### Util

#### The meta-ethic is desire.

#### 1] We can’t obtain evidence of goodness without desire – a posteriori knowledge outweighs.

Sayre-McCord 01

Geoffrey Sayre-McCord, Philosophy, University of North Carolina, Chapel Hill, "Mill's “Proof” Of The Principle of Utility: A More Than Half-Hearted Defense", Social Philosophy and Policy, 2001, accessed: 1 April 2020, <https://www.cambridge.org/core/journals/social-philosophy-and-policy/article/mills-proof-of-the-principle-of-utility-a-more-than-halfhearted-defense/FDBE07CBE08D4E17523930BF8C7BBC32>, R.S.

How is the argument supposed to go, if not by way of these multiple fallacies? Let us start with the principle of evidence and the analogy Mill draws between visibility and desirability. What is the analogy supposed to be if not one that commits Mill to interpreting "desirable" as "capable of being desired"? When it comes to visibility, no less than desirability, Mill explicitly denies that a "proof" in the "ordinary acceptation of the term" can be offered.25 As he notes, "To be incapable of proof by reasoning is com mon to all first principles; to the first premises of our knowledge, as well as to those of our conduct."26 Nonetheless, support -- that is, evidence, though not proof -- for the first premises of our **knowledge** is **provided by** "our **senses, and** our internal **consciousness.**" Mill's suggestion is that, when it comes to the first principles of conduct, desire play the same epistemic role that the senses play, when it comes to the first principles of knowledge. To understand this role, it is important to distinguish the fact that someone is sensing something from what is sensed, which is a distinction mirrored in the contrast bet ween the fact that someone is desiring something and what is desired. In the case of our senses, the evidence we have for our judgments concerning sensible qualities traces back to what is sensed, to the content of our sense-experience. Likewise, Mill is suggesting, in the case of value, the evidence we have for our judgments concerning value traces back to what is desired, to the content of our desires. Ultimately, the grounds we have for holding the principles we do must, he thinks, be traced back to our experience, to our senses and desires. Yet the evidence we have is not that we are sensing or desiring something but what it is that is sensed or desired.27 When we are having sensations of red, when what we are looking at appears red to us, we have evidence (albeit overrideable and defeasible evidence) that the thing is red. Moreover, if things never looked red to us, we could never get evidence that things were red, and would indeed never have developed the concept of redness. Similarly, when we are desiring things, when what we are considering appears good to us, we have evidence (albeit overrideable and defeasible evidence) that the thing is good. Moreover, **if we never desired** things, **we could never get evidence** that **things were good, and** would indeed **never have developed** the concept of **value.** 28 Recall that desire, for Mill, like taste, touch, sight, and smell, is a "passive sensibility." All of these, he holds, provide us with both the content that makes thought possible and the evidence we have for the conclusions that thought leads us to embrace. "Desiring a thing" and "thinking of it as desirable (unless for the sake of its consequences)" are treated by Mill as one an d the same, just as seeing a thing as red and thinking of it as red are one and the same.29 Accordingly, a person who desires x is a person who ipso facto sees x as desirable.30 Desiring something, for Mill, is a matter of seeing it under the guise of the good.31 This means that it is important, in the context of Mill's argument, that one not think of desires as mere preferences or as just any sort of motive. They constitute, according to Mill, a distinctive subclass of our motivational states, and are distinguished (at least in part) by t heir evaluative content.32 Thus, Mill is neither assuming nor arguing that something is good because we desire it; rather, he is depending on our desiring it as establishing that we see it as good. Mill's aim is to take what people already, and he thinks inevitably, see as desirable and argue that those views commit them to the value of the general happiness (whet her or not their desires follow the deliverances of t heir reason). Those who, like Mill, desire the general happiness already hold the view that the general happiness is desirable. They accept the claim that Mill is trying to defend. As Mill knows, however, there are many who do not have this desire -- many who desire only their own happiness, and some who even desire that others suffer. These are the people he sets out to persuade, along with others who are more generous and benevolent, but who nonetheless do not see happiness as desirable, and the only thin g desirable, as an end. Mill's argument is directed at convincing t hem all -- whether their desires follow or not -- that they have grounds for, and are in fact already com mitted to, regarding the happiness of others as valuable as an end. At the same time, while desiring something is a matter of seeing it as good, one could, on Mill's view, believe that something is good without desiring it, just as one can believe something is red without seeing it as red. While desire is supposed to be the fundamental source of our concept of, and evidence for, desirability, once the concept is in place there are contexts in which we will have reason to think it applies even when the corresponding sensible experience is lacking. Indeed, in Chapter IV, Mill is concerned not with generating a desire but with justifying the belief that happiness is desirable, and the only thing desirable, as an end, and so concerned with defending the standard for determining what should be desired.33 Mill recognizes that whatever argument he might hope to offer will need to appeal to evaluative claims people already accept (since he takes to heart Hume's caution concerning inferring an 'ought' from an 'is').34 The claim Mill thinks he can appeal to -- that one's own happiness is a good (i.e. desirable) -- is something licensed as available by people desiring their own happiness. Yet he is not supposing here that the fact that they desire their own happiness, or anything else, is proof that it is desirable, just as he would not suppose that the fact that someone sees something as red is proof that it is. Rather, he is supposing that if people desire their own happiness, or see something as red, one can rely on t hem having available, as a premise for further argument, the claim that their own happiness is desirable or that the thing is red (at least absent contrary evidence).35 As he puts it in the third paragraph, "If the end which the utilitarian doctrine proposes to itself were not, in theory and in practice, acknowledged to be an end nothing could ever convince any person that it was so." Thus, in appealing to the analogy bet ween judgments of sensible qualities and judgments of value, Mill is not trading on an ambiguity, nor does his argument here involve identifying being desirable with being desired or assuming that "desirable" means "desired." He is instead relying consistently on an empiricist account of concepts and their application -- on a view according to which we have the concepts, evidence, and knowledge we do only thanks to our having experiences of a certain sort. In the absence of the relevant experiences, he holds (with other empiricists), we would not only lack the required evidence for our judgments, we would lack the capacity to make the judgments in the first place. **In** the **presence of** the relevant **experience**s, though, **we have** both the concepts and the required **evidence** -- "not only all the proof which the case admits of, but all which it is possible to require."36

#### 2] Indifference – Even if there are a priori moral truths, I can choose to ignore them. Cognition is binding – if I put my hand on a hot stove, I can’t turn off my natural aversion to it.

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

#### 1] Pleasure and pain are intrinsic value and disvalue – everything else regresses. Evolutionary knowledge is reliable – broad consensus and robust neuroscience prove.

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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] Lexical prerequisite: threats to bodily security preclude the ability for moral actors to effectively act upon other moral theories since they are in a constant state of crisis that inhibits the ideal moral conditions which other theories presuppose

#### 3] Actor specificity:

#### ---A] Aggregation – every policy benefits some and harms others, so side constraints freeze action.

#### ---B] States lack wills or intentions since policies are collective actions.

#### ---C] No act-omission distinction—governments are responsible for everything in the public sphere, so inaction is implicit authorization of action: they have to yes/no bills, which means everything collapse to aggregation.

#### ---D] Actor-specificity first since different agents have different ethical standings. Link turns calc indicts because the alt would be *no* action.

#### 4] Extinction comes first 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, recut BWSEKL.

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)

#### 5] Use epistemic modesty – that’s multiplying the probability of a framework being true by its general contention impact –

#### ---A] It maximizes the probability of achieving net most moral value—beating a framework acts as mitigation to their impacts but the strength of that mitigation is contingent

#### ---B] EC is too high a burden—thousands of years of philosophy can’t be resolved in 40 minutes.

### PIC

(plan text) expect for medical workers which strikes would be conditional

#### COVID-19 has increased healthcare worker strikes risking health care effectiveness

Ryan Essex, Ph.D., and Sharon M. Weldon, Ph.D. 6/17/21, “Health Care Worker Strikes and the Covid Pandemic,” NEJM, www.nejm.org/doi/full/10.1056/NEJMp2103327//lhs-ap

While the heroics of health care workers have been celebrated and we’ve gained a renewed appreciation of the risks that many frontline workers face while providing fundamental services, less attention has been paid to those who have refused to work under such dangerous conditions and those who have pointed out that no health care workers needed to be placed at such high risk. Many have rightly argued that heroics were required only because of government neglect, underfunding, and lack of preparation for a pandemic that we knew was coming. Many workers are justifiably angry. Although there are no official figures, Covid-19 appears to have led to a substantial uptick in strike actions by health care workers.

In February 2020, facing an unknown “pneumonia,” experts in Hong Kong called for closing the borders in an effort to mitigate its spread until more could be ascertained about the nature of the virus (which would be labeled Covid-19 on February 11 and deemed a pandemic roughly a month later). The Hong Kong government failed to act, despite calls from experts and health care workers, with support from the general public. In late January, labor unions repeatedly called for dialogue with the government regarding border closure. When that effort failed, a vote was held on strike action, for which there was overwhelming support. From February 3 through 7, 2020, health care workers in Hong Kong went on strike, making a number of demands, including the closure of borders and a sufficient supply of PPE and facilities to manage the potential spread of the virus.

Such action has not been restricted to Hong Kong. Amid multiplying cases of Covid-19, health care workers in Zimbabwe went on strike in June 2020 because of a lack of PPE and low salaries. Indeed, strike action by health care workers has been a global phenomenon. In the United States, nurses have gone on strike, and in the United Kingdom pharmacists and nurses have threatened strike action. Doctors in South Korea launched a nationwide strike in August, and health care workers in Kenya, Spain, Bosnia, and Peru have all gone on strike at some point during the pandemic.

Health care workers even went on strike after the military coup in Myanmar in February 2021, with a spokesperson noting that they “simply [did] not want to work for the regime that staged the military coup.”1 Such action must be understood in the context of broader unrest. In Venezuela, for example, many health care workers have had no option to stop working during the pandemic. In what has been described as a crisis within a crisis, Covid-19 has exacerbated many of the problems of Venezuela’s ailing health care system. Though there has been unrest, the Venezuelan government has attempted to silence critics, deny PPE shortages, and blame health care workers. The government also denies that an estimated 200 health care workers have died, contending that there have been only 12 deaths attributable to Covid-19.2

Though these situations are distinct in multiple ways and health care workers have gone on strike (or protested) for myriad reasons, common demands underlying nearly all these actions relate to inadequate responses to Covid-19 and inadequate protections for frontline workers; every group taking action has explicitly demanded more PPE.

Experts in law, ethics, and medicine have long debated whether and when strike action by health care personnel can be justified. Although these debates have centered on the risks that strikes carry for patients, these actions also pose risks for health care workers — they may damage morale and team cohesion, for example, and in many countries strikes have been repressed violently. Other risks relate to public perceptions and to potentially broader harms for both society and the health care community as a whole.3 Perhaps most fundamentally, however, strikes raise questions about what health care workers owe society and what society owes them.

#### Strikes negatively affect healthcare. Gruber & Kleiner 10

Do Strikes Kill? Evidence from New York State Jonathan Gruber and Samuel A. Kleiner NBER Working Paper No. 15855 March 2010 JEL No. I12,I23,J52,J62 <https://www.nber.org/system/files/working_papers/w15855/w15855.pdf>

We have gathered data on every hospital strike over the 1984 to 2004 period in New York State. We carefully match each striking hospital over this period with a set of control hospitals in their area, and examine the evolution of outcomes before, during, and after the strike in the striking versus control hospitals. Our results are striking: there is a meaningful increase in both hospital mortality and hospital readmission among patients admitted during a hospital strike. Our central estimates suggest that the rate of hospital mortality is 19.4% higher, and rates of hospital readmission are 6.5% higher, among those admitted during a strike than among patients in nearby hospitals at the same time. We show that this deterioration in outcomes occurs only for those patients admitted during the strike, and not for those admitted before or after to the same hospitals. And we find that these changes are not associated with any meaningful change in the composition of patients admitted during the strike or the treatment intensity for patients admitted during these strikes. We also find evidence of a more severe impact of these strikes on patients whose conditions require more intensive nursing inputs, and that outcomes are no better for patients admitted to striking hospitals who employ replacement workers. Overall, our findings suggest that strikes lead to lower quality of medical care in hospitals.

#### Poor healthcare leads to rapidly spreading of pandemics and disease and hurts economy.

Gregory HäRtl, 7-5-2018, "Low quality healthcare is increasing the burden of illness and health costs globally," WHO , <https://www.who.int/news/item/05-07-2018-low-quality-healthcare-is-increasing-the-burden-of-illness-and-health-costs-globally> (Loyola IB)

Poor quality health services are holding back progress on improving health in countries at all income levels, according to a new joint report by the OECD, World Health Organization (WHO) and the World Bank.

Today, inaccurate diagnosis, medication errors, inappropriate or unnecessary treatment, inadequate or unsafe clinical facilities or practices, or providers who lack adequate training and expertise prevail in all countries.

The situation is worst in low and middle-income countries where 10 percent of hospitalized patients can expect to acquire an infection during their stay, as compared to seven percent in high income countries. This is despite hospital acquired infections being easily avoided through better hygiene, improved infection control practices and appropriate use of antimicrobials.. At the same time, one in ten patients is harmed during medical treatment in high income countries.

These are just some of the highlights from Delivering Quality Health Services – a Global Imperative for Universal Health Coverage. The report also highlights that sickness associated with poor quality health care imposes additional expenditure on families and health systems.

There has been some progress in improving quality, for example in survival rates for cancer and cardiovascular disease. Even so, the broader economic and social costs of poor quality care, including long-term disability, impairment and lost productivity, are estimated to amount to trillions of dollars each year.

“At WHO we are committed to ensuring that people everywhere can obtain health services when and where they need them,” said WHO Director-General Dr Tedros Adhanom Ghebreyesus. “We are equally committed to ensuring that those services are good quality. Quite honestly, there can be no universal health coverage without quality care.”

“Without quality health services, universal health coverage will remain an empty promise,” said OECD Secretary-General Ángel Gurría. “The economic and social benefits are clear and we need to see a much stronger focus on investing in and improving quality to create trust in health services and give everyone access to high-quality, people-centred health services.”

“Good health is the foundation of a country’s human capital, and no country can afford low-quality or unsafe healthcare,” World Bank Group President Jim Yong Kim said. “Low-quality care disproportionately impacts the poor, which is not only morally reprehensible, it is economically unsustainable for families and entire countries.”

Other key findings in the report paint a picture of quality issues in healthcare around the world:

Health care workers in seven low- and middle-income African countries were only able to make accurate diagnoses one third to three quarters of the time, and clinical guidelines for common conditions were followed less than 45 percent of the time on average.

Research in eight high-mortality countries in the Caribbean and Africa found that effective, quality maternal and child health services are far less prevalent than suggested by just looking at access to services. For example, just 28 percent of antenatal care, 26 percent of family planning services and 21 percent of sick-child care across these countries qualified as ‘effective.’

Around 15 percent of hospital expenditure in high-income countries is due to mistakes in care or patients being infected while in hospitals.

 The three organisations outline the steps governments, health services and their workers, together with citizens and patients, urgently need to take to improve health care quality.  Governments should lead the way with strong national health care quality policies and strategies. Health systems should focus on competent care and user experience to ensure confidence in the system. Citizens should be empowered and informed to actively engage in health care decisions and in designing new models of care to meet the needs of their local communities. Health care workers should see patients as partners and commit themselves to providing and using data to demonstrate the effectiveness and safety of health care.

#### Extinction – defense is wrong

Piers Millett 17, Consultant for the World Health Organization, PhD in International Relations and Affairs, University of Bradford, Andrew Snyder-Beattie, “Existential Risk and Cost-Effective Biosecurity”, Health Security, Vol 15(4), http://online.liebertpub.com/doi/pdfplus/10.1089/hs.2017.0028

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 theWestern Abenaki (which suffered a staggering 98% loss of population).

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-

## Case