# 1AR

## Case

### Bioterror---AT: !/D---2AC

#### Burnout, coevolution, isolation, countermeasures, intent and capability do NOT check – consilience of best studies, experts and empirics – AND Russian sponsorship overcomes all barriers

Leeson 2K [Kate Leeson, Editorial Assistant of the Hawke Institute at the University of South Australia, “Biological Weapons: Bioterrorism and the Public Health,” 2000, https://www.mapw.org.au/files/downloads/Leeson%20-Bioterrorism%20and%20the%20public%20health\_0.pdf]

It is important to acknowledge, though, that there is some danger of a successful bioterrorist attack, especially if the terrorist group in question receives assistance and substantial resources from a state which is itself developing biological weapons. There is also a danger that these weapons could become more devastating in the future because of advances in biotechnology, or if the knowledge acquired in large-scale state-run biological weapons programs (such as the former program of the Soviet Union) is purchased by other states.

#### Bioterror defense is scientifically false---especially if Russia sponsors.

Millett & Snyder-Beattie 17 [Piers Millett, PhD, Senior Research Fellow, Future of Humanity Institute, University of Oxford; and Andrew Snyder-Beattie, MS, Director of Research, Future of Humanity Institute, University of Oxford; “Existential Risk and Cost-Effective Biosecurity,” Health Security, 15(4), 8-1-2017, PubMed]

In the decades to come, advanced bioweapons could threaten human existence. Although the probability of human extinction from bioweapons may be low, the expected value of reducing the risk could still be large, since such risks jeopardize the existence of all future generations. We provide an overview of biotechnological extinction risk, make some rough initial estimates for how severe the risks might be, and compare the cost-effectiveness of reducing these extinction-level risks with existing biosecurity work. We find that reducing human extinction risk can be more cost-effective than reducing smaller-scale risks, even when using conservative estimates. This suggests that the risks are not low enough to ignore and that more ought to be done to prevent the worst-case scenarios. How worthwhile is it spending resources to study and mitigate the chance of human extinction from biological risks? The risks of such a catastrophe are presumably low, so a skeptic might argue that addressing such risks would be a waste of scarce resources. In this article, we investigate this position using a cost-effectiveness approach and ultimately conclude that the expected value of reducing these risks is large, especially since such risks jeopardize the existence of all future human lives. Historically, disease events have been responsible for the greatest death tolls on humanity. The 1918 flu was responsible for more than 50 million deaths,1 while smallpox killed perhaps 10 times that many in the 20th century alone.2 The Black Death was responsible for killing over 25% of the European population,3 while other pandemics, such as the plague of Justinian, are thought to have killed 25 million in the 6th century—constituting over 10% of the world's population at the time.4 It is an open question whether a future pandemic could result in outright human extinction or the irreversible collapse of civilization. A skeptic would have many good reasons to think that existential risk from disease is unlikely. Such a disease would need to spread worldwide to remote populations, overcome rare genetic resistances, and evade detection, cures, and countermeasures. Even evolution itself may work in humanity's favor: Virulence and transmission is often a trade-off, and so evolutionary pressures could push against maximally lethal wild-type pathogens.5,6 While these arguments point to a very small risk of human extinction, they do not rule the possibility out entirely. Although rare, there are recorded instances of species going extinct due to disease—primarily in amphibians, but also in 1 mammalian species of rat on Christmas Island.7,8 There are also historical examples of large human populations being almost entirely wiped out by disease, especially when multiple diseases were simultaneously introduced into a population without immunity. The most striking examples of total population collapse include native American tribes exposed to European diseases, such as the Massachusett (86% loss of population), Quiripi-Unquachog (95% loss of population), and the Western Abenaki (which suffered a staggering 98% loss of population).9 In the modern context, no single disease currently exists that combines the worst-case levels of transmissibility, lethality, resistance to countermeasures, and global reach. But many diseases are proof of principle that each worst-case attribute can be realized independently. For example, some diseases exhibit nearly a 100% case fatality ratio in the absence of treatment, such as rabies or septicemic plague. Other diseases have a track record of spreading to virtually every human community worldwide, such as the 1918 flu,10 and seroprevalence studies indicate that other pathogens, such as chickenpox and HSV-1, can successfully reach over 95% of a population.11,12 Under optimal virulence theory, natural evolution would be an unlikely source for pathogens with the highest possible levels of transmissibility, virulence, and global reach. But advances in biotechnology might allow the creation of diseases that combine such traits. Recent controversy has already emerged over a number of scientific experiments that resulted in viruses with enhanced transmissibility, lethality, and/or the ability to overcome therapeutics.13-17 Other experiments demonstrated that mousepox could be modified to have a 100% case fatality rate and render a vaccine ineffective.18 In addition to transmissibility and lethality, studies have shown that other disease traits, such as incubation time, environmental survival, and available vectors, could be modified as well.19-21 Although these experiments had scientific merit and were not conducted with malicious intent, their implications are still worrying. This is especially true given that there is also a long historical track record of state-run bioweapon research applying cutting-edge science and technology to design agents not previously seen in nature. The Soviet bioweapons program developed agents with traits such as enhanced virulence, resistance to therapies, greater environmental resilience, increased difficulty to diagnose or treat, and which caused unexpected disease presentations and outcomes.22 Delivery capabilities have also been subject to the cutting edge of technical development, with Canadian, US, and UK bioweapon efforts playing a critical role in developing the discipline of aerobiology.23,24 While there is no evidence of state-run bioweapons programs directly attempting to develop or deploy bioweapons that would pose an existential risk, the logic of deterrence and mutually assured destruction could create such incentives in more unstable political environments or following a breakdown of the Biological Weapons Convention.25 The possibility of a war between great powers could also increase the pressure to use such weapons—during the World Wars, bioweapons were used across multiple continents, with Germany targeting animals in WWI,26 and Japan using plague to cause an epidemic in China during WWII.27 Non-state actors may also pose a risk, especially those with explicitly omnicidal aims. While rare, there are examples. The Aum Shinrikyo cult in Japan sought biological weapons for the express purpose of causing extinction.28 Environmental groups, such as the Gaia Liberation Front, have argued that “we can ensure Gaia's survival only through the extinction of the Humans as a species … we now have the specific technology for doing the job … several different [genetically engineered] viruses could be released”(quoted in ref. 29). Groups such as R.I.S.E. also sought to protect nature by destroying most of humanity with bioweapons.30 Fortunately, to date, non-state actors have lacked the capabilities needed to pose a catastrophic bioweapons threat, but this could change in future decades as biotechnology becomes more accessible and the pool of experienced users grows.31,32 What is the appropriate response to these speculative extinction threats? A balanced biosecurity portfolio might include investments that reduce a mix of proven and speculative risks, but striking this balance is still difficult given the massive uncertainties around the low-probability, high-consequence risks. In this article, we examine the traditional spectrum of biosecurity risks (ie, biocrimes, bioterrorism, and biowarfare) to categorize biothreats by likelihood and impact, expanding the historical analysis to consider even lower-probability, higher-consequence events (catastrophic risks and existential risks). In order to produce reasoned estimates of the likelihood of different categories of biothreats, we bring together relevant data and theory and produce some first-guess estimates of the likelihood of different categories of biothreat, and we use these initial estimates to compare the cost-effectiveness of reducing existential risks with more traditional biosecurity measures. We emphasize that these models are highly uncertain, and their utility lies more in enabling order-of-magnitude comparisons rather than as a precise measure of the true risk. However, even with the most conservative models, we find that reduction of low-probability, high-consequence risks can be more cost-effective, as measured by quality-adjusted life year per dollar, especially when we account for the lives of future generations. This suggests that despite the low probability of such events, society still ought to invest more in preventing the most extreme possible biosecurity catastrophes.

## Buddhism K

### AT: Definitions

#### Should concedes our framework interp

Lambertson ’9 [Floyd Wesley; August 18; Professor of Speech at the University of Northern Iowa; Quarterly Journal of Speech, “The meaning of the word ‘should’ in a question of policy,” vol. 28]

"In a question of policy—or any other question—the word 'should' means 'that the policy advocated is necessary and desirable; that 'this house' commit itself to that policy, principle, or theory. But also, as a matter of common sense, it implies to some extent the 'could' and the 'would.' What is the sense of discussing at length the 'should' of a principle if it is not feasible, practicable, or attainable? In other words it is necessary for the affirmative to justify the policy or theory (the 'should') and also to some extent to set out— at least suggest—the technique of establishing it." Lew Sarett, Northwestern University.

### AT: Long---1AR

#### Their ballot key warrant relies on neuroplasticity and describes changes to the brain as a long-term process---proves other rounds and life outside of debate thump. MSU is blue.

Long ’21 [William; 2021; Professor of Political Science at Georgia State University; A Buddhist Approach to International Relations, “A Buddhist Alternative,” Ch. 6]

In addition to revolutionary changes in the physical sciences, the life sciences too have made remarkable new discoveries that challenge our thinking about human nature as irreversibly self-interested and expand the possibilities for considering our cooperative potential and corresponding social arrangements. Until relatively recently, the prevailing view in neuroscience was that the brain contained all its neurons at birth, and the number and circuitry of these neurons were set within the first few years of life. Scientists believed that the only lifelong brain changes were minor alterations in synaptic (interneuronal) connections and accelerating cell death with aging. Social scientists in the Western tradition assumed that this relatively fixed brain was, by nature, first and always primarily self-interested and self-serving.

In the 1990s, however, neuroscientists discovered that the brain continues to generate new neurons throughout life (neurogenesis) and that new and existing neurons undergo structural and functional changes in their circuitry in response to their environments, by training and experience (neuroplasticity). Contrary to what was once believed, the brain is highly dynamic (Eriksson et al. 1998). When referring to changes in the brain, it is important to distinguish between gross morphology and cellular structure and function. The overall structure and pattern of brain development is under genetic control and does not change markedly. But our 35,000 genes are not up to the job of prescribing the wiring for the brain’s 100 trillion or more synapses. These connections are shaped by our ongoing experiences. It is at this cellular level that the brain is remarkably plastic. Neuroplasticity refers to altering connections in the brain, the strengthening, withering, or rerouting of synaptic connections.

Neuroplasticity is more than mere learning or storing a memory. The brain is far more flexible than that. It can make wholesale topographical reorganizations throughout life (Elbert et al. 1995). For example, experiments demonstrate that some brain areas that were thought to be “hardwired” for one function can in response to injury and adaptive effort, take on a totally different function, what scientists call cross-modal functional plasticity. Altering connections in the brain in a way that strengthens the efficacy of a neuronal circuit over the long term is the essence of neuroplasticity.

How does the brain accomplish these adaptive feats? Various new technologies are giving us a glimpse of this process. These new technologies are illuminating the neural correlates for specific adaptations wrought through repeated experiences. These technologies can also show us the brain areas and patterns of electrochemical activation associated with a mental process. In discovering and observing the link between brain circuitry and mental states, some scientists are also suggesting that the causal connection between brain and mind works in both directions (Lutz et al. 2004). Specifically, they offer intriguing new evidence to suggest that the processes of brain wiring and rewiring may be shaped by mental (nonphysical) events. This work reveals that it is not just experience that molds the brain. Rather, changes in brain circuitry are generated only when behavior is specifically attended to. Attention (mindfulness), is required for use-dependent brain changes. In fact, imagined physical movements, if repeated with concentration, can produce the same synaptic changes as actual repetitive body movements (Schwartz and Begley 2002; Slotnick 2004). Similarly, mental imagery correlates with the activation of the same brain areas as those associated with the actual perception of the imagined object. In short, mental force appears to express itself through the brain, but it is not reducible to the brain.

Some neuroscientists began looking at the brain activity (“brain state”) and cognitive and neural characteristics (“brain traits”) of meditators to better understand the immediate and long-term effects of focused awareness. These studies produced preliminary evidence for the possibility that mental training may alter brain activity, shape the physical brain, and affect human behavior. Early work by Richard Davidson, Antoine Lutz, and others found that sustained thoughts activated certain neuronal pathways in the brain associated with the regulation of positive affect (like compassion), reduced negative thoughts and feelings such as anxiety and depression, and subdued self-referential thoughts (See Davidson et al. 2003; Pollard 2003; Lutz et al. 2004). These early studies lent support to the notion that a willful refocusing of mental awareness could bring about important changes in brain activity and structure (Brefczynski-Lewis et al. 2007; Lazar et al. 2005).

These initial investigations have led to hundreds of recent studies on the impact of various forms of mindfulness and meditation on brain functioning and morphology. Two “metastudies” (studies of studies) reviewed these experiments looking for methodological reliable and comparable results. One of these metastudies concluded “that meditation appears to be reliably associated with altered anatomical structure in several brain regions” (Fox et al. 2014 at p. 69). The brains of meditators were altered in eight brain regions including areas related to meta awareness (our ability to watch our own minds), body awareness, memory consolidation and reconsolidation, self and emotional regulation, and infra and interhemispheric communication (Fox et al. 2014; see also Afonso et al. 2020). The second metastudy concluded that meditation produces positive effects on cognitive and emotional processes (Sedlmeier et al. 2012). Several individual studies raise intriguing possibilities. For example, one study found that meditators, unlike control subjects, had reduced activity in “self-referential processing,” i.e., mind wandering, which appears to be our default mechanism and is often correlated with unhappiness (Brewer et al. 2011). Another study found that meditation increased compassionate responses to suffering, even in the face of social pressures to avoid so doing (Condon et al. 2013).

As noted, these changes in brain function and form do not occur without sustained and repeated effort, however. Absent focused attention, the brain will produce predictable patterns of brain activity, that is, our default mode of thinking. Through choice and willful attention, however, it appears that an alternative synaptic path may be activated and perpetuated. The idea that immaterial forces such as intention and attention could shape the brain’s function and form runs counter to classical materialist science. Working in the materialist tradition, most scientists, including almost all neuroscientists, have assumed that mental processes are inefficacious byproducts of purely physical brain processes. To the extent that one can recognize the mind at all, brain to mind is a one-way street. All our thoughts and actions are reducible to impersonal, microscopic, physical processes. Nothing that is nonphysical, such as the mind, consciousness, or will, can even exist in the sense of being a measurable, real entity much less shape physical outcomes.

This classical approach has been unable to explain how brain activity gives rise to consciousness (subjectively felt mental states), however, and what role consciousness might play in the brain’s workings. Why, if exclusively local physical processes in the brain control us, do we possess a stream of conscious thoughts capable of understanding large-scale phenomena? After 350 years of classical material science and more than half a century of neuroscience, materialist approaches have done a good job of linking structure and function in the brain, but have made no progress in explaining consciousness, something we all experience most all the time. In the materialist paradigm, accounting for consciousness is the “hard problem,” and because consciousness cannot be effectively explained by reference to material forces, for most scientists in the classical material tradition, consciousness either is not a legitimate area of inquiry or, if it is, they have promised, since the eighteenth century, that a materialist answer to the hard problem of consciousness is only a matter of time (Araujo 2012).

The idea that the process of brain wiring and rewiring is shaped by immaterial mental events may confound classical materialist science (which either denies mind or separates mind from matter), but it is not inconsistent with quantum science (which sees mind and matter as inextricably entwined). Recall that in the quantum world, the subject determines which of many possible realities becomes actualized through its intention and attention. Quantum theory reunites consciousness with the causal structure of nature, joining subjective experience and objective outcomes. Thus, quantum theory creates a “causal opening for the mind,” a point of entry by which mind could alter the functioning and shape the physical structure of the brain.

Is there evidence for the existence of a “quantum brain” or “quantum consciousness?” At this point we do not know, and it remains to be seen where, if anywhere, there exists a demonstrable locus for quantum effects in the brain. Because the environment for sustained quantum effects to operate in the brain has not been sufficiently established, traditional neuroscience argues that brain functions can, indeed must, be understood as the interactions of neurons operating under classical physical principles. Still, we know that quantum physics operates sub-atomically everywhere, and we know that mechanical explanations of neuronal function cannot account for the processing speed of the human brain. Furthermore, there is evidence that sustained thought alters brain states and traits; we just do not know how or precisely where this occurs. Quantum theory raises the following question to material neuroscience: How can the mind and consciousness be reduced to the function of atoms within the brain if we know that ultimately these atoms have no fixed or non-probabilistic existence outside of subjective mental events? If atoms derive their properties from interaction with consciousness [in quantum], how can consciousness depend only on those same atoms? (Schwartz and Begley 2002).

In truth, at this moment, both materialism and quantum approaches toward mind are meta-physical assertions awaiting more evidence, an epistemic exercise. Science should be about epistemic pursuits, not metaphysical closure, so let us keep an open mind. Asserting that a nonmaterial force (thought) can shape a material object such as the brain, as quantum theory does, is no more speculative than asserting a material basis for nonmaterial consciousness, which is the prevailing materialist neuroscience view. With the advent of quantum theory, the nature of matter has become as problematic as the nature of mind.

Implications of New Scientific Discoveries for Social Theory

I only report on this ongoing scientific debate to consider its possible implications for the discussion at hand. As noted, some social scientists wonder “Are Buddhist ideas harmonious with science?” The answer, I suggest, is “yes,” they are remarkably consistent with the latest findings in the physical and biological sciences, not “otherworldly.”

Coming back to the focus of this discussion (and firmer footing for the author), the quantum explanations for brain plasticity and a causal role for mind carry potentially important behavioral and moral consequences for social thought and action coming from the world of science (Wendt 2015). If true, they would imply that, although we are endowed with a given brain morphology and basic circuitry, not all aspects of our responses are passively determined by neurobiological mechanisms. Instead, our volitional choices moment to moment to attend to one bit of environmental stimulation over another and to form, through our intention and attention (the driving force of karma, for Buddhists), one thought pattern rather than another, can sculpt our brain and make us who we are.

#### Clash over a subject does NOT rewire the brain, alt causes outweigh, and unfairness turns it

Gazerani 25 [Parisa Gazerani, Doctorate degree in pharmacy, Ph.D. in biomedical sciences, and postdoctoral experience in neuropharmacology, Associate professor affiliated with the Pharmacy program at Oslo Metropolitan University, “The neuroplastic brain: current breakthroughs and emerging frontiers,” Brain Research, Vol. 1858, July 2025, https://www.sciencedirect.com/science/article/pii/S0006899325002021#ab010]

1. Introduction

Neuroplasticity is the brain’s remarkable capacity to reorganize itself by forming, modifying, and strengthening neural connections in response to both internal experiences and external stimuli (Diniz and Crestani, 2023). Historically viewed as largely confined to early development, it is now recognized as a lifelong process supporting a range of essential functions, including learning, memory, and adaptation (Parisi et al., 2019). Beyond its individual impact, enabling recovery from injury and resilience against cognitive decline, neuroplasticity carries significant societal implications by informing strategies to combat neurological disorders and optimize mental health (Kumar et al., 2023, Zotey et al., 2023).

At the same time, neuroplasticity can act as a double-edged sword (Allred and Jones, 2008). While adaptive changes foster skill acquisition, rehabilitation, and healthy aging, maladaptive processes can entrench pathological states such as chronic pain, addiction, and neurological and psychiatric conditions (Marzola et al., 2023). Understanding the mechanisms governing this dual nature is pivotal for designing targeted interventions that harness beneficial plasticity while mitigating harmful rewiring (Hu et al., 2023). This overview provides an in-depth exploration of neuroplasticity’s mechanisms, examines emerging therapeutic approaches including pharmacological, lifestyle, and technological strategies, and addresses the broader implications of utilizing neuroplasticity to maintain brain health and prevent disease. By highlighting both the opportunities and challenges inherent in manipulating neuroplastic processes, the aim is to offer a comprehensive framework for advancing research and clinical applications in this rapidly evolving field.

To strengthen the foundation of this review, it is essential to integrate both seminal and recent findings that have shaped our current understanding of neuroplasticity. Landmark studies on long-term potentiation (LTP) and adult neurogenesis laid the groundwork for contemporary models of brain adaptability, while large-scale clinical trials exploring neuromodulation and gene-editing approaches have expanded these concepts toward translational applications, where interested readers are encouraged to explore in depth (McEachern and Shaw, 1999, Will et al., 2008, Bruel-Jungerman et al., 2006, Stuchlik, 2014, von Bernhardi et al., 2017, Mateos-Aparicio and Rodríguez-Moreno, 2019, Johnson and Cohen, 2023).

2. Mechanisms of neuroplasticity

Neuroplasticity (Innocenti, 2022) is underpinned by four interconnected processes (Voss et al., 2017): synaptic plasticity, structural plasticity, neurogenesis, and functional reorganization that enable the brain to adapt to both internal and external changes throughout the lifespan (Sale et al., 2014, Power and Schlaggar, 2017, Pickersgill et al., 2022). These mechanisms are essential for learning, memory, and the restoration of function following injury, yet they can also contribute to the development of pathological states when dysregulated (Mateos-Aparicio and Rodríguez-Moreno, 2019, Appelbaum et al., 2023). Although each mechanism has traditionally been examined in isolation, current research increasingly highlights their interdependence (Wenger et al., 2021).

Many foundational insights into these mechanisms derive from animal studies (Wolpaw, 2012) and small-scale human trials (Mishra and Gazzaley, 2016), highlighting the need for larger, more diverse clinical cohorts. Recognizing such methodological constraints sharpens our critical perspective and shows the necessity of robust translational frameworks or alternatives (Rudroff, 2024) for applying these findings in clinical practice.

2.1. Synaptic plasticity

Synaptic plasticity refers to the modulation of synaptic strength, primarily through LTP and long-term depression (LTD) (Bliss and Cooke, 2011). LTP, widely regarded as a key molecular basis for learning and memory (Hayashi, 2022), enhances synaptic efficacy by increasing receptor density and neurotransmitter release at activated synapses. Frequent practice or repetition of a skill consolidates these strengthened connections, reinforcing the neural circuits involved. For example, an adaptive form of plasticity emerges when synaptic connections are strengthened during skill acquisition, such as practicing a musical instrument, or during the formation of long-term memories, such as learning a new language (Wenger et al., 2021, Wenger et al., 2017, Dayan and Cohen, 2011). In contrast, a maladaptive form arises when excessive LTP in pain pathways leads to central sensitization, contributing to chronic pain conditions like fibromyalgia (Nijs et al., 2023, Ji et al., 2018) or neuropathic pain (Costigan et al., 2009).

LTD, on the other hand, reduces synaptic efficacy, refining synaptic networks and helping maintain an optimal excitatory–inhibitory balance (Castillo et al., 2011). A beneficial instance of LTD occurs when the brain prunes unnecessary synapses to refine motor skills and optimize information processing (Piochon et al., 2016). However, excessive LTD in hippocampal circuits (Tim et al., 2006) may contribute to cognitive decline and memory impairments, as seen in disorders such as Alzheimer’s disease (Henstridge et al., 2019).

Nonetheless, contradictory findings persist regarding whether increased synaptic strength always translates directly to improved cognitive or motor function. For instance, while some studies link LTP enhancement to better memory performance, others suggest that overactivation of excitatory pathways can tip the balance toward excitotoxicity (Huang et al., 2005, Gonçalves-Ribeiro et al., 2019). Suboptimal LTD (Monsorno et al., 2023) could also impair necessary synaptic refinement (Cooke and Bliss, 2006), but conclusive evidence in human populations (Howes and Onwordi, 2023) remains limited by small sample sizes and variability in measurement techniques.

Beyond LTP and LTD, homeostatic plasticity (Pozo and Goda, 2010, Chen et al., 2022) sustains an overall balance of excitation and inhibition in neuronal networks. Disruption of these homeostatic mechanisms is implicated in conditions (Lepeta et al., 2016) including epilepsy, schizophrenia, and Alzheimer’s disease (Meftah and Gan, 2023), showing the therapeutic potential of interventions that modulate synaptic remodeling, such as pharmacological agents targeting NMDA receptors (Nicosia et al., 2024) or synaptic scaling factors (Wu et al., 2023). A failure in homeostatic plasticity can manifest in conditions like chronic pain (Thapa et al., 2021), where local circuits become persistently over- or underactivated. While animal models have illuminated these pathways (Lamichhane et al., 2024, Sandkühler, 2009), reliable clinical biomarkers and standardized intervention protocols remain underdeveloped.

2.2. Structural plasticity

Structural plasticity involves physical changes in neural architecture, including synaptogenesis (the formation of new synapses), dendritic branching, and synaptic pruning (Leuner and Gould, 2010, Kolb and Gibb, 2011). Synaptogenesis and dendritic remodeling can lead to adaptive outcomes (Schlaug, 2001), as demonstrated by increases in dendritic complexity in the motor cortex when individuals learn to play a musical instrument (Schlaug, 2001), ultimately resulting in refined and more efficient motor control. However, these same mechanisms can turn maladaptive if abnormal dendritic growth or spine formation occurs in response to certain drugs of abuse, such as cocaine, reinforcing addictive behaviors (Nestler and Lüscher, 2019, Russo and Nestler, 2013) by over-strengthening reward-related circuits (Forbes and Goodman, 2014). Similarly, synaptic pruning serves an essential adaptive function by eliminating underused or weak synapses during adolescence and adulthood, thereby optimizing neural pathways (Sakai, 2020). When pruning is excessive, however, it may contribute to disorders such as schizophrenia, in which key synaptic connections are disrupted (Howes and Onwordi, 2023, Chafee and Averbeck, 2022, Germann et al., 2021).

In the context of injury recovery, structural plasticity is vital for re-establishing function. Following a stroke, for instance, peri-infarct areas often undergo dendritic sprouting and synapse formation, facilitating the restoration of motor or language capabilities (Campos et al., 2023). Recent advances in two-photon microscopy (Li et al., 2023) and other high-resolution imaging tools make it possible to observe these structural changes in real-time, both in animal models and clinical settings, providing deeper insights into the mechanisms and potential therapeutic applications of structural plasticity (Murphy, 2015, Xiong et al., 2023). In practice, translating knowledge of structural plasticity into therapy presents hurdles. Techniques like two-photon microscopy have validated structural changes in animal models, but real-time imaging in humans is complex, expensive, and rarely standardized across research centers (Ayaz et al., 2022).

2.3. Neuronal plasticity and network adaptations

Neuronal plasticity refers to the ability of individual neurons to modify their intrinsic properties in response to learning, experience, and environmental stimuli (von Bernhardi et al., 2017). This adaptation occurs through two primary mechanisms: (1) intrinsic plasticity, which involves changes in the electrophysiological properties of individual neurons, and (2) network-level plasticity, which reflects large-scale reorganization of neural circuits and connectivity patterns (von Bernhardi et al., 2017, Oberman and Pascual-Leone, 2013, Stampanoni Bassi et al., 2019). While synaptic plasticity and structural remodeling dictate changes at the level of synapses and dendrites, neuronal plasticity ensures that neurons themselves adjust their excitability, responsiveness, and integration into functional networks (Wenger et al., 2021, Gipson and Olive, 2017, Chen and Nedivi, 2010). These adaptations are fundamental to learning, memory consolidation, and functional recovery after neurological injury. However, when dysregulated, they can contribute to maladaptive conditions such as epilepsy, addiction, and psychiatric disorders (Marzola et al., 2023, Sale et al., 2014, Cramer et al., 2011).

Intrinsic Neuronal Plasticity: Ion Channel Regulation and Excitability

At the single-cell level, neurons undergo intrinsic plasticity by adjusting their excitability and firing properties through modifications in ion channel expression, neurotransmitter receptor density, and metabolic activity (Debanne et al., 2019). Changes in ion channel dynamics, such as the upregulation of voltage-gated sodium and calcium channels, can increase neuronal excitability, enhancing responsiveness to incoming stimuli (Daoudal and Debanne, 2003). Conversely, the downregulation of excitatory ion channels or increased potassium channel activity can reduce neuronal firing rates, promoting stability in neural networks and preventing excessive activation (Desai, 2003).

This intrinsic plasticity is particularly crucial for homeostatic regulation in response to prolonged synaptic activity. For instance, during skill acquisition, neurons in the motor cortex display a reduced threshold for firing and increased action potential precision, optimizing motor control (Paz et al., 2009). Similarly, in hippocampal neurons, the expression of NMDA and AMPA receptors is dynamically regulated to fine-tune synaptic strength during memory formation (Titley et al., 2017). However, dysregulation of these mechanisms can lead to pathological states: in epilepsy, excessive excitability and impaired inhibition drive hyperactive neuronal circuits, leading to recurrent seizures (Scharfman, 2002, Knowles et al., 2022).

Network-Level Plasticity: Functional Connectivity and Large-Scale Adaptations

Beyond changes in individual neurons, network-level plasticity encompasses shifts in functional connectivity between brain regions, allowing for adaptation to new experiences, learning, and compensatory mechanisms after injury (Pascual-Leone et al., 2011). This process involves synaptic remodeling, axonal sprouting, and dynamic reconfiguration of neuronal ensembles, which refine cognitive and motor functions (Kelly and Castellanos, 2014).

One of the most well-documented examples of network plasticity occurs during memory consolidation, where neuronal circuits in the hippocampus and prefrontal cortex undergo coordinated oscillatory changes to stabilize newly learned information (Squire et al., 2015, Geva-Sagiv et al., 2023). These network shifts facilitate the transition from hippocampal-dependent learning to long-term storage in cortical regions, ensuring more efficient retrieval (Buzsáki, 2015, Preston and Eichenbaum, 2013).

In response to neurological injury, the brain can undergo compensatory network reorganization (Chen et al., 2002, Hylin et al., 2017). After a stroke affecting motor areas, adjacent cortical regions, and contralateral motor networks are recruited to assume lost function, enabling partial motor recovery (Ward et al., 2003, Jones, 2017). This reorganization is facilitated by increased interhemispheric connectivity and strengthening of pre-existing but underutilized pathways (Grefkes and Fink, 2011). However, when network-level plasticity is maladaptive, it can reinforce pathological states (Costigan et al., 2009). In chronic pain conditions, for instance, excessive connectivity between pain-processing regions (e.g., thalamus, somatosensory cortex, anterior cingulate cortex) leads to persistent pain perception, even in the absence of ongoing nociceptive stimuli (Kuner and Flor, 2016).

Therapeutic Interventions Targeting Neuronal Plasticity

Given the profound role of neuronal plasticity in both adaptive and maladaptive processes, various therapeutic strategies (Kumar et al., 2023, Colavitta and Barrantes, 2023) aim to enhance beneficial neuronal adaptation while mitigating pathological remodeling.

1.

Pharmacological Modulation

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BDNF-enhancing drugs: Brain-derived neurotrophic factor (BDNF) plays a key role in neuronal plasticity. Drugs that increase BDNF expression, such as SSRIs (fluoxetine) or ketamine, have been shown to restore synaptic and neuronal plasticity in depression (Casarotto et al., 2022, Casarotto et al., 2021).

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Ion channel modulators: Anti-epileptic drugs such as gabapentin and lamotrigine regulate neuronal excitability by stabilizing ion channel function, reducing hyperexcitability in conditions like epilepsy (Czapińska-Ciepiela et al., 2024) and neuropathic pain (Eijkelkamp et al., 2012).

2.

Non-Invasive Neuromodulation

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Transcranial Magnetic Stimulation (TMS): Repetitive TMS can enhance neuronal excitability in underactive brain regions (e.g., dorsolateral prefrontal cortex in depression) or inhibit hyperactive circuits (e.g., motor cortex in dystonia) (Kricheldorff et al., 2022, Jannati et al., 2023, Suppa et al., 2022, Lefaucheur et al., 2020).

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Transcranial Direct Current Stimulation (tDCS): tDCS can induce long-term changes in neuronal excitability (Lang et al., 2004), facilitating motor and cognitive recovery in stroke and neuropsychiatric disorders (Nitsche and Paulus, 2011).

3.

Cognitive and Behavioral Interventions

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Intensive skill training and cognitive therapy can promote functional network reorganization in neurorehabilitation. For example, constraint-induced movement therapy in stroke patients enhances motor network connectivity (Taub and Morris, 2001, Wang et al., 2022).

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Mindfulness and cognitive-behavioral therapy (CBT) have been shown to reshape maladaptive prefrontal-limbic circuits in anxiety and PTSD, reducing hyperactivation of fear-related pathways (Yuan et al., 2022, Hölzel et al., 2011).

Neuronal plasticity, therefore, represents a fundamental mechanism by which the brain adapts at both the cellular and network levels. While intrinsic neuronal plasticity fine-tunes individual excitability and firing properties, network-level plasticity dictates how functional circuits reorganize for learning and recovery. However, when these mechanisms become dysregulated, they contribute to disorders ranging from chronic pain and epilepsy to neuropsychiatric diseases. Understanding these processes provides crucial insights into therapeutic strategies, including pharmacological agents, neuromodulation, and behavioral interventions, to harness beneficial plasticity and mitigate maladaptive changes.

2.4. Neurogenesis

Neurogenesis refers to the generation of new neurons, predominantly in the hippocampus (dentate gyrus) and, to a lesser extent, in the subventricular zone (Ming and Song, 2011, Vaz et al., 2022). Although the rate of neurogenesis declines with age, it remains integral to learning, memory, emotional regulation, and cognitive flexibility (Toda et al., 2019, Amelchenko et al., 2023). When neurogenesis is robust, such as in individuals exposed to enriched environments that include complex spatial tasks, exercise, and social interaction, it can enhance cognitive performance and help regulate stress responses (Schloesser et al., 2010). However, chronic stress and elevated cortisol levels can diminish hippocampal neurogenesis, potentially exacerbating conditions like depression and anxiety (Schoenfeld and Gould, 2012). Current research exploring pharmacological approaches, including agents that upregulate brain-derived neurotrophic factor (BDNF) (Ibrahim et al., 2022), and genetic strategies aimed at stimulating hippocampal growth shows promise for mitigating age-related cognitive decline and improving outcomes in neurodegenerative diseases (Numakawa and Kajihara, 2024) such as Parkinson’s and Huntington’s (Vassal et al., 2025).

Elevated neurogenesis, supported by exercise (van Praag, 2008) and enriched environments (Brown et al., 2003), correlates with improved mood and memory. However, some studies fail to show robust gains in humans, and the degree to which newly generated neurons integrate into existing circuits remains debated (Gonçalves et al., 2016, Kempermann et al., 2018, Gage and Temple, 2013). Inter-individual variability (e.g., genetic predispositions, sex hormones) may also complicate the reproducibility of findings.

2.5. Functional reorganization

Functional reorganization allows the brain to compensate for neural damage by reallocating tasks to alternative or underutilized regions (Castellanos et al., 2011). For example, following a stroke that affects the left motor cortex, the contralateral (right) motor cortex may assume partial control of motor functions in the affected limb, thereby facilitating recovery (Ward, 2004). This adaptive shift can be further enhanced through targeted therapies such as constraint-induced movement therapy (Sawaki et al., 2008). However, in cases of chronic pain or phantom limb pain, reorganization in somatosensory or motor cortices may perpetuate pain signals or aberrant sensations in the absence of actual peripheral input (Karl et al., 2001, Culp and Abdi, 2022). Modern neuroimaging techniques including functional MRI (fMRI) and positron emission tomography (PET) have illustrated how function can be redistributed to adjacent or contralateral regions (Seifert and Maihöfner, 2011, Gunduz et al., 2020). In addition, non-invasive neuromodulation methods (e.g., transcranial magnetic stimulation) and BCIs seek to optimize this reorganization, thereby improving rehabilitation outcomes in stroke, spinal cord injury, and other conditions, though clinical outcomes are often heterogeneous, reflecting diverse patient profiles and inconsistent stimulation protocols (Li et al., 2023, Calderone et al., 2024, Eliason et al., 2024).

In summary, these four mechanisms highlight the dynamic capacity of the brain to reshape itself in response to changing conditions (Table 1). While adaptive (good) plasticity facilitates learning, recovery, and healthy aging, maladaptive (bad) plasticity can entrench chronic pain, addiction, and other neurological or psychiatric conditions. Ongoing research seeks to harness beneficial plasticity while mitigating maladaptive processes, reflecting a promising avenue for improving clinical outcomes across a range of disorders.

Table 1. Snapshot of Mechanisms, Examples, and Interventions.

CNS Component Adaptive (Good) Plasticity Maladaptive (Bad) Plasticity Current State-of-the-Art Interventions

Synaptic Plasticity (LTP, LTD, synaptic scaling) − LTP driving skill acquisition (e.g., musical training, language learning).

− LTD refining synaptic networks for efficiency. − Excessive LTP in pain pathways contributing to chronic pain.

− Excessive LTD in the hippocampus (e.g., Alzheimer’s disease). − Pharmacological modulators (NMDA receptor agonists/antagonists).

− Synaptic scaling therapies.

− Cognitive training programs.

Neuronal Plasticity (Neurogenesis, dendritic remodeling) − Enhanced hippocampal neuron production via exercise and enriched environments.

− Improved memory and emotional regulation. − Reduced neurogenesis due to chronic stress (e.g., elevated cortisol).

− Links to depressive and anxiety disorders. − Pharmacological agents boosting BDNF or growth factors.

− Gene therapy approaches.

− Lifestyle interventions (diet/exercise).

Supporting Tissue Plasticity (Glial, vascular, extracellular matrix changes) − Astrocyte-mediated synaptic support for learning.

− Microglial pruning optimizing synaptic efficiency.

− Vascular adaptations promoting oxygenation and metabolic support. − Reactive gliosis disrupting neural function.

− Excessive synaptic pruning linked to schizophrenia.

− Blood-brain barrier breakdown in neurodegenerative conditions. − Anti-inflammatory therapies targeting glial dysregulation.

− Vascular-targeted interventions (e.g., exercise, neurovascular drugs).

− Real-time plasticity monitoring.

Functional Reorganization (Result of the above plasticity mechanisms) − Contralateral cortical takeover in stroke rehab.

− Brain-computer interfaces (BCIs) supporting recovery. − Aberrant reorganization in chronic or phantom limb pain.

− Unhelpful rewiring sustaining compulsive/addictive behaviors. − Non-invasive neuromodulation (TMS, tDCS).

− Constraint-induced movement therapy. − BCI training for motor/cognitive improvement.

Abbreviations: LTP, long-term potentiation; LTD, long-term depression; BDNF, brain-derived neurotrophic factor; BCI, brain-computer interface; TMS, transcranial magnetic stimulation; tDCS, transcranial direct current stimulation.

While these four mechanisms of neuroplasticity—synaptic plasticity, structural plasticity, neurogenesis, and functional reorganization—enable the brain to adapt and recover, their effects are not universally beneficial. Depending on the context, plasticity can manifest as either adaptive (beneficial) or maladaptive (harmful) changes, influencing both normal brain function and disease progression. The following section explores this dual nature of neuroplasticity, highlighting its role in learning, recovery, and pathological conditions.

3. Adaptive and maladaptive plasticity

As summarized in Table 1, each of the four neuroplastic mechanisms can manifest as either adaptive or maladaptive changes, depending on factors such as the nature of the stimulus, individual genetic predispositions, and environmental conditions. While neuroplasticity is essential for brain health, its effects are not universally beneficial. The distinction between adaptive and maladaptive plasticity highlights the importance of targeted interventions to promote positive outcomes.

Adaptive plasticity supports learning, memory, and recovery from injury by allowing the brain to compensate for age-related changes, maintain cognitive function, and rebound from neurological insults. For instance, physical rehabilitation programs for stroke patients utilize adaptive plasticity by encouraging the use of impaired limbs, thus reinforcing neural circuits that promote functional recovery, effectively “re-teaching” the brain how to execute movements (Aderinto et al., 2023). Lifelong intellectual engagement and social interaction may build cognitive reserve (Scarmeas and Stern, 2003), buffering age-related declines (Piolatto et al., 2022). Results from prospective cohort studies have been encouraging, though causality is often difficult to disentangle from confounding factors (Lindenberger, 2014).

The point to consider is that neuroplasticity processes can be beneficial or harmful depending on context (Price and Duman, 2020), genetic background, and environmental exposures. For instance, while chronic stress can upregulate synaptic connections in fear-related circuits, reinforcing anxiety, appropriate exposure-based therapies can reshape these pathways adaptively (Kenwood et al., 2022, Daviu et al., 2019, Zhang et al., 2021).

By contrast, maladaptive plasticity reinforces negative patterns that exacerbate neurological or psychiatric conditions. Chronic pain (Kiritoshi et al., 2024) is a prime example, in which heightened connectivity within pain-processing circuits continues to generate discomfort long after the initial injury has healed. Neuroimaging (Martucci and Mackey, 2018, Martucci et al., 2014) has shown hyperconnectivity in pain-processing networks among chronic pain sufferers. Targeted interventions (e.g., neuromodulation, cognitive-behavioral therapy) can sometimes break this cycle (Bazzari and Bazzari, 2022), though optimal timing and individual variability remain hotly debated.

Likewise, maladaptive changes in reward pathways can underlie addiction, and anxiety disorders are often characterized by excessive connectivity in fear-processing networks (Shin and Liberzon, 2010, Lüthi and Lüscher, 2014). Addressing these pathological patterns necessitates interventions that disrupt harmful circuitry and restore equilibrium. Cognitive-behavioral therapy, neuromodulation, and pharmacological agents targeting these maladaptive pathways are all critical components in this effort. For instance, in terms of addiction (Cooper et al., 2017), repeated drug exposure can strengthen reward-related circuits, entrenching compulsive behaviors. While this is well-documented in preclinical models, clinical translation is hampered by variability in addiction subtypes and comorbid mental health conditions (Russo and Nestler, 2013, Corley et al., 2024).

Interactions Between Neuroplasticity Mechanisms Under Pathological Conditions

Neuroplasticity mechanisms do not operate in isolation − rather, they interact dynamically across different levels, particularly in pathological states where dysregulated plasticity at one level often triggers maladaptive changes at others. The complex interplay between synaptic, structural, neuronal, and network-level plasticity is crucial for understanding how neurological and psychiatric disorders emerge and persist (Oberman and Pascual-Leone, 2013, Gulyaeva, 2017).

One striking example of cross-mechanism interactions is found in chronic pain. Persistent pain is not merely a result of enhanced synaptic plasticity in pain pathways (excessive LTP in nociceptive circuits) but also involves structural remodeling, including dendritic spine reorganization in the somatosensory cortex and anterior cingulate cortex. Over time, these maladaptive changes lead to network-level alterations, increasing connectivity between pain-processing regions such as the thalamus, insula, and prefrontal cortex (Kuner and Flor, 2016). This progressive shift in functional connectivity reinforces pain perception even in the absence of continued nociceptive input, making chronic pain a disorder of network reorganization rather than a simple response to peripheral injury (Kim et al., 2017, Song and Zhang, 2024). Interestingly, glial plasticity (Sancho et al., 2021, Bellamy et al., 2015) is recognized as playing a role in neuronal plasticity (Sancho et al., 2021, Dzyubenko and Hermann, 2023, Ben Achour and Pascual, 2010). For instance, microglia play a crucial role in modulating post-injury neuroplasticity, influencing different types of chronic pain—nociceptive, neuropathic, and nociplastic. Understanding ectopic plasticity in somatosensory circuits may help uncover distinct pain mechanisms. Future molecular and genetic studies on microglia-mediated neuroplasticity could pave the way for novel chronic pain therapies (Hiraga et al., 2022).

Similarly, schizophrenia illustrates the devastating effects of excessive structural and functional plasticity (Stephan et al., 2006). Recent evidence suggests that exaggerated synaptic pruning during adolescence (structural plasticity) may contribute to the disruption of prefrontal-limbic connectivity (network-level plasticity), weakening top-down regulation of emotional and cognitive processes (Howes and Onwordi, 2023, Chafee and Averbeck, 2022). This excessive pruning reduces synaptic density, impairing information processing in the prefrontal cortex and contributing to core cognitive and emotional deficits characteristic of schizophrenia (Howes and Onwordi, 2023).

In addiction, maladaptive synaptic plasticity interacts with long-term network reorganization in dopaminergic reward circuits. Repeated drug exposure induces LTP at excitatory synapses in the ventral tegmental area and nucleus accumbens, strengthening compulsive drug-seeking behaviors (Nestler and Lüscher, 2019). These synaptic changes eventually remodel structural plasticity, altering dendritic architecture in reward-related brain regions, making addiction a disorder of deeply engrained maladaptive neuroplasticity (Volkow et al., 2019). Interestingly, addictive substances and drugs commonly target the mesocorticolimbic dopamine (DA) system, which originates in the ventral tegmental area (VTA) and extends its projections primarily to the nucleus accumbens (NAc) and prefrontal cortex (PFC). These drugs influence glutamatergic and GABAergic synaptic transmission within these brain regions. The resulting modifications, referred to as drug-evoked synaptic plasticity (Lüscher and Malenka, 2011), persist beyond the drug’s presence in the brain and contribute to the restructuring of neural circuits. While these initial alterations alone may not be sufficient to drive addiction, repeated exposure can reinforce them, ultimately playing a role in the development of addictive behaviors (Lüscher and Malenka, 2011).

Moreover, neurogenesis dysfunction in depression exemplifies the intricate relationship between plasticity mechanisms. Chronic stress and elevated cortisol levels suppress hippocampal neurogenesis, leading to reduced structural plasticity and impaired synaptic integration of new neurons (Schoenfeld and Gould, 2012). This loss of neurogenesis correlates with weakened hippocampal-prefrontal connectivity (network-level dysfunction), a hallmark of depression-related cognitive deficits. Given the role of neurogenesis in emotional regulation, impaired neurogenesis may underlie the persistent affective and cognitive symptoms of major depressive disorder (Liu et al., 2017, Leschik et al., 2021).

Given these cross-mechanism interactions, effective therapeutic interventions must target multiple levels of neuroplasticity simultaneously (Kumar et al., 2023). Attempts, including cognitive training, physical activity, non-invasive brain stimulation, and pharmaceuticals that enhance neuroplasticity and improve function, have been reported. Emerging approaches like virtual reality (VR), music therapy, neural rehabilitation techniques, and brain-computer interfaces (BCIs) show promise (Evancho et al., 2023, Chatterjee et al., 2021), though challenges remain, such as the need for personalized treatments and standardized protocols (Kumar et al., 2023).

4. Neuroplasticity across the lifespan

Understanding how neuroplastic mechanisms interact under pathological conditions is also crucial for developing precise, multimodal therapies that address both local synaptic dysfunction and large-scale network imbalances. Differentiating between adaptive and maladaptive plasticity is key to designing interventions that enhance beneficial outcomes while minimizing harmful effects. However, neuroplasticity is not static throughout life − it evolves from early development to aging, shaped by genetic, environmental, and lifestyle factors (Oberman and Pascual-Leone, 2013, Pascual-Leone et al., 2011). The next section explores these lifespan-related changes and their implications for cognitive resilience, neurodegenerative diseases, and rehabilitation strategies.

Neuroplasticity is not a fixed process; rather, it evolves throughout the lifespan (Marzola et al., 2023) and is significantly influenced by age (Navakkode and Kennedy, 2024), gender, hormonal fluctuations, and environmental factors (Power and Schlaggar, 2017). While the fundamental mechanisms of plasticity remain similar at different stages (Stiles, 2000), their capacity and efficiency can vary considerably, shaping cognitive function, emotional resilience, and susceptibility to neurological disorders (Valenzuela, 2019). Epigenetic modifications, which dynamically regulate gene expression in response to environmental influences, also contribute to these lifespan changes in neuroplastic potential (Nayak et al., 2022). Collectively, it is important to recall that plasticity’s efficiency also evolves with age, shaped by hormonal dynamics, environmental contexts, and epigenetic factors.

4.1. Early development

During early development, neuroplasticity is at its peak (Hensch, 2005), enabling rapid learning and adaptation (Oberman and Pascual-Leone, 2013). Synaptogenesis and pruning occur extensively in this period (Hensch, 2005), sculpting neural circuits based on environmental inputs. Critical periods (Cisneros-Franco et al., 2020), windows of heightened sensitivity to specific stimuli, underlie language acquisition, visual processing, and other core skills. Positive experiences, such as stimulation, nurturing caregiving, and rich social interaction, bolster healthy circuit formation and long-term cognitive outcomes (Hensch and Bilimoria, 2012). By contrast, adverse childhood experiences—including neglect, trauma, or chronic stress can result in maladaptive plasticity and enduring susceptibility to mood disorders (Murphy et al., 2022, Bick and Nelson, 2016), emphasizing the importance of supportive environments in establishing a resilient neurobiological foundation.

4.2. Adulthood

In adulthood, neuroplasticity persists but is modulated by lifestyle factors (Phillips, 2017) like education, physical activity (Erickson et al., 2011), social engagement, and diet (Fakhoury et al., 2022). Cognitive reserve, built through sustained learning and intellectual pursuits, confers resilience against age-related decline and certain neurological diseases (Cordeiro et al., 2024). Although less pronounced than in early development, adults can still form new synaptic connections, reorganize existing circuits, and adapt to evolving cognitive demands, particularly when regularly engaged in mentally stimulating or physically active pursuits (Hauptman et al., 2024). Studies also highlight the role of psychosocial factors, such as meaningful social relationships and stress management, in preserving and even enhancing adult neuroplasticity (Davidson and McEwen, 2012).

Nonetheless, contradictory data suggest that not all adults respond equally; genetic factors (e.g., BDNF polymorphisms (Antal et al., 2010)) may underlie differential responsiveness.

### AT: Reincarnation

#### Zero risk of reincarnation

Yahya 3 (Harun, Turkish Intellectual, “There is no Reincarnation; Death and Resurrection happen only once,” Media Monitors, 7-27, http://www.mediamonitors.net/harunyahya51.html)

Reincarnation is a superstitious belief unconfirmed by any divine source. However, there are people all over the world, apart from those who follow Indian religions, who believe in reincarnation, or rather would wish that the idea of reincarnation were true. The reason for this is that people who do not believe in religion, who deny the existence of an afterlife and are afraid of either ceasing to exist or of living in Hell forever after their death, see reincarnation as a way of overcoming these fears. Belief in reincarnation necessitates believing that one should not be afraid of death; it misleads one into thinking that he will achieve his desires by means of rebirth. However, in the Qur’an it is told us that death and resurrection occur only once. Every one of us has only one life to live in this world; after that life, we die, and after death we are brought to life again. Then we stay in the Garden or in the Fire for eternity, depending on what we have done in this world and whether we have worshipped Allah without associating partners to Him. In other words, we only live once in this world, and then we have an afterlife, which goes on forever. The Qur’an tells us quite clearly that we will not be able to return to this world after death: There is a ban on any city We have destroyed; they will not return. (Surat al-Anbiya: 95) When death comes to one of them, he says, "My Lord, send me back again. so that perhaps I may act rightly regarding the things I failed to do!" No indeed! It is just words he utters. Before them there is an interspace until the Day they are raised up. (Surat al-Muminun: 99-100) As we see from these verses, when some people meet death, they cherish the hope that they will be brought back to life again. However, at that moment it will be explained to them that this is not possible. In another verse, Allah says this about our death and rising from the dead: How can you reject Allah, when you were dead and then He gave you life, then He will make you die and then give you life again, then you will be returned to Him? (Surat al-Baqara: 28) As we see from this verse, at the very beginning we are dead: we have no existence whatsoever. Then Allah gives us life and human form from this state. Some time after this when our lives are over, our bodies decay and disintegrate into earth once again. This is our second transition to the state of being dead. All that remains is for us to rise again. This happens in the next world. We will all rise again in the afterlife; then we will understand that we can never return again to the world, and we will account for everything we did in this world. However much one may wish to adopt superstitious beliefs such as a belief in reincarnation in order to overcome the fear of death and the afterlife and so console himself, the reality is that we will never come back to this world again after our death. Everyone will die just once, and after death, in accordance with Allah's will, we will begin our everlasting life in the next world. Allah will reward a person in the Garden or punish him in the Fire for his living a life in harmony with His unity or his covering over that truth in this world. Allah is the source of eternal justice. He is infinitely Merciful and Kind, and gives humans the true and just reward for their belief or disbelief and actions.

### AT: Consequences

#### unethical

White 12 [Richard White is associate professor of philosophy Creighton University "Levinas, the Philosophy of Suffering, and the Ethics of Compassion" The Heythrop Journal Volume 53, Issue 1, pages 111–123, Published online esept 27 2011, official publication date: January 2012, Wiley]

Levinas's phenomenology of suffering is compelling and to a great extent his account is confirmed by Scarry and Amery who write from very different standpoints. However, we may note one point of concern: Levinas does not dwell on the distinction between suffering that is voluntary and suffering that is not chosen. The fighter who is hurt in the ring may actually suffer more than the innocent person who is beaten, but his suffering is not experienced as a violation because it is chosen as a means to the end of success. This suggests that suffering is not always measured by the sheer amount of physical pain. Likewise, it is more traumatic to be hurt deliberately by others, even though one may suffer exactly the same amount of pain during the course of a physical illness. Of course, Levinas argues that in a sense all pain is experienced as ‘personally directed’, but the fact that someone else has deliberately inflicted this pain on me gives it an even more repellent aspect. As Amery comments: ‘Whoever has succumbed to torture can no longer feel at home in the world. The shame of destruction cannot be erased … That one's fellow man was experienced as the antiman remains in the tortured person as accumulated horror. It blocks the view into a world in which the principle of hope rules’.[30] We would ask Levinas to what extent one can ever speak about suffering in general; perhaps it is possible, but the origin of any given suffering is an essential aspect of it.¶ The second question is: Can suffering ever be meaningful? As we have already noted, some philosophers, including Nietzsche, exalt the value of suffering because it promotes ‘character’. In Beyond Good and Evil, for example, Nietzsche claims that, ‘profound suffering makes noble’;[31] in The Gay Science, he says that, ‘only great pain is the ultimate liberator of the spirit’;[32] and in Thus Spoke Zarathustra, he describes the sickness of the modern world in trying to eliminate all pain: ‘We have invented happiness’, say the last men, ‘and they blink’.[33] Of course, it may well be the case that some kind of a struggle is necessary for living a flourishing life; and the possibility of a successful struggle also comes with the possibility of failure and the suffering that failure entails. Levinas, however, is more concerned with the most extreme forms of suffering that completely destroy the well-being of people without any possibility of redemption in the future. For example, the suffering of those who are mentally handicapped, who cannot understand their suffering or communicate it to others: ‘One can go further – and doubtless thus arrive at the essential facts of pure pain – by evoking the “pain illnesses” of beings who are psychically deprived, backward, handicapped, in their relational life and in their relationships to the Other, relationships where suffering, without losing anything of its savage malignancy, no longer covers up the totality of the mental and comes across novel lights within new horizons’.[34] How could such pure, unmediated pain finally be recuperated into the context of a meaningful life? Or following Ivan's ruminations in The Brothers Karamazov, how can we reconcile the suffering and the murder of children with the goodness of creation? How could such things ever be ‘for the best?’¶In his essay, ‘Useless Suffering’, Levinas focuses on the mountain of suffering that belongs to the 20th century. Such excessive suffering – the torture and destruction of millions of innocent people, the elderly, children, even babies, calls into question the traditional conception of God as supremely powerful and supremely good, and appears to undermine ‘theodicy’ as a lie. And today, when some people even deny that the Holocaust ever happened, it would seem that the suffering of those who endured the Holocaust has become more pointless than ever. Levinas explains: ‘This would be pain in its undiluted malignity, suffering for nothing. It renders impossible and odious every proposal and every thought which would explain it by the sins of those who have suffered or are dead’.[35] This leads him to argue that theodicy as the justification of the other person's pain, is ‘certainly the source of all immorality’.[36] – In the case of the Holocaust, it was the bystanders, and not just the perpetrators, who allowed this great evil to happen, using rationalization and justification which implied that the victims deserved their fate. In this regard, theodicy is not a good thing, but actually an evil and a temptation insofar as it turns us away from the reality of someone else's suffering by framing it within the context of a larger metaphysical order.¶ But Levinas does not claim that all suffering is meaningless. As we have already noted, he claims that even though the suffering of the other cannot be made meaningful from my perspective, my own suffering can have meaning if I suffer for the other and thereby respond to her suffering: ‘In this perspective a radical difference develops between suffering in the other, which for me is unpardonable and solicits me and calls me, and suffering in me, my own adventure of suffering, whose constitutional or congenital uselessness can take on a meaning, the only meaning to which suffering is susceptible, in becoming a suffering for the suffering – be it inexorable – of someone else’.[37] This is the perspective that Levinas intimated earlier in his essay, ‘Transcendence and Evil’, the inter-human region of being that exists insofar as I am bound to acknowledge the suffering of the Other and his claims upon me. According to Levinas: ‘It is this attention to the Other which, across the cruelties of our century – despite these cruelties, because of these cruelties – can be affirmed as the very bond of human subjectivity, even to the point of being raised to a supreme ethical principle – the only one which it is not possible to contest – a principle which can go so far as to command the hopes and practical discipline of vast human groups’.[38] This is the breakthrough of the Good, which inaugurates subjectivity; for I am summoned to responsibility by the face of the Other which commands my obedience now.¶ At one point in his essay, ‘Useless Suffering’, Levinas elaborates his position on the possibility of useful suffering, in terms of compassion, which other thinkers, including Schopenhauer, Rousseau, Nussbaum and a long line of Buddhist writers, have also viewed as the key to morality. According to Martha Nussbaum, for example, compassion is fundamental and it must be cultivated because it is the basic social emotion.[39] Levinas himself puts it this way: ‘Must not humanity now, in a faith more difficult than ever, in a faith without theodicy, continue Sacred History; a history which now demands even more of the resources of the self in each one, and appeals to its suffering inspired by the suffering of the other person, to its compassion which is a non-useless suffering (or love), which is no longer suffering “for nothing”, and which straightaway has a meaning?’[40] – In this passage, Levinas is claiming that my own suffering only becomes meaningful when it is suffering which is for the other person. But how would this be possible? One response is to say that by cultivating an awareness of the suffering of others, and by using my moral imagination to identify with their plight, I can develop my natural compassion, and in this way I could become more responsive to the needs of other people. Also, by facing up to my own suffering – and not just ignoring it – I can learn to be more aware of the sufferings of others, and in this way I will be more available to help them. Something like this would be the Buddhist position on suffering which we will briefly consider in the final section of this paper. But is this what Levinas has in mind in the passage just quoted?

#### Buddhist ethics demands pragmatism and consequentialism. Selective violations of ethical ideals are necessary if they prevent greater suffering. You should view voting aff as an ethical act of ‘skillful means’ (or, upāya).

Gordon F. Davis 24, Associate Professor of Philosophy, Carleton University, Ph.D., University of Oxford, 2024, “Why Would a Buddha Lie? Varieties of Buddhist Consequentialism,” In *Ethical Theory in Global Perspective*, State University of New York Press, pp. 159-176, https://doi.org/10.1515/9781438496870-012, JL

Rather than delving into the nuances of rule consequentialism, however, it would be worth contextualizing Goodman’s project in terms of a much simpler theme that is more integral to Buddhism’s own traditional framing: suffering. This is understood via the “four noble truths:” the truth of suffering, the truth of the cause of suffering, the truth of the end of suffering, and the truth of the path that leads to the end of suffering. Goodman discusses the issue of suffering, but pivots from the “negative” ideal of simple cessation of suffering toward a quite striking emphasis on positive goods such as enjoyment, flourishing, and happiness.

The foundational discourse in which the Buddha presented the four “noble truths” lends itself to a consequentialist interpretation. It clearly presents a guide for incrementally improving the well-being of practitioners. Meanwhile there is little in the four noble truths that sounds deontological or virtue oriented. The one possible exception is the mention of “moral discipline” (sila), consisting of right speech, right action, and right livelihood. But Goodman (2009, 222, n. 10) shows that, from the Mahāyānists’ viewpoint, this element of practice cannot coherently be equated with all of morality, since they accept that ethics also encompasses generosity and compassion, which are not covered by “moral discipline.”

In any case, Goodman does not dwell for long on the four “noble truths,” because demonstrating a role for consequentialist reasoning in Buddhist ethics requires much more. Showing that Buddhism is consequentialist requires parsing any statements of Buddhist doctrine along the lines of “end justifies means.” Goodman suggests that, for both Theravada and Mahāyāna, the ends include a plurality of positive attainments, including joy, material well-being, character development, and spiritual attainment.

But merely highlighting end(s)—whether simple, single or plural—does not make a theory consequentialist. After all, virtue theories are also “teleological,” that is, they pursue an end, and deontological theories often have a place for goal-directed action. The key question therefore seems to be whether Mahāyānists would nonetheless reject such non-consequentialist views, perhaps implicitly, by holding that (a) any rule that regulates a Buddhist’s own practice may be broken on some occasions, and that (b) trade-offs between the well-being of different groups of people are justified (e.g., sacrificing some to save others), when required for the sake of the greater good. Both of these features are characteristic of consequentialist ethics, but not of virtue-based or deontological ethics.

To determine whether Mahāyānists subscribe to (a) and (b), Goodman relies on classical texts in the Buddhist tradition rather than on surveys of contemporary Buddhist attitudes, and he highlights one author in particular: Śāntideva, the eighth-century monk who wrote a revered work called the Bodhicaryāvatāra (2006). Goodman argues persuasively that Śāntideva’s view permits—and even requires—the kind of assessment that implies both (a) and (b). That is, the view requires not only a pragmatic readiness to adapt rules skillfully (at least for advanced practitioners), but also requires transgressive actions in suitable circumstances.

Such permissible—and sometimes required—transgressions have long been envisaged in Mahāyāna texts under the heading of “skillful means” (upāya), noted earlier. But Goodman argues that Śāntideva understood how such transgressions can be theorized systematically in relation to an impartial concern for all sentient beings (something that is also characteristic of many consequentialist ethical theories). The idea is that bodhisattvas reshape their moral agency in the form of constant service to all others. On this view, for example, not only kind lies may be justified, but also some calculated ones, that is, lies intended to bring about certain desirable consequences. This idea of calculating outcomes applies to other so-called “transgressions.”

But a problem seems to arise immediately: in Buddhism, whereas most sentient beings are deluded and require help, the bodhisattva is a specially qualified agent, guided by advanced practical wisdom. Is there not then something special about bodhisattvas? Could they have a status or importance that others lack (whereas a commitment to consequentialism would have ruled out such preferential treatment)? One of the most interesting aspects of Goodman’s discussion of Śāntideva is his teasing out, from the latter’s texts, a number of indications that Śāntideva would not, in fact, prioritize his own agency or that of any bodhisattva, or give either any special status. This may sound simply equitable in spirit, but there is more to this move than meets the eye.

Goodman connects this to another theme in Buddhist philosophy—one clearly signaled in Śāntideva—namely, the ethical significance of the no-self insight, or anātman. This insight casts doubt not only on the so-called “seat of consciousness” (e.g., self as underlying substance), but also on the notion of a unified agent.6 If we accept this insight, then not only do “one’s own” benefits matter no more than any other’s; but also, one’s ability to perform a good deed is not to be cherished, especially whenever some other agent is in a better position to deliver the best result, and yet not to be cherished in other cases, either, insofar as people often—and often deleteriously—indulge an illusory “sense of agency.” In this way, Goodman explains how the Buddhist no-self insight can justify what is called “agent-neutrality” (rather than “agent-relative” priorities), even in moral contexts.7 And agent-neutrality is sometimes taken as the hallmark of consequentialist morality.

In the spirit of Goodman’s approach, it is worth emphasizing that these consequentialist themes are not merely a few minor considerations lurking in specialized contexts or in a few selected texts. A case could be made that they are key to the deepest message of Mahāyāna philosophy. No-self is liberating; all Buddhists accept that. But in recommending the path to liberation, earlier Buddhists implied that an enlightened person should never sacrifice their spiritual attainments. But should they not—on the contrary—be willing to sacrifice anything “of their own” to help others access similar attainments? Such sacrifices—for lack of a better word—seem to follow from accepting the no-self insight; in other words, one’s “own” status cannot stand in the way, as there is really no such thing. It is not that taking oneself out of the picture is a way of “annihilating” an existing thing; emptiness means that “oneself” was never there in the first place, and thus could never have justified any agent-relativity.