitive aid, and Otto with a stimulator, who does not directly control his stimulator.

This then complicates the question of whether DBS should fall under the category of an internal cognitive modulator or whether it should be considered also as an external tool that becomes incorporated into an individual's cognition. If we use the draw the line between internal and external influences based on modification of biological function, the stimulator works by disrupting the endogenous electrical signals that result in neurological symptoms, so in that regard it could be characterized as modifying the biological function of the brain just like pharmacological treatments. On the other hand, one could argue that because the lead has to

be inserted into the brain to modify the signal, DBS does not modify endogenous neurological function in the same way pharmacological interventions do. DBS could be characterized as changing the input to certain areas of the brain, which perhaps makes it more akin to a pair of glasses which modify vision by changing the refraction of the light rather than changing how the light is processed within the visual system. Based on this view, DBS could probably be more aptly characterized as an element of the environment being inserted into the skull instead of being characterized as an internal cognitive resource. Hence, depending on which view one takes, DBS could be characterized as either an internal or an external modification to cognitive function.

Given that I have adopted the criterion of cognitive integration to determine whether cognitive character would undermine cognitive agency, I can then just assess to what extent DBS is likely to threaten the maintenance of cognitive character. Given that at least some individuals who utilize DBS for the treatment of Parkinson have reported psychological changes that indicate that the use of DBS is not seamlessly incorporated into an individual's psychology, there might be reason to think that perhaps DBS used to improve cognition might affect cognitive character. If the individual, for example, feels like they are being externally controlled, this might undermine their inclination to automatically endorse beliefs formed after the activation of the device. But making this judgment would be too rash given the current paucity of empirical evidence about the nontherapeutic effects of DBS, especially as it might pertain to its uses to improve cognitive function. Only some number of patients who have utilized DBS for the treatment of Parkinson's have reported feelings of diminished control and there is no evidence

that speaks to whether this feeling actually interferes with cognitive integration. After all feelings of alienation might not have any corollary effects on how individuals justify beliefs. In order to determine whether DBS could undermine cognitive integration, more empirical work would be needed. There are no *a priori* reasons, however, to assume that such integration could not happen for many patients if DBS ever becomes available as a memory enhancer.

Conclusion

The extended mind hypothesis, as utilized by Clark and Pritchard (2019), is aimed at defending the use of cognitive enhancers against the axiological objection. The reliance on the extended mind hypothesis, however, limits the scope of the argument for enhancers to only those interventions that are external to the human organism. I argued that it is important to broaden the defense to include cognitive enhancers that effect primarily internal changes to the organism. In particular, I argued that at least some proponents of the axiological objection also endorse the treatment and enhancement distinction and utilize it to adjudicate the use of medicine and technology along those lines, whereby the use of enhancers to restore normal function is deemed permissible but their use to improve normal function is not. To defend the use of enhancers more broadly, I argued that we should provide a defense of cognitive enhancement that does not rely solely on the extended mind hypothesis. In this chapter, I considered a number of different enhancers, including those that change cognition by changing the knowers environment, but I focused on pharmacological enhancers that effect cognitive change within the boundaries of the human body and leave the environment intact. I argued that a defense of cognitive enhancers that relies on the extended mind hypothesis leaves open

to attack the use of enhancers that are, arguably, the most likely to become available for current use, e.g., methylphenidate and dextroamphetamine. I proposed instead that the criteria of cognitive integration proposed by Pritchard (2010) can be used to defend most cognitive enhancers that do not undermine cognitive agency regardless of whether they promote internal or external changes to the organism.

References:

Agid, Y., M. Schüpbach, M. Gargiulo, et al. (2006). Neurosurgery in Parkinson's disease: The doctor is happy the patient less so? *Journal of Neural Transmission*, 70: 400–414.

Aldehri, M., Temel, Y., Alnaami, I., Jahanshahi, A., & Hescham, S. (2018). Deep brain stimulation for Alzheimer's Disease: An update. *Surgical Neurology International*, *9*, 58. https://doi.org/10.4103/sni.sni_342_17

Carter, J. A., & Pritchard, D. (2019). The Epistemology of Cognitive Enhancement. *The Journal of Medicine and Philosophy: A Forum for Bioethics and Philosophy of Medicine*, *44*(2), 220–242. https://doi.org/10.1093/jmp/jhy040

Clark, A. (2008). *Supersizing the Mind: Embodiment, Action, and Cognitive Extension*. https://doi.org/10.1093/acprof:oso/9780195333213.001.0001

Clark, A., & Chalmers, D. (1998). The Extended Mind. *Analysis*, *58*(1), 7–19.

Daniels, N. (2000). Normal Functioning and the Treatment-Enhancement Distinction. *Cambridge Quarterly of Healthcare Ethics*, *9*(3), 309–322. https://doi.org/10.1017/S0963180100903037

Elliott, R., Sahakian, B. J., Matthews, K., Bannerjea, A., Rimmer, J., & Robbins, T. W. (1997). Effects of methylphenidate on spatial working memory and planning in healthy young adults. *Psychopharmacology*, *131*(2), 196–206. https://doi.org/10.1007/s002130050284

Farah, M. J., Illes, J., Cook-Deegan, R., Gardner, H., Kandel, E., King, P., Parens, E., Sahakian, B., & Wolpe, P. R. (2004). Neurocognitive enhancement: What can we do and what should we do? *Nature Reviews Neuroscience*, *5*(5), 421–425. https://doi.org/10.1038/nrn1390

Gordon, E. C., & Dunn, L. (2021). Pharmacological Cognitive Enhancement and Cheapened Achievement: A New Dilemma. *Neuroethics*, *14*(3), 409–421. https://doi.org/10.1007/s12152-021-09477-5

Grady, S., Aeschbach, D., Wright, K. P., & Czeisler, C. A. (2010). Effect of modafinil on impairments in neurobehavioral performance and learning associated with extended wakefulness and circadian misalignment. *Neuropsychopharmacology: Official Publication of the American College of Neuropsychopharmacology*, *35*(9), 1910–1920. https://doi.org/10.1038/npp.2010.63

Harris, J. (2011). Moral Enhancement and Freedom. *Bioethics*, *25*(2), 102–111. https://doi.org/10.1111/j.1467-8519.2010.01854.x

Izquierdo, I., Bevilaqua, L. R., Rossato, J. I., Lima, R. H., Medina, J. H., & Cammarota, M. (2008). Age-dependent and age-independent human memory persistence is enhanced by delayed posttraining methylphenidate administration. *Proceedings of the National Academy of Sciences*, *105*(49), 19504–19507. https://doi.org/10.1073/pnas.0810650105

Lozano, A. M., Lipsman, N., Bergman, H., Brown, P., Chabardes, S., Chang, J. W., Matthews, K., McIntyre, C. C., Schlaepfer, T. E., Schulder, M., Temel, Y., Volkmann, J., & Krauss, J. K. (2019). Deep brain stimulation: Current challenges and future directions. *Nature Reviews Neurology*, *15*(3), 148–160. https://doi.org/10.1038/s41582-018-0128-2

Mehta, M. A., Owen, A. M., Sahakian, B. J., Mavaddat, N., Pickard, J. D., & Robbins, T. W. (2000). Methylphenidate enhances working memory by modulating discrete frontal and parietal lobe regions in the human brain. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, *20*(6), RC65.

Persson, I., & Savulescu, J. (2008). The Perils of Cognitive Enhancement and the Urgent Imperative to Enhance the Moral Character of Humanity. *Journal of Applied Philosophy*, *25*(3), 162–177. https://doi.org/10.1111/j.1468-5930.2008.00410.x

Pritchard, D. (2010). Cognitive ability and the extended cognition thesis. *Synthese*, *175*(S1), 133–151. https://doi.org/10.1007/s11229-010-9738-y

Sandel, M. J. (2004, April 1). *The Case Against Perfection*. The Atlantic. https://www.theatlantic.com/magazine/archive/2004/04/the-case-against-perfection/302927/

Sugden, C., Housden, C. R., Aggarwal, R., Sahakian, B. J., & Darzi, A. (2012). Effect of pharmacological enhancement on the cognitive and clinical psychomotor performance of sleep-deprived doctors: A randomized controlled trial. *Annals of Surgery*, *255*(2), 222–227. https://doi.org/10.1097/SLA.0b013e3182306c99

Synofzik, M. (2009). Ethically Justified, Clinically Applicable Criteria for Physician Decision-Making in Psychopharmacological Enhancement. *Neuroethics*, *2*(2), 89–102. https://doi.org/10.1007/s12152-008-9029-1

Wang, J. (2021). Cognitive Enhancement and the Value of Cognitive Achievement. *Journal of Applied Philosophy*, *38*(1), 121–135. https://doi.org/10.1111/japp.12460

Yesavage, J. A., Mumenthaler, M. S., Taylor, J. L., Friedman, L., O'Hara, R., Sheikh, J., Tinklenberg, J., & Whitehouse, P. J. (2002). Donepezil and flight simulator performance: Effects on retention of complex skills. *Neurology*, *59*(1), 123–125. https://doi.org/10.1212/wnl.59.1.123