Cabot RH Progressive Affirmative

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I stand in firm affirmation of today's resolution, "Resolved; The member nations of the World Trade Organization ought to reduce intellectual property protections for medicines."

Before we begin, the affirmative provides the following definitions;

Intellectual property is best defined as a unique original creation to which a party has legal ownership of, whether in the form of a patent, copyright or trademark, and is protected by law from unauthorized use from others.

The value for today's round should be Morality per the word ought in the resolution.

The standard is maximizing expected well-being, or hedonistic act utilitarianism.

1] Neuroscience- pleasure and pain *are* intrinsic value and disvalue – everything else regresses.

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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.

2] Actor spec—governments must use util because they don’t have intentions and are constantly dealing with tradeoffs—outweighs since different agents have different obligations—takes out calc indicts since they are empirically denied.

Advantage one is improving health conditions throughout the world.

The status quo has a unique problem of affordability of medicine to those who need it. Medicine prices are skyrocketed due to a unique instance in which those that own the medicine have control on distribution of price.

Medicines are inherently unaffordable to the underprivileged.

National Academy of Sciences. (2017, November 30). The affordability conundrum. Making Medicines Affordable: A National Imperative. https://www.ncbi.nlm.nih.gov/books/NBK493099/ R.H.

Determining the “value” of a drug and what constitutes “fair” pricing is a contentious and confounding topic. Various stakeholders have different concepts of the value of a drug and what a fair price for it would be. Within this dynamic, participants in the biopharmaceutical sector can each assert that their ultimate goal is to make safe and effective medicines and provide “value” to patients. However, an inherent conflict exists between the desire of patients (and society) for affordable drugs and the expectations of—as well as legal obligations to—corporate shareholders and other investors in biopharmaceutical companies for a competitive return on investment.3 In short, patients emphasize value in terms of their direct personal benefit rather than in business or economic terms (Buzaglo et al., 2016). Presently, different patients pay different prices for identical drugs, with individual prices depending mainly on the specifics of their health insurance plans, which generally include cost-sharing features such as copays, deductibles, and coinsurance. In severe financial circumstances, patients' health care expenses also adversely affect other members of their families. Consider, for example, an individual with rheumatoid arthritis who has an annual income of $55,000 (near the national median), a spouse, and two dependents. Assume that the individual's monthly payroll contribution to purchase health insurance is $400 ($4,800 yearly) and that the deductible is $3,500, coinsurance is 20 percent, and the annual out-of-pocket maximum under the individual's insurance policy is $7,000. The yearly cost of that person's medications may

well reach $30,000 if the rheumatoid arthritis is treated with an expensive specialty drug; thus, that individual will need to pay $11,800 ($4,800 for the insurance plus $7,000 for the maximum out-of-pocket expenses) each year for health-related expenses. The individual would then need to cover the rest of the family expenses with the remainder of his or her income, after taxes. This is a reality that many patients face when medical expenses consume much of their gross income. For those who are uninsured, the situation is far bleaker. Drug manufacturers often attribute the high cost of medications to the complexity of the technology and of the testing required of new products, the high failure rates associated with drugs under development, and national and international regulations intended to ensure that medicines are safe and effective (Rosenblatt, 2017; Rosenblatt and Termeer, 2017). Drug candidates must first be discovered and then tested, with each step requiring a series of intricate experiments. If the initial tests are promising, the drug candidate is then put through a series of clinical trials to determine its safety and efficacy. Gaining approval from the U.S. Food and Drug Administration (FDA) requires large, complex, multicenter—and often multinational—trials that are carried out by a network of clinical investigators, statisticians, consultants, and other professionals, all of which is very expensive. Despite the generally recognized expense of developing drugs, many individuals believe that drug companies and intermediaries in the supply chain are exploiting the complexity of the system by charging high prices for drugs without transparency and without justification. These views are common even for drugs that have moved into the generic market (Bach, 2015). To understand this concern among the public, consider, again as just one example, the case of the leukemia drug imatinib (Gleevec). Upon its U.S. release in 2001, it cost $4,540 per month of treatment. In 2016, after 15 years on the market, it cost $8,500 per month in the United States, but cost $4,500 and $3,300 per month in Germany and France, respectively (Bach, 2016). This increasing cost over time is not unique to Gleevec; cancer drug prices, for example, have on average quadrupled in the United States over the past 20 years (Bach, 2017; Conti et al., 2015; Dusetzina, 2016). In the case of Gleevec, this price increase occurred despite the presence of two factors that would normally bring prices down. First, because leukemia patients are living longer due to the drug's effectiveness and because new indications for the drug have been approved (Bennette et al., 2016), the population treated with the drug has expanded, which has increased sales volume of the drug. Second, other drugs that target the same abnormal protein have entered the market. For most types of non-medical products, such a combination would result in more options and lower costs and prices. Most other cancer drugs are considered to be less effective than Gleevec in extending the lives of cancer patients, yet when new cancer drugs enter the market, their prices are similarly high (Dusetzina and Keating, 2015). A lack of competition, combined with state and federal regulations specifying that insurers must include cancer drugs in their formularies (Bach, 2009), provides sellers with considerable pricing flexibility. These factors—and others—tend to drive the already high prices of drugs in the United States even higher, but it is not clear exactly how large a role each factor plays or how the various factors interact. One powerful force, however, is the extensive and increasing health insurance coverage for prescription drugs that blunts—and in the case of full coverage, eliminates—normal consumer-related market forces that might otherwise control prices.

The facts are clear, high drug prices cause higher insurance prices. For those that can afford it, they're left with barely anything left for their families. For those that can't, they suffer far worse.

Furthermore, there is currently a vaccine shortage during a worldwide pandemic, leaving millions in developing countries at serious risk of death;

Khatun 2021 [Dr. Fahmida, Executive Director at the Centre for Policy Dialogue, The Daily Star, "Can patent waiver for Covid-19 treatment bring vaccine equity? July 12, https://www.thedailystar.net/opinion/macro-mirror/news/can-patent-waiver-covid-19-treatment-bring-vaccine-equity-2127591

The inequality in accessing vaccines to tackle the Covid-19 pandemic is growing. Since the invention of the vaccines, there was apprehension regarding the accessibility of the vaccines by the citizens of poorer countries. The supply of vaccines is also far less than the demand. And whatever is being manufactured are being purchased by developed countries in advance and in plenty, leaving low and lower middle-income countries far behind in the vaccination drive. Many high-income countries have already managed to vaccinate a large number of their population. Whereas, most people in the least developed countries (LDCs) are still waiting for their shots and struggling to recover from the pandemic, both in terms of health and economy

Reduction of vaccines is inherently needed to end the Pandemic, all other options exhausted:

Silverman 2020 [Ed, columnist, senior writer, Stat News, "South Africa and India urge WTO to waive IP rights, widen access to Covid-19 drugs and vaccines" Oct. 3
https://www.statnews.com/pharmalot/2020/10/03/wto-covid19-coronavirus-patents-india-southafrica/
The move comes as several wealthy nations — notably, the U.S., the U.K., Germany, and France — have signed deals with various drug makers for hundreds of millions of doses of vaccines that are still being tested. But poorer countries lack the means to place such orders and global health officials fear that inequitable access will cause further immeasurable suffering and the coronavirus will not be contained. "It is crucial that other member governments of the WTO support this as we need to ensure that vaccines, drugs, and other medical tools needed for tackling COVID-19 can be scaled up by countries and their manufacturers without facing protracted negotiations for licenses that in most cases exclude many high burden countries," said Leena Menghaney, who heads the Doctors Without Borders access campaign in South Asia, in a statement. Specifically, India and South Africa proposed waiving rules that govern patents, industrial designs, copyrights, and protection of undisclosed information, a reference to trade secrets.

This should not be taken lightly, as COVID-19 has caused millions of deaths, and variants will continue the destruction:

David H. **Freedman**, 8-4-**2021**, "A doomsday COVID variant worse than Delta and Lambda may be coming, scientists say," Newsweek,
https://www.newsweek.com/2021/08/13/doomsday-covid-variant-worse-delta-lambda-may-coming-scientists-say-1615874.html/rsm
Scientists keep underestimating the coronavirus. In the beginning of the pandemic, they said mutated versions of the virus wouldn't be much of a problem—until the more-infectious Alpha caused a spike in cases last fall. Then Beta made young people sicker and Gamma reinfected those who'd already recovered from COVID-19. Still, by March, as the winter surge in the U.S. receded, some epidemiologists were cautiously optimistic that the rapid vaccine rollout would soon tame the variants and cause the pandemic to wind down. Delta has now shattered that optimism. This variant, first identified in India in December, spreads faster than any previous strain of SARS-CoV-2, as the COVID-19 virus is officially named. It is driving up infection rates in every state of the U.S., prompting the Centers for Disease Control and Prevention (CDC) to once again recommend universal mask-wearing. The Delta outbreak is going to get much worse, warns Michael Osterholm, an epidemiologist who leads the Center for Infectious Disease Research and Policy at the University of Minnesota. "The number of intensive-care beds needed could be higher than any time we've seen," he says. He adds that his team's analysis shows that almost every single one of the 100 million unvaccinated Americans who hasn't had COVID-19 yet will likely get it in the coming months, short of taking the sort of strong isolation and masking precautions that seem unlikely in the vaccine-hesitant population. The variant is so contagious that it's set to smash through every previous prediction of how soon the U.S. might reach herd immunity. "We've failed to shut this down as we have other pandemics," says Jonathan Eisen, a biologist at the University of California, Davis, who studies how pathogens evolve. "It may be around forevermore, leaving us continually trying to figure out what to do next." FE_COVID_Doomsday_01 The Delta variant, which spreads faster than any previous strain of SARS-CoV-2, is driving up infection rates in the U.S. From Booster Shots to Returning to the Office It's too soon to say whether Lambda will turn out to be the next big, bad thing that COVID-19 unleashes on us. But it's a good time to wonder: Just how destructive can these variants get? Will future variants expand their attack from the lungs to the brain, the heart and other organs? Will they take a page from HIV and trick people into thinking they've recovered, only to make them sick later? Is there a Doomsday variant out there that shrugs off vaccines, spreads like wildfire and leaves more of its victims much sicker than anything we've yet seen? The odds are not high that we will see such a triple threat, but experts can't rule it out. Delta has already shown how much worse things can get. Its extreme contagiousness, with room to run freely through the tens of millions of Americans who haven't been vaccinated and millions more who have no access to vaccines in developing countries, has good odds of turning into something even more troublesome. "The next variant," says Osterholm, "could be Delta on steroids." Caught Off-Guard It wasn't supposed to happen this way. Early in the pandemic, most experts closely studying COVID-19 mutations downplayed the notion that variants would cause such serious problems. "They don't seem to make much of a difference," said Richard Neher, an evolutionary biologist at Switzerland's University of Basel, in August last year. "We probably only need to worry about it on a timescale of about five years." Today he calls Delta and other COVID-19 variants "the pandemic within the pandemic." FE_COVID_Doomsday_02 Colorized scanning electron micrograph of an apoptotic cell (tan) heavily infected with SARS-COV-2 virus particles (orange), isolated from a patient sample. NIAID Delta, more than any other variant, has reset scientists' understanding of how quickly a virus can evolve into devastating new forms. "All coronaviruses mutate, and we knew this one was mutating, too," says Sharone Green, a physician and infectious disease researcher at the University of Massachusetts Medical School. "But we didn't think the mutations would so strikingly affect transmissibility and possible evasion of immunity." It may seem surprising that scientists were caught off-guard by the rapid emergence of a more dangerous variant. But unlike most other pathogens, Eisen notes SARS-CoV-2 was largely unknown when it emerged. In

the absence of data, scientists assumed it would follow other viruses in being relatively slow to spin off much more contagious mutations. Even more important, he adds, scientists underestimated the sheer scale the pandemic would eventually achieve—a critical factor, because the more people a virus infects, the more opportunities it has to develop significant mutations. "Having billions of people infected presents a breeding ground for variants unlike anything we've ever seen with these sorts of viruses," he says. READ MORE U.S. COVID Vaccination Rate Lagging As Doomsday Variant Looms How to Convince Your Loved Ones to Get COVID Vaccine 'I Was Wrong Not To Get Vaccinated:' COVID Patient Cries From Hospital Bed SARS-CoV-2 doesn't mutate particularly quickly, compared to many pathogens. Just as with most human and other cells, a mutation occurs in a virus when it replicates but fails to make a perfect copy of its genetic material. That imperfect copy is a mutant. The COVID-19 virus doesn't have a lot of genetic material to scramble compared to most organisms—about 15 genes, versus about 3,000 genes in an E. coli bacterium, a run-of-the-mill stomach bug, and about 20,000 in a human cell. What's more, COVID-19 has genetic checking mechanisms that make it reasonably adept at avoiding replication mistakes compared to most viruses. But while COVID-19's mutation rate is on the low side—about one mutation for every 10 replications, or around a fifth of the flu's mutation rate and a tenth of HIV's—COVID-19 takes advantage of a grim numbers game. A single person infected with COVID-19 might carry 10 billion copies of the virus, enough to produce billions of mutated viruses every day. What happens to all those mutations? Almost always the answer is: nothing. The genetic scrambling is random, with the result that virtually all mutations either have no effect whatsoever on the virus, or else do something that makes the virus less effective or even renders it entirely non-functional. COVID-19 Could Increase Dementia, Other Brain Disorders for Decades to ComeREAD MORECOVID-19 Could Increase Dementia, Other Brain Disorders for Decades to Come But once in a while—perhaps every million trillion times—a random mutation confers some potentially dangerous new characteristic. What's more, much of what makes the virus dangerous has to do with a relatively small portion—the so-called spike proteins that protrude from its surface and enable the virus to latch onto and penetrate human cells. Most of the mutations we've seen so far represent tweaks to these spikes, which means it only takes a minimal change within any of the few viral genes that control the spikes to create a newly threatening mutation. But even when a virus hits the jackpot with a mutation that sharpens its ability to wreak havoc, that doesn't mean a dangerous new variant has emerged. To become a significant variant, a mutated virus has to out-replicate the far more numerous copies of the virus that already predominate in the population, and to do that it needs features that give it big advantages. What specific features will help the mutation become a better replicator and spreader in the population is determined by the environment. For example, in the case of a respiratory virus like COVID-19, the ability to travel longer distances in the air, and to latch more firmly onto cells in the nasal passage, would likely make a new strain a better contender to become a widely spreading variant. "A virus' job is just to keep propagating," says Green. "Any mutation that helps the virus survive and spread will make it more successful as a variant." All told, the chances that a virus in the population will produce a much more dangerous variant in the course of a year would normally be extremely low. But when billions of people are infected with billions of copies of a virus, all bets are off. Thanks to Delta's infectiousness, and the huge number of people whose refusal or inability to get vaccinated leaves them primed to become living COVID-19 mutation labs, the conditions are ripe to produce yet more, potentially more dangerous, variants in the coming months. FE_COVID_Doomsday_06 Thanks to Delta's infectiousness, and the huge number of people whose refusal or inability to get vaccinated leaves them primed to become living COVID-19 mutation labs, the conditions are ripe to produce yet more, potentially more dangerous, variants in the coming months Here: Anti-vaxxers in Raleigh, North Carolina. PETER ZAY/ANADOLU AGENCY/GETTY "It's going to be very difficult to stop it from happening with masks and social distancing at this point," says Preeti Malani, a physician and infectious disease researcher and chief health officer at the University of Michigan. "Vaccines are the key, and vaccine hesitancy is the obstacle." The growing number of people with natural immunity, from having recovered from COVID-19, won't save the day either, says Eric Vail, director of molecular pathology at Cedars-Sinai Medical Center. "At best it's now a third of the U.S. population with natural immunity, and that may be an overestimation," he says. "It won't be enough to guarantee that Delta will be the last big variant."

People in underdeveloped countries, along with citizens that live in poverty, don't have access to life saving medicines.

Thus the plan - The member nations of the World Trade Organization ought to reduce intellectual property rights by establishing a competition policy for the pharmaceutical field.

Intellectual property inherently removes competitive markets:

Hovenkamp, H. J., **et al. (2019**). Intellectual property and competition. Penn Law: Legal Scholarship Repository. https://scholarship.law.upenn.edu/faculty_scholarship/1807/ R.H.

IP rights and competition policy were traditionally regarded as in conflict. IP rights create monopoly, which was thought to be inimical to competition. By contrast, competition policy values free entry and asset mobility, which IP rights limit in order to create incentives. Today our view of this relationship is more complex. First, most IP rights are insufficient to produce durable monopoly, although they do facilitate product differentiation. Second, we tend to see IP rules as creating a property rights system in which competition exists for the property rights themselves. Firms compete by innovating and appropriating whatever payoffs they are able to capture, including IPRs. Third, we define competition in terms of output or welfare rather than simple rivalry. A market structure or practice that increases output is more "competitive" than a lower output alternative, even though the amount of daily rivalry among firms is less. For example, output in the cellular phone market is much higher because hardware, software, and telecommunications links are all networked by cooperative agreements and standard setting. Under conventional neoclassical assumptions, both innovation and competition increase output, whether measured by the number of units or their quality. At the same time, however, excessive IP protection limits competition by reducing asset mobility further than necessary to facilitate innovation.

Competitive markets are necessary to higher quality products, along with lower prices:

Schmitt, E. (2021). The Importance of Market Competition. https://ago.mo.gov/civil-division/consumer/antitrust-laws/the-importance-of-competition. R.H.
Competition stimulates firms to lower their own costs and run their businesses as efficiently as possible. But when competition is restricted – such as by one company acquiring most competitors or reaching agreements on prices with other competitors – prices are likely to increase and quality is likely to also suffer. If a business does not face competition, it has little incentive to lower its prices or improve its products. If there is only one seller in the market, it may charge higher prices without fearing a loss of sales to a competitor.

Competition is necessary to lower prices, and lower prices ensure higher availability of goods, inherently good. Impact is better quality of life and extension of life, and status quo currently has lack of access, so any change would be morally correct, meaning impact outweighs any disad. Furthermore, lower prices inherently cause majority net happiness, moral under hedonistic util; Pleasure most important aspect of this debate, and aff solves for saving of lives; Lives are pre-req to pleasure, thus aff outweigh any neg disad on standard debate.