## 1

#### The meta-ethic is Naturalism. Pleasure and pain *are* intrinsic value and disvalue.

Blum et al. 18 Kenneth Blum, 1Department of Psychiatry, Boonshoft School of Medicine, Dayton VA Medical Center, Wright State University, Dayton, OH, USA 2Department of Psychiatry, McKnight Brain Institute, University of Florida College of Medicine, Gainesville, FL, USA 3Department of Psychiatry and Behavioral Sciences, Keck Medicine University of Southern California, Los Angeles, CA, USA 4Division of Applied Clinical Research & Education, Dominion Diagnostics, LLC, North Kingstown, RI, USA 5Department of Precision Medicine, Geneus Health LLC, San Antonio, TX, USA 6Department of Addiction Research & Therapy, Nupathways Inc., Innsbrook, MO, USA 7Department of Clinical Neurology, Path Foundation, New York, NY, USA 8Division of Neuroscience-Based Addiction Therapy, The Shores Treatment & Recovery Center, Port Saint Lucie, FL, USA 9Institute of Psychology, Eötvös Loránd University, Budapest, Hungary 10Division of Addiction Research, Dominion Diagnostics, LLC. North Kingston, RI, USA 11Victory Nutrition International, Lederach, PA., USA 12National Human Genome Center at Howard University, Washington, DC., USA, Marjorie Gondré-Lewis, 12National Human Genome Center at Howard University, Washington, DC., USA 13Departments of Anatomy and Psychiatry, Howard University College of Medicine, Washington, DC US, Bruce Steinberg, 4Division of Applied Clinical Research & Education, Dominion Diagnostics, LLC, North Kingstown, RI, USA, Igor Elman, 15Department Psychiatry, Cooper University School of Medicine, Camden, NJ, USA, David Baron, 3Department of Psychiatry and Behavioral Sciences, Keck Medicine University of Southern California, Los Angeles, CA, USA, Edward J Modestino, 14Department of Psychology, Curry College, Milton, MA, USA, Rajendra D Badgaiyan, 15Department Psychiatry, Cooper University School of Medicine, Camden, NJ, USA, Mark S Gold 16Department of Psychiatry, Washington University, St. Louis, MO, USA, “Our evolved unique pleasure circuit makes humans different from apes: Reconsideration of data derived from animal studies”, U.S. Department of Veterans Affairs, 28 February 2018, accessed: 19 August 2020, <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6446569/>, R.S.

**Pleasure** is not only one of the three primary reward functions but it also **defines reward.** As homeostasis explains the functions of only a limited number of rewards, the principal reason why particular stimuli, objects, events, situations, and activities are rewarding may be due to pleasure. This applies first of all to sex and to the primary homeostatic rewards of food and liquid and extends to money, taste, beauty, social encounters and nonmaterial, internally set, and intrinsic rewards. Pleasure, as the primary effect of rewards, drives the prime reward functions of learning, approach behavior, and decision making and provides the **basis for hedonic theories** of reward function. We are attracted by most rewards and exert intense efforts to obtain them, just because they are enjoyable [10]. Pleasure is a passive reaction that derives from the experience or prediction of reward and may lead to a long-lasting state of happiness. The word happiness is difficult to define. In fact, just obtaining physical pleasure may not be enough. One key to happiness involves a network of good friends. However, it is not obvious how the higher forms of satisfaction and pleasure are related to an ice cream cone, or to your team winning a sporting event. Recent multidisciplinary research, using both humans and detailed invasive brain analysis of animals has discovered some critical ways that the brain processes pleasure [14]. Pleasure as a hallmark of reward is sufficient for defining a reward, but it may not be necessary. A reward may generate positive learning and approach behavior simply because it contains substances that are essential for body function. When we are hungry, we may eat bad and unpleasant meals. A monkey who receives hundreds of small drops of water every morning in the laboratory is unlikely to feel a rush of pleasure every time it gets the 0.1 ml. Nevertheless, with these precautions in mind, we may define any stimulus, object, event, activity, or situation that has the potential to produce pleasure as a reward. In the context of reward deficiency or for disorders of addiction, homeostasis pursues pharmacological treatments: drugs to treat drug addiction, obesity, and other compulsive behaviors. The theory of allostasis suggests broader approaches - such as re-expanding the range of possible pleasures and providing opportunities to expend effort in their pursuit. [15]. It is noteworthy, the first animal studies eliciting approach behavior by electrical brain stimulation interpreted their findings as a discovery of the brain’s pleasure centers [16] which were later partly associated with midbrain dopamine neurons [17–19] despite the notorious difficulties of identifying emotions in animals. Evolutionary theories of pleasure: The love connection BO:D Charles Darwin and other biological scientists that have examined the biological evolution and its basic principles found various mechanisms that steer behavior and biological development. Besides their theory on natural selection, it was particularly the sexual selection process that gained significance in the latter context over the last century, especially when it comes to the question of what makes us “what we are,” i.e., human. However, the capacity to sexually select and evolve is not at all a human accomplishment alone or a sign of our uniqueness; yet, we humans, as it seems, are ingenious in fooling ourselves and others–when we are in love or desperately search for it. It is well established that modern biological theory conjectures that **organisms are** the **result of evolutionary competition.** In fact, Richard Dawkins stresses gene survival and propagation as the basic mechanism of life [20]. Only genes that lead to the fittest phenotype will make it. It is noteworthy that the phenotype is selected based on behavior that maximizes gene propagation. To do so, the phenotype must survive and generate offspring, and be better at it than its competitors. Thus, the ultimate, distal function of rewards is to increase evolutionary fitness by ensuring the survival of the organism and reproduction. It is agreed that learning, approach, economic decisions, and positive emotions are the proximal functions through which phenotypes obtain other necessary nutrients for survival, mating, and care for offspring. Behavioral reward functions have evolved to help individuals to survive and propagate their genes. Apparently, people need to live well and long enough to reproduce. Most would agree that homo-sapiens do so by ingesting the substances that make their bodies function properly. For this reason, foods and drinks are rewards. Additional rewards, including those used for economic exchanges, ensure sufficient palatable food and drink supply. Mating and gene propagation is supported by powerful sexual attraction. Additional properties, like body form, augment the chance to mate and nourish and defend offspring and are therefore also rewards. Care for offspring until they can reproduce themselves helps gene propagation and is rewarding; otherwise, many believe mating is useless. According to David E Comings, as any small edge will ultimately result in evolutionary advantage [21], additional reward mechanisms like novelty seeking and exploration widen the spectrum of available rewards and thus enhance the chance for survival, reproduction, and ultimate gene propagation. These functions may help us to obtain the benefits of distant rewards that are determined by our own interests and not immediately available in the environment. Thus the distal reward function in gene propagation and evolutionary fitness defines the proximal reward functions that we see in everyday behavior. That is why foods, drinks, mates, and offspring are rewarding. There have been theories linking pleasure as a required component of health benefits salutogenesis, (salugenesis). In essence, under these terms, pleasure is described as a state or feeling of happiness and satisfaction resulting from an experience that one enjoys. Regarding pleasure, it is a double-edged sword, on the one hand, it promotes positive feelings (like mindfulness) and even better cognition, possibly through the release of dopamine [22]. But on the other hand, pleasure simultaneously encourages addiction and other negative behaviors, i.e., motivational toxicity. It is a complex neurobiological phenomenon, relying on reward circuitry or limbic activity. It is important to realize that through the “Brain Reward Cascade” (BRC) endorphin and endogenous morphinergic mechanisms may play a role [23]. While natural rewards are essential for survival and appetitive motivation leading to beneficial biological behaviors like eating, sex, and reproduction, crucial social interactions seem to further facilitate the positive effects exerted by pleasurable experiences. Indeed, experimentation with addictive drugs is capable of directly acting on reward pathways and causing deterioration of these systems promoting hypodopaminergia [24]. Most would agree that pleasurable activities can stimulate personal growth and may help to induce healthy behavioral changes, including stress management [25]. The work of Esch and Stefano [26] concerning the link between compassion and love implicate the brain reward system, and pleasure induction suggests that social contact in general, i.e., love, attachment, and compassion, can be highly effective in stress reduction, survival, and overall health. Understanding the role of neurotransmission and pleasurable states both positive and negative have been adequately studied over many decades [26–37], but comparative anatomical and neurobiological function between animals and homo sapiens appear to be required and seem to be in an infancy stage. Finding happiness is different between apes and humans As stated earlier in this expert opinion one key to happiness involves a network of good friends [38]. However, it is not entirely clear exactly how the higher forms of satisfaction and pleasure are related to a sugar rush, winning a sports event or even sky diving, all of which augment dopamine release at the reward brain site. Recent multidisciplinary research, using both humans and detailed invasive brain analysis of animals has discovered some critical ways that the brain processes pleasure. Remarkably, there are pathways for ordinary liking and pleasure, which are limited in scope as described above in this commentary. However, there are **many brain regions**, often termed hot and cold spots, that significantly **modulate** (increase or decrease) our **pleasure or** even produce **the opposite** of pleasure— that is disgust and fear [39]. One specific region of the nucleus accumbens is organized like a computer keyboard, with particular stimulus triggers in rows— producing an increase and decrease of pleasure and disgust. Moreover, the cortex has unique roles in the cognitive evaluation of our feelings of pleasure [40]. Importantly, the interplay of these multiple triggers and the higher brain centers in the prefrontal cortex are very intricate and are just being uncovered. Desire and reward centers It is surprising that many different sources of pleasure activate the same circuits between the mesocorticolimbic regions (Figure 1). Reward and desire are two aspects pleasure induction and have a very widespread, large circuit. Some part of this circuit distinguishes between desire and dread. The so-called pleasure circuitry called “REWARD” involves a well-known dopamine pathway in the mesolimbic system that can influence both pleasure and motivation. In simplest terms, the well-established mesolimbic system is a dopamine circuit for reward. It starts in the ventral tegmental area (VTA) of the midbrain and travels to the nucleus accumbens (Figure 2). It is the cornerstone target to all addictions. The VTA is encompassed with neurons using glutamate, GABA, and dopamine. The nucleus accumbens (NAc) is located within the ventral striatum and is divided into two sub-regions—the motor and limbic regions associated with its core and shell, respectively. The NAc has spiny neurons that receive dopamine from the VTA and glutamate (a dopamine driver) from the hippocampus, amygdala and medial prefrontal cortex. Subsequently, the NAc projects GABA signals to an area termed the ventral pallidum (VP). The region is a relay station in the limbic loop of the basal ganglia, critical for motivation, behavior, emotions and the “Feel Good” response. This defined system of the brain is involved in all addictions –substance, and non –substance related. In 1995, our laboratory coined the term “Reward Deficiency Syndrome” (RDS) to describe genetic and epigenetic induced hypodopaminergia in the “Brain Reward Cascade” that contribute to addiction and compulsive behaviors [3,6,41]. Furthermore, ordinary “liking” of something, or pure pleasure, is represented by small regions mainly in the limbic system (old reptilian part of the brain). These may be part of larger neural circuits. In Latin, hedus is the term for “sweet”; and in Greek, hodone is the term for “pleasure.” Thus, the word Hedonic is now referring to various subcomponents of pleasure: some associated with purely sensory and others with more complex emotions involving morals, aesthetics, and social interactions. The capacity to have pleasure is part of being healthy and may even extend life, especially if linked to optimism as a dopaminergic response [42]. Psychiatric illness often includes symptoms of an abnormal inability to experience pleasure, referred to as anhedonia. A negative feeling state is called dysphoria, which can consist of many emotions such as pain, depression, anxiety, fear, and disgust. Previously many scientists used animal research to uncover the complex mechanisms of pleasure, liking, motivation and even emotions like panic and fear, as discussed above [43]. However, as a significant amount of related research about the specific brain regions of pleasure/reward circuitry has been derived from invasive studies of animals, these cannot be directly compared with subjective states experienced by humans. In an attempt to resolve the controversy regarding the causal contributions of mesolimbic dopamine systems to reward, we have previously evaluated the three-main competing explanatory categories: “liking,” “learning,” and “wanting” [3]. That is, dopamine may mediate (a) liking: the hedonic impact of reward, (b) learning: learned predictions about rewarding effects, or (c) wanting: the pursuit of rewards by attributing incentive salience to reward-related stimuli [44]. We have evaluated these hypotheses, especially as they relate to the RDS, and we find that the incentive salience or “wanting” hypothesis of dopaminergic functioning is supported by a majority of the scientific evidence. Various neuroimaging studies have shown that anticipated behaviors such as sex and gaming, delicious foods and drugs of abuse all affect brain regions associated with reward networks, and may not be unidirectional. Drugs of abuse enhance dopamine signaling which sensitizes mesolimbic brain mechanisms that apparently evolved explicitly to attribute incentive salience to various rewards [45]. Addictive substances are voluntarily self-administered, and they enhance (directly or indirectly) dopaminergic synaptic function in the NAc. This activation of the brain reward networks (producing the ecstatic “high” that users seek). Although these circuits were initially thought to encode a set point of hedonic tone, it is now being considered to be far more complicated in function, also encoding attention, reward expectancy, disconfirmation of reward expectancy, and incentive motivation [46]. The argument about addiction as a disease may be confused with a predisposition to substance and nonsubstance rewards relative to the extreme effect of drugs of abuse on brain neurochemistry. The former sets up an individual to be at high risk through both genetic polymorphisms in reward genes as well as harmful epigenetic insult. Some Psychologists, even with all the data, still infer that addiction is not a disease [47]. Elevated stress levels, together with polymorphisms (genetic variations) of various dopaminergic genes and the genes related to other neurotransmitters (and their genetic variants), and may have an additive effect on vulnerability to various addictions [48]. In this regard, Vanyukov, et al. [48] suggested based on review that whereas the gateway hypothesis does not specify mechanistic connections between “stages,” and does not extend to the risks for addictions the concept of common liability to addictions may be more parsimonious. The latter theory is grounded in genetic theory and supported by data identifying common sources of variation in the risk for specific addictions (e.g., RDS). This commonality has identifiable neurobiological substrate and plausible evolutionary explanations. Over many years the controversy of dopamine involvement in especially “pleasure” has led to confusion concerning separating motivation from actual pleasure (wanting versus liking) [49]. We take the position that animal studies cannot provide real clinical information as described by self-reports in humans. As mentioned earlier and in the abstract, on November 23rd, 2017, evidence for our concerns was discovered [50] In essence, although nonhuman primate brains are similar to our own, the disparity between other primates and those of human cognitive abilities tells us that surface similarity is not the whole story. Sousa et al. [50] small case found various differentially expressed genes, to associate with pleasure related systems. Furthermore, the dopaminergic interneurons located in the human neocortex were absent from the neocortex of nonhuman African apes. Such differences in neuronal transcriptional programs may underlie a variety of neurodevelopmental disorders. In simpler terms, the system controls the production of dopamine, a chemical messenger that plays a significant role in pleasure and rewards. The senior author, Dr. Nenad Sestan from Yale, stated: “Humans have evolved a dopamine system that is different than the one in chimpanzees.” This may explain why the behavior of humans is so unique from that of non-human primates, even though our brains are so surprisingly similar, Sestan said: “It might also shed light on why people are vulnerable to mental disorders such as autism (possibly even addiction).” Remarkably, this research finding emerged from an extensive, multicenter collaboration to compare the brains across several species. These researchers examined 247 specimens of neural tissue from six humans, five chimpanzees, and five macaque monkeys. Moreover, these investigators analyzed which genes were turned on or off in 16 regions of the brain. While the differences among species were subtle, **there was** a **remarkable contrast in** the **neocortices**, specifically in an area of the brain that is much more developed in humans than in chimpanzees. In fact, these researchers found that a gene called tyrosine hydroxylase (TH) for the enzyme, responsible for the production of dopamine, was expressed in the neocortex of humans, but not chimpanzees. As discussed earlier, dopamine is best known for its essential role within the brain’s reward system; the very system that responds to everything from sex, to gambling, to food, and to addictive drugs. However, dopamine also assists in regulating emotional responses, memory, and movement. Notably, abnormal dopamine levels have been linked to disorders including Parkinson’s, schizophrenia and spectrum disorders such as autism and addiction or RDS. Nora Volkow, the director of NIDA, pointed out that one alluring possibility is that the neurotransmitter dopamine plays a substantial role in humans’ ability to pursue various rewards that are perhaps months or even years away in the future. This same idea has been suggested by Dr. Robert Sapolsky, a professor of biology and neurology at Stanford University. Dr. Sapolsky cited evidence that dopamine levels rise dramatically in humans when we anticipate potential rewards that are uncertain and even far off in our futures, such as retirement or even the possible alterlife. This may explain what often motivates people to work for things that have no apparent short-term benefit [51]. In similar work, Volkow and Bale [52] proposed a model in which dopamine can favor NOW processes through phasic signaling in reward circuits or LATER processes through tonic signaling in control circuits. Specifically, they suggest that through its modulation of the orbitofrontal cortex, which processes salience attribution, dopamine also enables shilting from NOW to LATER, while its modulation of the insula, which processes interoceptive information, influences the probability of selecting NOW versus LATER actions based on an individual’s physiological state. This hypothesis further supports the concept that disruptions along these circuits contribute to diverse pathologies, including obesity and addiction or RDS.

#### The standard is maximizing expected well-being. Prefer:

#### [1] Actor specificity: util is the best for governments, which is the actor in the rez – multiple warrants – a] Governments must aggregate since every policy benefits some and harms others, which also means side constraints freeze action b] No intent-foresight distinction – the actions we take are inevitably informed by predictions from certain mental states, meaning consequences are a collective part of the will c] No act omission distinction – governments are responsible for everything in the public sphere so inaction is an implicit authorization of action d] Actor-specificity comes first since different agents have different ethical standings. Takes out util calc indicts since they’re empirically denied and link turns them because the alt would be no action.

#### [2] No calc indicts – a] no philosophy actually says that consequences don’t matter at all since otherwise it would indict every theory since they use causal events for ethics b] winning hedonism proves we’re the only one with impacts to it so a risk of offense is sufficient c] they’re blippy nibs that set us at a disadvantage since they only have to win one while we have to beat them all – kills fairness

## 2

#### CP text: A just government ought to recognize a conditional right of workers to strike. The right to strike ought to be conditional upon one’s profession, with all workers except healthcare workers being guaranteed an unconditional right to strike.

#### It’s competitive – a] the CP offers a conditional right, meaning it only applies in some instances, so it’s necessarily competitive and b] the plan defends all workers – 1ar clarification causes shiftiness that means we lose every time since we can’t generate new links in the 2nr.

#### Nurse strikes devastates hospitals

Wright 10 Sarah H. Wright July 2010 "Evidence on the Effects of Nurses' Strikes" <https://www.nber.org/digest/jul10/evidence-effects-nurses-strikes> (Researcher at National Bureau of Economic Research)

U.S. hospitals were excluded from collective bargaining laws for three decades longer than other sectors because of fears **that strikes by nurses might imperil patients' health**. Today, while unionization has been declining in general, it is growing rapidly in hospitals, with the number of unionized workers rising from 679,000 in 1990 to nearly one million in 2008. In Do Strikes Kill? Evidence from New York State (NBER Working Paper No. 15855), co-authors Jonathan Gruber and Samuel Kleiner carefully examine the effects of nursing strikes on patient care and outcomes. The researchers match data on nurses' strikes in New York State from 1984 to 2004 to data on hospital discharges, including information on treatment intensity, patient mortality, and hospital readmission. They conclude that nurses' strikes were **costly to hospital patients**: in-hospital mortality **increased by 19.4 percent** and hospital readmissions **increased by 6.5 percen**t for patients admitted during a strike. Among their sample of 38,228 such patients, an estimated **138 more individuals died than would have without a stri**ke, and 344 more patients were readmitted to the hospital than if there had been no strike. "Hospitals functioning during nurses' strikes **do so at a lower quality of patient care,"** they write. Still, at hospitals experiencing strikes, the measures of treatment intensity -- that is, the length of hospital stay and the number of procedures performed during the patient's stay -- show no significant differences between striking and non-striking periods. Patients appear to receive the same intensity of care during union work stoppages as during normal hospital operations. Thus, the poor outcomes associated with strikes suggest that they might reduce hospital productivity. These poor health outcomes increased for both emergency and non-emergency hospital patients, even as admissions of both groups decreased by about 28 percent at hospitals with strikes. The poor health outcomes were not apparent either before or after the strike in the striking hospitals, suggesting that they are attributable to the strike itself. And, the poor health outcomes do not appear to do be due to different types of patients being admitted during strike periods, because patients admitted during a strike are very similar to those admitted during other periods. Hiring replacement workers apparently does not help: hospitals that hired replacement workers **performed no better** during strikes than those that did not hire substitute employees. In each case, patients with conditions that required intensive nursing were more likely to fare worse in the presence of nurses' strikes.

#### Hospitals are the critical internal link for pandemic preparedness.

Al Thobaity 20, Abdullelah, and Farhan Alshammari. "Nurses on the frontline against the COVID-19 pandemic: an Integrative review." Dubai Medical Journal 3.3 (2020): 87-92. (Associate Professor of Nursing at Taif University)

The majority of infected or symptomatic people seek medical treatment in medical facilities, particularly hospitals, as a high number of cases, especially those in critical condition, will have an impact on hospitals [4]. The concept of hospital resilience in disaster situations is defined as the ability to recover from the damage caused by huge disturbances quickly [2]. The resilience of hospitals to pandemic cases depends on the preparedness of the institutions, and not all hospitals have the same resilience. A lower resilience will affect the **sustainability of the health services**. This also affects healthcare providers such as doctors, nurses, and allied health professionals [5, 6]. Despite the impact on healthcare providers, excellent management of a pandemic depends on the level of **preparedness of healthcare providers, including nurses**. This means that if it was impossible to be ready before a crisis or disaster, responsible people will do all but the impossible to save lives.

#### New Pandemics are deadlier and faster are coming – COVID is just the beginning

Antonelli 20 Ashley Fuoco Antonelli 5-15-2020 <https://www.advisory.com/daily-briefing/2020/05/15/weekly-line> "Weekly line: Why deadly disease outbreaks could become more common—even after Covid-19" (Associate Editor — American Health Line)

While the new coronavirus pandemic suddenly took the world by storm, the truth is public health experts for years have warned that a virus similar to the new coronavirus would cause the next pandemic—and they say **deadly infectious disease outbreaks could become more common**. Infectious disease experts are always on the lookout for the next pandemic, and in a report published two years ago, researchers from the Johns Hopkins Bloomberg School of Public Health **predicted that the pathogen most likely to cause the next pandemic would be a virus similar to the common cold**. Specifically, the researchers predicted that the pathogen at fault for the next pandemic would be: A microbe for which people have not yet **developed immunities**, meaning that a large portion of the human population would be susceptible to infection; Contagious during the so-called "incubation period"—the time when people are infected with a pathogen but are not yet showing symptoms of the infection or are showing only mild symptoms; and Resistant to any known prevention or treatment methods. The researchers also concluded that such a pathogen would have a "low but significant" fatality rate, meaning the pathogen wouldn't kill human hosts fast enough to inhibit its spread. As **Amesh Adalja**—a senior scholar at the Johns Hopkins Center for Health Security, who led the report—told Live Science's Rachael Rettner at the time, "**It just has to make a lot of people sick" to disrupt society**. The researchers said RNA viruses—which include the common cold, influenza, and severe acute respiratory syndrome (or SARS, which is caused by a type of coronavirus)—fit that bill. And even though we had a good bit of experience dealing with common RNA viruses like the flu, Adalja at the time told Rettner that there were "a whole host of viral families that get very little attention when it comes to pandemic preparedness." Not even two years later, the new coronavirus, which causes Covid-19, emerged and quickly spread throughout the world, reaching pandemic status in just a few months. To date, officials have reported more than 4.4 million cases of Covid-19 and 302,160 deaths tied to the new coronavirus globally. In the United States, the number of reported Covid-19 cases has reached more than 1.4 million and the number of reported deaths tied to the new coronavirus has risen to nearly 86,000 in just over three months. Although public health experts had warned about the likelihood of a respiratory-borne RNA virus causing the next global pandemic, many say the world was largely unprepared to handle this type of infectious disease outbreak. And as concerning as that revelation may be on its own, **perhaps even more worrisome is that public health experts predict life-threatening infectious disease outbreaks are likely to become more common—meaning we could be susceptible to another pandemic in the future**. Why experts think deadly infectious disease outbreaks could become more common As the Los Angeles Times's Joshua Emerson Smith notes, infectious disease experts for more than ten years now have noted that "[o]utbreaks of dangerous new diseases with the potential to become pandemics have been on the rise—from HIV to swine flu to SARS to Ebola." For instance, a report published in Nature in 2008 found that **the number of emerging infectious disease events that occurred in the 1990s was more than three times higher than it was in the 1940s**. Many experts believe the recent increase in infectious disease outbreaks is tied to human behaviors that disrupt the environment, "such as **deforestation and poaching**," which have led "to increased contact between highly mobile, urbanized human populations and wild animals," Emerson Smith writes. In the 2008 report, for example, researchers noted that about 60% of 355 emerging infectious disease events that occurred over a 50-year period could be largely linked to wild animals, livestock, and, to a lesser extent, pets. Now, researchers believe the new coronavirus first jumped to humans from animals at a wildlife market in Wuhan, China. Along those same lines, some experts have argued that global climate change has driven an increase in infectious diseases—and could continue to do so. A federally mandated report released by the U.S. Global Change Research Program in 2018 warned that warmer temperatures could expand the geographic range covered by disease-carrying insects and pests, which could result in more Americans being exposed to ticks carrying Lyme disease and mosquitos carrying the dengue, West Nile, and Zika viruses. And experts now say continued warming in global temperatures, deforestation, and other environmentally disruptive behaviors have broadened that risk by bringing more people into contact with disease-carrying animals. Further, experts note that infectious diseases today are able to spread much faster and farther than they could decades ago because of increasing globalization and travel. While some have suggested the Covid-19 pandemic could stifle that trend, others argue globalization is likely to continue—meaning so could infectious diseases' far spread.

#### Future pandemics will cause extinction – it only takes one ‘super-spreader’ – US prevention is key

Bar-Yam 16 Yaneer Bar-Yam 7-3-2016 “Transition to extinction: Pandemics in a connected world” <http://necsi.edu/research/social/pandemics/transition> (Professor and President, New England Complex System Institute; PhD in Physics, MIT)

Watch as one of the more aggressive—brighter red — strains rapidly expands. After a time it goes extinct leaving a black region. Why does it go extinct? The answer is that it spreads so rapidly that it kills the hosts around it. Without new hosts to infect it then dies out itself. That the rapidly spreading pathogens die out has important implications for evolutionary research which we have talked about elsewhere [1–7]. In the research I want to discuss here, what we were interested in is the effect of adding long range transportation [8]. This includes natural means of dispersal as well as unintentional dispersal by humans, like adding airplane routes, which is being done by real world airlines (Figure 2). When we introduce long range transportation into the model, the success of more aggressive strains changes. They can use the long range transportation to find new hosts and escape local extinction. Figure 3 shows that the more transportation routes introduced into the model, the more higher aggressive pathogens are able to survive and spread. As we add more long range transportation, there is a critical point at which pathogens become so aggressive that the entire host population dies. The pathogens die at the same time, but that is not exactly a consolation to the hosts. We call this the phase transition to extinction (Figure 4). With increasing levels of global transportation, human civilization may be approaching such a critical threshold. In the paper we wrote in 2006 about the dangers of global transportation for pathogen evolution and pandemics [8], we mentioned the risk from Ebola. Ebola is a horrendous disease that was present only in isolated villages in Africa. It was far away from the rest of the world only because of that isolation. Since Africa was developing, it was only a matter of time before it reached population centers and airports. While the model is about evolution, it is really about which pathogens will be found in a system that is highly connected, and Ebola can spread in a highly connected world. The traditional approach to public health uses historical evidence analyzed statistically to assess the potential impacts of a disease. As a result, many were surprised by the spread of Ebola through West Africa in 2014. As the connectivity of the world increases, past experience is not a good guide to future events. A key point about the phase transition to extinction is its suddenness. Even a system that seems stable, can be destabilized by a few more long-range connections, and connectivity is continuing to increase. So how close are we to the tipping point? We don’t know but it would be good to find out before it happens. While Ebola ravaged three countries in West Africa, it only resulted in a handful of cases outside that region. One possible reason is that many of the airlines that fly to west Africa stopped or reduced flights during the epidemic [9]. In the absence of a clear connection, public health authorities who downplayed the dangers of the epidemic spreading to the West might seem to be vindicated. As with the choice of airlines to stop flying to west Africa, our analysis didn’t take into consideration how people respond to epidemics. It does tell us what the outcome will be unless we respond fast enough and well enough to stop the spread of future diseases, which may not be the same as the ones we saw in the past. As the world becomes more connected, the dangers increase. Are people in western countries safe because of higher quality health systems? Countries like the U.S. have highly skewed networks of social interactions with some very highly connected individuals that can be “superspreaders.” The chances of such an individual becoming infected may be low but events like a mass outbreak pose a much greater risk if they do happen. If a sick food service worker in an airport infects 100 passengers, or a contagion event happens in mass transportation, an outbreak could very well prove unstoppable.

#### Extinction outweighs under any framework

Pummer 15 [Theron, Junior Research Fellow in Philosophy at St. Anne's College, University of Oxford. “Moral Agreement on Saving the World” Practical Ethics, University of Oxford. May 18, 2015] AT

There appears to be lot of disagreement in moral philosophy. Whether these many apparent disagreements are deep and irresolvable, I believe there is at least one thing it is reasonable to agree on right now, whatever general moral view we adopt: that it is very important to reduce the risk that all intelligent beings on this planet are eliminated by an enormous catastrophe, such as a nuclear war. How we might in fact try to reduce such existential risks is discussed elsewhere. My claim here is only that we – whether we’re consequentialists, deontologists, or virtue ethicists – should all agree that we should try to save the world. According to consequentialism, we should maximize the good, where this is taken to be the goodness, from an impartial perspective, of outcomes. Clearly one thing that makes an outcome good is that the people in it are doing well. There is little disagreement here. If the happiness or well-being of possible future people is just as important as that of people who already exist, and if they would have good lives, it is not hard to see how reducing existential risk is easily the most important thing in the whole world. This is for the familiar reason that there are so many people who could exist in the future – there are trillions upon trillions… upon trillions. There are so many possible future people that reducing existential risk is arguably the most important thing in the world, even if the well-being of these possible people were given only 0.001% as much weight as that of existing people. Even on a wholly person-affecting view – according to which there’s nothing (apart from effects on existing people) to be said in favor of creating happy people – the case for reducing existential risk is very strong. As noted in this seminal paper, this case is strengthened by the fact that there’s a good chance that many existing people will, with the aid of life-extension technology, live very long and very high quality lives. You might think what I have just argued applies to consequentialists only. There is a tendency to assume that, if an argument appeals to consequentialist considerations (the goodness of outcomes), it is irrelevant to non-consequentialists. But that is a huge mistake. Non-consequentialism is the view that there’s more that determines rightness than the goodness of consequences or outcomes; it is not the view that the latter don’t matter. Even John Rawls wrote, “All ethical doctrines worth our attention take consequences into account in judging rightness. One which did not would simply be irrational, crazy.” Minimally plausible versions of deontology and virtue ethics must be concerned in part with promoting the good, from an impartial point of view. They’d thus imply very strong reasons to reduce existential risk, at least when this doesn’t significantly involve doing harm to others or damaging one’s character. What’s even more surprising, perhaps, is that even if our own good (or that of those near and dear to us) has much greater weight than goodness from the impartial “point of view of the universe,” indeed even if the latter is entirely morally irrelevant, we may nonetheless have very strong reasons to reduce existential risk. Even egoism, the view that each agent should maximize her own good, might imply strong reasons to reduce existential risk. It will depend, among other things, on what one’s own good consists in. If well-being consisted in pleasure only, it is somewhat harder to argue that egoism would imply strong reasons to reduce existential risk – perhaps we could argue that one would maximize her expected hedonic well-being by funding life extension technology or by having herself cryogenically frozen at the time of her bodily death as well as giving money to reduce existential risk (so that there is a world for her to live in!). I am not sure, however, how strong the reasons to do this would be. But views which imply that, if I don’t care about other people, I have no or very little reason to help them are not even minimally plausible views (in addition to hedonistic egoism, I here have in mind views that imply that one has no reason to perform an act unless one actually desires to do that act). To be minimally plausible, egoism will need to be paired with a more sophisticated account of well-being. To see this, it is enough to consider, as Plato did, the possibility of a ring of invisibility – suppose that, while wearing it, Ayn could derive some pleasure by helping the poor, but instead could derive just a bit more by severely harming them. Hedonistic egoism would absurdly imply she should do the latter. To avoid this implication, egoists would need to build something like the meaningfulness of a life into well-being, in some robust way, where this would to a significant extent be a function of other-regarding concerns (see chapter 12 of this classic intro to ethics). But once these elements are included, we can (roughly, as above) argue that this sort of egoism will imply strong reasons to reduce existential risk. Add to all of this Samuel Scheffler’s recent intriguing arguments (quick podcast version available here) that most of what makes our lives go well would be undermined if there were no future generations of intelligent persons. On his view, my life would contain vastly less well-being if (say) a year after my death the world came to an end. So obviously if Scheffler were right I’d have very strong reason to reduce existential risk. We should also take into account moral uncertainty. What is it reasonable for one to do, when one is uncertain not (only) about the empirical facts, but also about the moral facts? I’ve just argued that there’s agreement among minimally plausible ethical views that we have strong reason to reduce existential risk – not only consequentialists, but also deontologists, virtue ethicists, and sophisticated egoists should agree. But even those (hedonistic egoists) who disagree should have a significant level of confidence that they are mistaken, and that one of the above views is correct. Even if they were 90% sure that their view is the correct one (and 10% sure that one of these other ones is correct), they would have pretty strong reason, from the standpoint of moral uncertainty, to reduce existential risk. Perhaps most disturbingly still, even if we are only 1% sure that the well-being of possible future people matters, it is at least arguable that, from the standpoint of moral uncertainty, reducing existential risk is the most important thing in the world. Again, this is largely for the reason that there are so many people who could exist in the future – there are trillions upon trillions… upon trillions. (For more on this and other related issues, see this excellent dissertation). Of course, it is uncertain whether these untold trillions would, in general, have good lives. It’s possible they’ll be miserable. It is enough for my claim that there is moral agreement in the relevant sense if, at least given certain empirical claims about what future lives would most likely be like, all minimally plausible moral views would converge on the conclusion that we should try to save the world. While there are some non-crazy views that place significantly greater moral weight on avoiding suffering than on promoting happiness, for reasons others have offered (and for independent reasons I won’t get into here unless requested to), they nonetheless seem to be fairly implausible views. And even if things did not go well for our ancestors, I am optimistic that they will overall go fantastically well for our descendants, if we allow them to. I suspect that most of us alive today – at least those of us not suffering from extreme illness or poverty – have lives that are well worth living, and that things will continue to improve. Derek Parfit, whose work has emphasized future generations as well as agreement in ethics, described our situation clearly and accurately: “We live during the hinge of history. Given the scientific and technological discoveries of the last two centuries, the world has never changed as fast. We shall soon have even greater powers to transform, not only our surroundings, but ourselves and our successors. If we act wisely in the next few centuries, humanity will survive its most dangerous and decisive period. Our descendants could, if necessary, go elsewhere, spreading through this galaxy…. Our descendants might, I believe, make the further future very good. But that good future may also depend in part on us. If our selfish recklessness ends human history, we would be acting very wrongly.” (From chapter 36 of On What Matters)

PIC solves the aff – advantage is only about a small number of UNIONS being mobilized to solve their impact. We don’t PIC out of unions so we solve the Aff.

## 3

#### Interpretation – The Affirmative may not claim that 1AR theory is drop the debater, competing interps, no RVIs, and the highest layer.

#### Violation: Underview

#### Standards:

#### Infinite abuse – 1AR theory becomes a no-risk issue since you can dump a bunch of shells that I have to respond to but you can win on a risk of offense. I can’t group shells, gain a route to the ballot, or uplayer which kills norming since you can get away with the worst norms if I can’t respond.

#### Infinite abuse outweighs:

#### 1] Clash – lack of engagement tilts any education to only you. Turns and outweighs critical thinking since it refines our arguments which is more constitutive to debate.

#### Fairness is a voter – debate is competitive and needs objection evaluation. Outweighs since it determines engagement.

#### Drop the debater – 1] losing uniquely sets a norm for you and people watching that your strat won’t win 2] time reading theory trades off dropping you for it since substance is skewed for me.

#### No RVIs – 1] they’d bait theory and prep it out 2] its illogical to win for being fair – logic outweighs since it determines validity of our args

#### Competing-interps – a] race to top for norm-setting. Norming is an independent voter and outweighs since it’s the purpose of theory and better debates later on b] there’s no guarantee you will be fair always so we outweigh on scope

#### No cross-apps since we indict your practice and force them to win the counter-interp with offense – if they can be abusive and then just use random deflating takeouts it permits them getting away with abuse everytime.

## Case