Counterfactual harm

Anonymous Author(s)
Affiliation
Address
email

Abstract

To act safely and ethically in the real world, agents must be able to reason about harm and avoid harmful actions. In this paper we develop the first statistical definition of harm and a framework for incorporating harm into algorithmic decisions. We argue that harm is fundamentally a counterfactual quantity, and show that standard machine learning algorithms that cannot perform counterfactual reasoning are guaranteed to pursue harmful policies following certain distributional shifts. To resolve this we derive a family of counterfactual objective functions that robustly mitigate for harm. We demonstrate our framework on a real-world problem of identifying optimal doses for the drug Ariprazole, using a dose-response model learned form a meta-analysis for randomized control trial data.

1 Introduction

Machine learning algorithms are increasingly used for making highly consequential decisions in areas such as medicine [21, 63, 84], criminal justice [3, 45, 90], finance [2, 9, 42], and autonomous vehicles [76]. These algorithms optimize for outcomes such as survival time, recidivism rate, and financial cost. However, these are purely factual objectives, depending only on *what* outcome occurs. On the other hand, human decision making incorporates preferences that depend not only on the outcome, but *why* it occurred.

For example, consider two treatments for a disease which, when left untreated, has a 50% mortality rate. Treatment 1 has a 60% chance of curing a patient, and a 40% chance of having no effect, with the disease progressing as if untreated. Treatment 2 has an 80% chance of curing a patient and a 20% chance of killing them. Treatments 1 and 2 have identical recovery rates, hence any agent that selects actions based solely on the factual outcome statistics (e.g. the effect of treatment on the treated [39]) will not be able to distinguish between these two treatments. However, doctors systematically favour treatment 1 as it achieves the same recovery rate but never harms the patient—there are no patients that would have survived had they not been treated. On the other hand, doctors who administer treatment 2 risk harming their patients—there are patients who die following treatment who would have lived had they not been treated. While treatments 1 and 2 have the same mortality rates, the resulting deaths have different causes (the doctor or the disease) which have different ethical implications.

Determining what would have happened to a patient if the doctor hadn't treated them is a counterfactual inference, which cannot be identified from outcome statistics alone but requires knowledge of the functional causal relation between actions and outcomes [6]. However, standard machine learning algorithms are limited to making factual inferences—e.g. learning correlations between actions and outcomes. How then can we express these ethical preferences as objective functions and ultimately incorporate them into machine learning algorithms? This question has some urgency, given that machine learning algorithms currently under development or in deployment will be unable to reason about harm and factor it into their decisions.

Submitted to 36th Conference on Neural Information Processing Systems (NeurIPS 2022). Do not distribute.

The concept of harm is foundational to many ethical codes and principles; from the Hippocratic oath [81] to environmental policy [53], and from the foundations of classical liberalism [58], to Asimov's laws of robotics [4]. Despite this ubiquity, there is no formal statistical definition of harm. We address this by translating the predominant account of harm [29, 47] into a statistical measure—*counterfactual harm*. Our definition of harm enables us for the first time to precisely answer the following basic questions,

Question 1 Did the actions of an agent cause harm, and if so, how much?

Question 2 How much harm can we expect an action to cause prior to taking it?

Question 3 How can we identify actions that balance the expected harms and benefits?

In section 2 we introduce concepts from causality, ethics, and expected utility theory used to derive our results. In section 3 we present our definitions of harm and benefit, and in section 4 we describe how harm-aversion can be factored into algorithmic decisions. In section 5 we prove that algorithms based on standard statistical learning theory are incapable of reasoning about harm and are guaranteed to pursue harmful policies following certain distributional shifts, while our approach using counterfactual objective functions overcomes these problems. Finally, in section 6 we apply our methods to an interpretable machine learning model for determining optimal drug doses, and compare this to standard algorithms that maximize treatment effects.

2 Prelimiaries

In this section we introduce some basic principles from causal modelling, expected utility theory and ethics that we use to derive our results.

2.1 Structural causal models

We provide a brief overview of structural causal models (SCMs) (see Chapter 7 of [66] and [7] for reviews). SCMs represent observable variables X^i as vertices of a directed acyclic graph (DAG), with directed edges representing causal relations between X^i and their direct causes (parents), which includes observable (endogenous) parents pa_i and a single exogenous noise variable E^i. The value of each variable is assigned by a deterministic function f_i of these parents.

Definition 1 (Structural Causal Model (SCM))**.** *A structural causal model $\mathcal{M} = \langle \mathcal{G}, E, X, F, P \rangle$ specifies:*

1. *a set of observable (endogenous) random variables $X = \{X^1, \ldots, X^N\}$,*
2. *a set of mutually independent noise (exogenous) variables $E = \{E^1, \ldots, E^N\}$ with joint distribution $P(E = e) = \prod_{i=1}^{N} P(E^i = e^i)$*
3. *a directed acyclic graph $\mathcal{G}$, whose nodes are the variables $E \cup X$ with directed edges from pa_i, E^i to X^i, where $pa_i \subseteq X$ denotes the endogenous parents of X^i.*
4. *a set of functions (mechanisms) $F = \{f_1, \ldots, f_N\}$, where the collection F forms a mapping from E to X, $x^i := f_i(pa_i, e^i)$, for $i = 1, \ldots, N$,*

By recursively applying condition 4 every observable variable can be expressed as a function of the noise variables alone, $X^i(e) = x^i$ where $E = e$ is the joint state of the noise variables, and the distribution over unobserved noise variables induces a distribution over the observable variables,

$$P(X = x) = \int_{e:X(e)=x} P(E = e) \tag{1}$$

where $X = x$ is the joint state over X.

The power of SCMs is that they specify not only the joint distribution $P(X = x)$ but also the distribution of X under all interventions, including incompatible interventions (counterfactuals). Interventions describe an external agent changing the underlying causal mechanisms F. For example, $\text{do}(X^1 = x)$ denotes intervening to force $X^1 = x$ regardless of the state of its parents, replacing $x^1 := f_1(\text{pa}_1, e^1) \rightarrow x^1 := x$. The variables in this post-intervention SCM are denoted X^i_x or $X^i_{X_1=1}$ and their interventional distribution is given by (1) under the updated mechanisms. The trivial intervention (no intervention) is denoted $\text{do}(\emptyset)$ and leaves the SCM unchanged.

These interventional distributions quantify the effects of actions and are used to inform decisions. For example, the conditional average treatment effect (CATE) [78],

$$\text{CATE}(x) = \mathbb{E}[Y_{T=1}|x] - \mathbb{E}[Y_{T=0}|x] \tag{2}$$

measures the expected value of outcome Y for treatment $T = 1$ compared to control $T = 0$ conditional on X, and is used in decision making from precision medicine to economics [1, 77].

Counterfactual distributions determine the probability that other outcomes would have occurred had some precondition been different. For example, for $X, Y \in \{T, F\}$, the causal effect $P(Y_{X=T} = T)$ is insufficient to determine if $\text{do}(X = T)$ was a necessary cause of $Y = T$ [34]. Instead this is captured by the counterfactual probability of necessity [83],

$$\text{PN} = P(Y_{X=F} = F | X = T, Y = T) \tag{3}$$

which is the probability that Y would be false if X had been false (the counterfactual proposition), given that X and Y were both true (the factual data). For example, if PN = 0 then $Y = T$ would have occured regardless of $X = T$, whereas if PN = 1 then $Y = T$ could not have occurred without $X = T$. Equation (3) involves the joint distribution over incompatible states of Y under different interventions, $Y = T$ and $Y_{X=F} = F$, which cannot be jointly measured and hence (3) cannot be identified from data alone. However, (3) can be calculated from the SCM using (1) for the combined factual and counterfactual propositions,

$$P(Y_{X=F} = F, X = T, Y = T) = \int\limits_{\substack{Y_{X=F}(e)=F \\ Y(e)=T, X(e)=T}} P(E = e) \tag{4}$$

This ability to ascribe causal explanations for data has made SCMs and counterfactual inference a key ingredient in statistical definitions of blame, intent and responsibility [33, 46, 50], and has seen applications in AI research ranging from explainability [55, 86], safety [24, 25], and fairness [48], to reinforcement learning [14, 31] and medical diagnosis [72].

2.2 The counterfactual comparative account of harm

The concept of harm is deeply embedded in ethical principles, codes and law. One famous example is the bioethical principle "first, do no harm" [80], asserting that the moral responsibility for doctors to benefit patients is superseded by their responsibility not to harm them [73]. Another example is John Stuart Mill's harm principle which forms the basis of classical liberalism [58] and inspired the "zeroth law" of Asimov's fictional robot governed society, which states that "A robot may not harm humanity or, by inaction, allow humanity to come to harm" [4].

Despite these attempts to codify harm into rules for governing human and AI behaviour, it is not immediately clear what we mean when we talk about harm. This can lead to confusion—for example, does "allow humanity to come to harm" describe an agent causing harm by inaction [13] or failing to benefit? The meaning and measure of harm also has important practical ramifications. For example, establishing negligence in tort law requires establishing that significant harm has occurred [88].

This has motivated work in philosophy, ethics and law to rigorously define harm, with the most widely accepted definition being the *counterfactual comparative account* (CCA) [28, 35, 47],

Definition 2 (CCA)**.** *The counterfactual comparative account of harm and benefit states,*

> *"An event e or action a harms (benefits) a person overall if and only if she would have been on balance better (worse) off if e had not occurred, or a had not been performed."*

Causation is known plays a key role in moral and legal reasoning [49]. Simply stated, the CCA defines harm as causing someone to be worse off [71]. The judgement of what would have happened 'had the agent had not acted' is both causal and normative: it requires a causal model to compute the counterfactual, and normative assumptions to determine what counterfactual comparison should be used. We discuss these issues further in Appendix C. The second aspect of the CCA is determining if the person would be "better (worse) off", which we will address using expected utility theory [59].

While the CCA is the predominant definition of harm and forms the basis of our results, alternative definitions have been proposed [36, 62], motivated by scenarios where the CCA appears to give

counter-intuitive results [13]. Surprisingly, we find these problematic cases are identical to those raised in the study of actual causality [34], and can be resolved with a formal causal analysis. To our knowledge, this connection with actual causality has not been noted before, and we present a detailed account of the arguments against CCA and how they can be addressed in Appendix B in the hope this will stimulate discussion between these two fields.

2.3 Decision theoretic setup

In this section we present our setup for calculating the CCA (Definition 2). The CCA describes a person (referred to as the *user*) being made better or worse off due to the actions of an agent. We focus on the simplest case involving single agents performing single actions, though ultimately our definitions could be extended to multi-agent or sequential decision problems. For causal modelling in these scenarios we refer the reader to [20, 25, 38].

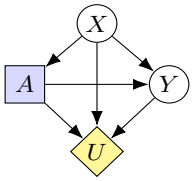


Figure 1: SCM $\mathcal{M}$ depicting causal relations between the agent's action A, context variables X, outcomes Y and the user's utility function U.

We describe the environment with a structural causal model $\mathcal{M}$ which specifies the distribution over environment states W as endogenous variables, $P(w; \mathcal{M})$. For single actions the environment variables can be divided into three disjoint sets $W = A \cup X \cup Y$ (Figure 1 a)); the agent's action A, outcome variables Y that are descendants of A and so can be influenced by the agent's action, and context variables X that are non-descendants of nor in A.

In the following, $\mathcal{M}$ denotes our SCM model for the environment (Figure 1). The user's preferences over environment states are specified by their utility function $U(w) = U(a, x, y)$, and for a given action the user's expected utility is,

$$\mathbb{E}[U|a, x] = \int_y P(y|a, x)U(a, x, y) \tag{5}$$

In sections 3-4 we focus on the case where the agent acts to maximize the user's expected utility, with optimal actions $a_{\text{max}} = \arg\max_a \mathbb{E}[U|a, x]$. While this is similar to the standard setup in expected utility theory [30, 59, 74], note that U describes the preferences of the user, and in general the agent can pursue any objective and could be entirely unaware of the user's preferences. In section 5 we extend our analysis to agents pursuing arbitrary objectives.

No factual measure can robustly identify harmful actions. Before giving a formal definition of harm, we return to our example from section 1 and show that any definition satisfying basic intuitions must be counterfactual. Treatment 1 has a 60% chance of curing the patient and 40% chance of having no effect, with the disease progressing as if untreated, giving a total recovery rate of 80%. Treatment 2 has 80% chance of curing the patient and 20% chance of killing them, likewise an 80% recovery rate. Therefore the treatments have identical factual outcome statistics $P(y|A=1) = P(y|A=2)$ where $Y=0$ indicates mortality and $Y=1$ recovery. However, treatment 2 is intuitively more harmful, as there are patients killed by the treatment who would have recovered if we hadn't treated them, whereas for treatment 1 any patients who die would have died if left untreated. Consider some factual measure of harm $H(a, P)$ which is a function of the treatment choice A and the factual outcome statistics $P = P(y = 1|A=1) = P(y = 1|A=2)$. Note there are no confounders so we can treat $P(y|a)$ and $P(y_a)$ interchangeably. To match our intuitions it must be that $H(2, P) > H(1, P)$. Consider a distributional shift where now treatment 1 has an 80% chance of curing and a 20% chance of killing, whereas treatment 2 has a 60% chance of curing and a 40% chance of having no effect. Essentially, we swap the causal mechanisms generating Y from T (see Appendix F for the SCM describing this example). Following this shift, treatment 1 is harmful and treatment 2 is not. However, P is not changed by this distributional shift. Therefore it must be that $H(2, P) > H(1, P)$ following the shift, and the harm measure incorrectly identifies treatment 1 as being less harmful than treatment 2. In section 5 we extend this to a formal proof for all decision tasks.

3 Counterfactual harm

We now present our definitions for harm and benefit based on Definition 2. For the examples described in this paper we focus on simple environments with single outcome variables, for which harm can be determined by comparing the factual utility received by the user to the utility they would have received had the agent taken some default action $A = \bar{a}$ (Definition 3). In Appendix C we describe how this default action can be determined. However, some environments studied in the harm literature involve multiple outcome variables and require a path-dependent measure of harm, which we provide in Appendix A along with examples in Appendices B-C. In the following, counterfactual states are identified with an $*$, e.g. $Z = z^*$. First we define the harm caused by an action given we observe the outcome (Question 1).

Definition 3 (Counterfactual harm & benefit)**.** *The harm caused by action $A = a$ given context $X = x$ and outcome $Y = y$ compared to the default action $A = \bar{a}$ is,*

$$h(a, x, y; \mathcal{M}) = \int_{y^*} P(Y_{\bar{a}} = y^* | a, x, y; \mathcal{M}) \max\{0, U(\bar{a}, x, y^*) - U(a, x, y)\} \tag{6}$$

and the expected benefit is

$$b(a, x, y; \mathcal{M}) = \int_{y^*} P(Y_{\bar{a}} = y^*, | a, x, y; \mathcal{M}) \max\{0, U(a, x, y) - U(\bar{a}, x, y^*)\} \tag{7}$$

where $\mathcal{M}$ is the SCM describing the environment and U is the user's utility.

The counterfactual harm (benefit) is the expected increase (decrease) in utility had the agent taken the default action $A = \bar{a}$, given they took action $A = a$ in context $X = x$ resulting in outcome $Y = y$. Taking the max in (6) and (7) ensures that we only include counterfactual outcomes where the utility is higher (lower) in the counterfactual world. Note that $X_{\bar{a}} = X$ as X is not a descendant of A, so the factual and counterfactual contexts are identical [79].

To determine the expected harm or benefit of an action prior to taking it (Question 2) we calculate the expected value of (6) and (7) over the outcome distribution, e.g. $\mathbb{E}[h|a, x; \mathcal{M}] = \int_y P(y|a, x; \mathcal{M}) h(a, x, y; \mathcal{M})$. Note these form a counterfactual decomposition of the expected utility.

Theorem 1 (harm-benefit trade-off)**.** *The difference in expected utility for action $A = a$ and the default action $A = \bar{a}$ is given by,*

$$\mathbb{E}[U|a, x] - \mathbb{E}[U|\bar{a}, x] = \mathbb{E}[b|a, x; \mathcal{M}] - \mathbb{E}[h|a, x; \mathcal{M}] \tag{8}$$

(Proof: see Appendix E).

Example 2: Returning to Example 1, the expected counterfactual harm for default action $\bar{a} = 0$ is, $\mathbb{E}[h|a; \mathcal{M}] = \sum_{y^*, y} P(Y_{\bar{a}} = y^*, y|a; \mathcal{M}) \max\{0, y^* - y\} = P(Y_0 = 1, Y_a = 0; \mathcal{M})$ where we have used $U(a, y) = y$ and the fact that $Y_{\bar{a}} \perp A$ and there are no latent confounders to give $P(Y_0 = y^*, Y = y|a) = P(Y_0 = y^*, Y_a)$. This is the probability that a patient dies following treatment $A = a$ and would have recovered had they received no treatment. In Appendix F we show that $P(Y_0 = 1, Y_1 = 0) = 0$ (treatment 1 causes zero harm) and $P(Y_0 = 1, Y_2 = 0) = 0.1$ (treatment 2 causes non-zero harm), reflecting our intuition that treatment 2 is more harmful than treatment 1, despite having the same causal effect / CATE. Hence, the counterfactual harm allows us to differentiate between these two treatments.

4 Harm in decision making

Now we have expressions for the expected harm and benefit, how can we incorporate these into the agent's decisions? Consider two actions a, a' such that $\mathbb{E}[b|a', x; \mathcal{M}] = \mathbb{E}[b|a, x; \mathcal{M}] + K$ and $\mathbb{E}[h|a', x; \mathcal{M}] = \mathbb{E}[h|a, x; \mathcal{M}] + K$ where $K \in \mathbb{R}$. By Theorem 1 they must have the same expected utility, as $\mathbb{E}[U|a', x] = \mathbb{E}[b|a', x; \mathcal{M}] - \mathbb{E}[h|a', x; \mathcal{M}] + \mathbb{E}[U|\bar{a}, x] = \mathbb{E}[b|a, x; \mathcal{M}] + K - \mathbb{E}[h|a, x; \mathcal{M}] - K + \mathbb{E}[U|\bar{a}, x] = \mathbb{E}[U|a, x]$. Therefore expected utility maximizers are indifferent to harm, and will are willing to increase harm for an equal increase in benefit.

We say that an agent is *harm averse* if they are willing to risk causing harm only if they expect a comparatively greater benefit. Theorem 1 suggests a simple way to overcome harm indifference in the expected utility by assigning a higher weight to the harm component of the decomposition (8). If instead we maximize an adjusted expected utility $\mathbb{E}[V|a, x; \mathcal{M}] := \mathbb{E}[U|x] + \mathbb{E}[b|a, x; \mathcal{M}] - (1 + \lambda)\mathbb{E}[h|a, x; \mathcal{M}]$ where $\lambda \in \mathbb{R}$, then if $\mathbb{E}[V|a, x; \mathcal{M}] = \mathbb{E}[V|a', x; \mathcal{M}]$ and $\mathbb{E}[h|a', x; \mathcal{M}] = \mathbb{E}[h|a, x; \mathcal{M}] + K$ then $\mathbb{E}[b|a', x; \mathcal{M}] = \mathbb{E}[b|a, x; \mathcal{M}] + (1 + \lambda)K$, and the agent is willing to trade harm for benefit at a $1 : (1 + \lambda)$ ratio. Taking inspiration from risk aversion [40], we refer to λ as the agent's *harm aversion*. To achieve the desired trade-off we replace the utility function with the harm-penalized utility (HPU),

Definition 4 (Harm penalized utility (HPU))**.** *For utility U and an environment described by SCM $\mathcal{M}$, the harm-penalized utility (HPU) is,*

$$V(a, x, y; \mathcal{M}) = U(a, x, y) - \lambda h(a, x, y; \mathcal{M}) \tag{9}$$

where $h(a, x, y; \mathcal{M})$ is the harm (Definition 3) and λ is the harm aversion.

For $\lambda = 0$ the agent is *harm indifferent* and optimizes the standard expected utility. For $\lambda > 0$ the agent is *harm averse*, and is willing to reduce the expected utility to achieve a greater reduction to the expected harm. For $\lambda < 0$ the agent is *harm seeking*, and is willing to reduce the expected utility in order to increase the expected harm.

Maximizing the expected HPU allows agents to choose actions that balance the expected benefits and harms (Quesiton 2 section 1). We refer to the expected HPU (9) as a *counterfactual objective function* as $V(a, x, y; \mathcal{M})$ is a counterfactual expectation that cannot be evaluated without knowledge of $\mathcal{M}$. We refer to the expected utility as a *factual objective function*, as it can be evaluated on data alone without knowledge of $\mathcal{M}$. As we show in the following example, harm-aversion can lead to very different behaviours compared to other factual approaches to safety such as risk aversion [40].

Example 3: assistant paradox. Alice invests $\$80$ in the stock market, and expects a normally distributed return $Y \sim \mathcal{N}(\mu, \sigma^2)$ with $\mu = \sigma = \$100$. She asks her new AI assistant to manage her investment, and after a lengthy analysis the AI identifies three possible actions;

1. Multiply the investment by $0 \leq K \leq 20$, resulting in a return $Y \to K\,Y$,
2. Perform a clever trade that Alice was unaware of, increasing her return by $+\$10$ with certainty, $Y \to Y + 10$,
3. Cancel Alice's investment, returning $\$80$.

Consider 3 agents; Agent 1 maximizes expected return, choosing $a = \arg\max_a \mathbb{E}[Y|a]$, Agent 2 is risk-averse, in the sense of [40], choosing $a = \arg\max_a \{\mathbb{E}[Y|a] - \lambda \text{Var}[Y|a]\}$,Agent 3 is harm-averse, choosing $a = \arg\max_a \{\mathbb{E}[Y|a] - \lambda \mathbb{E}[h|a; \mathcal{M}]\}$.

In Appendix G we derive the optimal policies for Agents 1-3, and calculate the harm with respect to the default action where the agent leaves Alice's investment unaltered (i.e. action 1, $K = 1$). Agent 1 chooses action 1 and $K = 20$, maximising Alice's return but also maximizing the expected harm—e.g. if Alice would have lost $\$100$ she will now lose $\$2000$. The standard approach in portfolio theory [56] is to use agent 2 with an appropriate risk aversion λ. However, agent 2 never chooses action 2 for any λ, despite action 2 always increasing Alice's return (causing zero harm and non-zero benefit). Agent 2 will reduce or even cancel Alice's investment (for $\lambda > 0.0032$) rather than take action 2. This is because the risk-averse objective is factual, and cannot differentiate between losses caused by the agent's actions from those that would have occurred regardless of the agent's actions. Consequently, agent 2 treats all outcomes as being caused by its actions, and is willing to harm Alice by reducing or even cancelling her investment in order to minimize her risk. On the other hand agent 3 never reduces Alice's expected return, choosing either action 1 or action 2 depending on the degree of harm aversion.

The surprising behaviour of agent 2 highlights that factual objectives (e.g. risk aversion) cannot capture preferences that depend not only on the outcome but on its cause. For example, Alice may assign a different weight to losses that are due to her assistant than due to exogenous market fluctuations or her own actions—e.g. if her bet would have made money but is overridden by her assistant. Consequently she may prefer action 2 over action 1 or action 3 with $K < 1$. While this can be achieved using a factual objective function that favours action 2 a priori (assuming Alice is aware of this action), next we show this approach fails to generalize and actually leads to harmful policies.

5 Counterfactual reasoning is necessary for harm aversion

In the previous sections we saw examples where expected utility maximizers harm the user. However, these examples describe specific environments and utility functions, and assume the agent maximizes the expected utility instead of some other (potentially safer) objective. In this section we consider users with arbitrary utility functions and agents with arbitrary objectives to answer; when is maximizing the expected utility harmful? And are there other factual objective functions that can overcome this?

Factual objective functions can be expressed as the expected value of a real-valued function $J(a, x, y)$. Examples include the expected utility, cost functions [10], and the cumulative discounted reward [82]. Once J is specified the optimal actions in environment $\mathcal{M}$ are given by,

$$a_{\text{max}} = \arg\max_a \mathbb{E}[J|a, x] = \arg\max_a \int_y P(y|a, x; \mathcal{M}) J(a, x, y) \tag{10}$$

Note that (10) can be evaluated from data alone (i.e. from samples of the joint distribution of A, X and Y) without knowledge of $\mathcal{M}$. In the following we relax our requirement that agents have some fixed harm aversion λ, requiring only that they don't take needlessly harmful actions.

Definition 5 (Harmful actions & policies)**.** *An action $A = a$ is needlessly harmful in context $X = x$ and environment $\mathcal{M}$ if there is $a' \neq a$ such that $\mathbb{E}[U|a', x] \geq E[U|a, x]$ and $\mathbb{E}[h|a', x; \mathcal{M}] < \mathbb{E}[h|a, x; \mathcal{M}]$. A policy is harmful if it assigns a non-zero probability to a needlessly harmful action.*

Definition 6 (Harmful objectives)**.** *An objective $\mathbb{E}[J|a, x]$ is harmful in environment $\mathcal{M}$ if there is a policy that maximizes the expected value of J and is harmful.*

Note that needlessly harmful actions violate any degree of harm aversion $\lambda > 0$, as the agent is willing to choose actions that are strictly more harmful for no additional benefit. In examples 1 and 2, treatment 2 is a needlessly harmful action and the CATE is a harmful objective. First, we show that maximizing the expected HPU is never a harmful objective in any environment,

Theorem 2. *For any utility functions U, environment $\mathcal{M}$ and default action $A = \bar{a}$ the expected HPU is never a harmful objective for $\lambda > 0$.* (Proof: see Appendix J).

It is vital that agents continue to pursue safe objectives following changes to the environment (distributional shifts). At the very least, agents should be able to re-train in the shifted environment without needing to tweak their objective functions. In SCMs distributional shifts are represented as interventions that change the exogenous noise distribution and/or the underlying causal mechanisms [69, 75]. We focus on distributional shifts that change the outcome distribution $P(y|a, x; \mathcal{M})$ alone.

Definition 7 (Outcome distributional shift)**.** *For an environment described by SCM $\mathcal{M}$, $\mathcal{M} \to \mathcal{M}'$ is a shift in the outcome distribution if the exogenous noise distribution for Y changes $P(e^Y; \mathcal{M}') \neq P(e^Y; \mathcal{M})$ and/or the causal mechanism changes $f'_Y(a, x, e^Y) \neq f_Y(a, x, e^Y)$.*

By Theorem 2 the HPU is never harmful following any distributional shift. On the other hand, we find that maximizing almost any factual utility function will result in harmful policies under distributional shifts. In the following we assume some degree of outcome dependence in the user's utility function.

Definition 8 (Outcome dependence)**.** *$U(a, x, y)$ is outcome dependent for a set of actions $C = \{a_1, \ldots, a_N\}$ in context $X = x$ if $\forall\, a_i, a_j \in C$, $i \neq j$, $\max_y U(a_i, x, y) > \min_y U(a_j, x, y)$.*

If there is no outcome dependence then the optimal action is independent of the outcome distribution $P(y|a, x)$, and no learning is required to determine the optimal policy. Typically we are interested in tasks that require some degree of learning and hence outcome dependence.

Theorem 3. *If for any context $X = x$ the user's utility function is outcome dependent for the default action in that context $\bar{a}(x)$ and another action $a \neq \bar{a}(x)$, then there is an outcome distributional shift such that U is harmful in the shifted environment.* (Proof: see Appendix J).

To understand the implications of Theorem 3 we can return to examples 1 & 2 and no longer assume a specific utility function or outcome statistics. Theorem 3 implies that for any agent maximizing the user's utility, there is always a a distributional shift such that the agent will cause unnecessary harm to patient in the shifted environment, compared to not treating them ($\bar{a} = 0$) or any other default action. Unlike in examples 1 & 2 we allow the patient's utility function to depend on the treatment choice, and for treatments to vary in effectiveness. The only assumption we make is that whatever the

user's utility function is, it is not true that $U(A = a, x, Y = 0) > U(A = 0, x, Y = 1)\ \forall\ a, x$. If this were true then the patient's preferences are dominated by the agents action choice alone, i.e. the user would always prefer to die so long as they are treated, rather than not being treated and surviving.

While robust harm aversion is not possible for expected utility maximizers in general, it seems likely at first that we can train robustly harm-averse agents using other learning objectives. One possibility would be to use human interactions and demonstrations [16, 51, 60] that *implicitly* encode harm aversion, with $J(a, x, y)$ representing user feedback or some learned (factual) reward model. In examples 1 & 2 users with utility $U(a, x, y) = y$ could assign a higher cost to deaths following treatment 2 compared to treatment 1, $J(A = 2, Y = 0) < J(A = 1, Y = 0)$, with J implicitly encoding the harm caused by treatment 2. This would allow robust harm-averse agents to be trained using standard statistical learning techniques, without needing to learn $\mathcal{M}$ or perform counterfactual inference. However, we now show this is not possible in general.

Theorem 4. *If for any context $X = x$ the user's utility function is outcome dependent for the default action in that context $\bar{a}(x)$ and two other actions $a_1, a_2 \neq \bar{a}(x)$, then for any factual objective function J there is an outcome distributional shift such that J is harmful in the shifted environment.* (Proof: see Appendix J).

Theorem 4 implies that agents optimizing factual objective functions may appear safe in their training environments but are guaranteed to pursue harmful policies following certain distributional shifts, even if they are allowed to re-train. This is true regardless of the (factual) objective function of the agent, and applies to any user whose utility function has some basic degree of outcome dependence. This is particularly concerning as all standard machine learning algorithms use factual objective functions. In section 6 we give a concrete example of a reasonable factual objective function and a distributional shift that renders it harmful.

Example 4: An AI assistant manages Alice's health including medical treatments, clinical testing and lifestyle interventions. These actions have a causal effect on Alice's health outcomes—e.g. disease progression, severity of symptoms, and medical costs, and Alice's preferences depend on these outcomes and not just on the action chosen by the agent (outcome dependence). The agent maximizes a reward function over these health states and actions (factual objective), including feedback from Alice and clinicians in-the-loop which penalize harmful actions (implicit harm aversion) by comparing to standardised treatment rules (default actions). The agent appears to be safe and is deployed at scale. However, by Theorem 4 there in principle exist distributionally shifted environments such that the agent chooses actions that needlessly harm patients in spite of retraining.

6 Illustration: Dose-response models

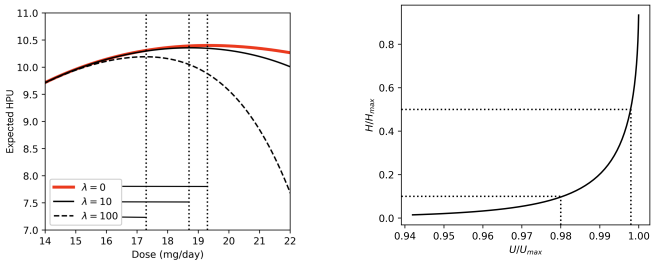
How does harm aversion affect behaviour and performance in real-world tasks? And how can reasonable objective functions result in harmful policies following distributional shifts? In this section we apply the methods developed in section 4 to the task of determining optimal treatments. This problem has received much attention in recent years following advances in causal machine learning techniques [89] and growing commercial interests including recommendation [52], personalized medicine [11], epidemiology and econometrics [41] to name a few.

While harm is important for decision making in many of these areas, existing approaches reduce the problem to identifying treatment effects—e.g. maximizing the CATE (2)—which is indifferent to harm as shown in sections 3 and 5. This could result in needlessly harmful decisions being made, perhaps overlooking equally effective decisions that are far less harmful.

To demonstrate this we use a model for determining the effectiveness of the drug Aripiprazole learned from a meta-analysis of randomised control trial data [18]. This dose-response model predicts the reduction in symptom severity Y following treatment with a dose A (mg/day) of Aripiprazole using a generalized additive model (GAM) [37]—an interpretable machine learning model commonly used for dose-response analysis [22],

$$y = f(a, \theta_1, \theta_2, \varepsilon) = \theta_1 a + \theta_2 f(a) + \varepsilon \tag{11}$$

where $\theta_i \sim \mathcal{N}(\hat{\theta}_i, V_i)$ are random effects parameters, $\varepsilon \sim \mathcal{N}(0, V_\varepsilon)$ is a noise parameter, and $f(a)$ is a cubic spline function (for details and parameters see Appendix I). We assume $U(a, y) = y$ (patients have no intrinsic preference for different doses) and measure harm in comparison to the zero dose



(a) (b)

Figure 2: a) Expected HPU (Def 4) for the dose-response model (11) v.s. dosage of for $\lambda = 0$, $\lambda = 10$ and 100. Dotted lines show optimal doses. b) $\mathbb{E}[h|a]/\mathbb{E}[h|a_{\text{max}}]$ v.s. $\mathbb{E}[U|a]/\mathbb{E}[U|a_{\text{max}}]$ for $a \leq a_{\text{max}}$. Dotted lines show doses reducing harm by 50% and 90% and utility by 0.2% and 2.0% respectively.

$\bar{a} = 0$. In Appendix I we derive a closed-form expression for the expected harm in GAMs. We calculate the expected HPU (4) for $\lambda = 0, 10, 100$ (Figure 2 a)). In this context a relatively large λ is appropriate to ensure a high ratio of expected benefit to harm required for medical interventions [27]. We find that the optimal dose is highly sensitive to λ, for example reducing from 19.3 mg/day to 17.3 mg/day for $\lambda = 100$. On the other hand, these lower-harm doses achieve a similar improvement in symptom severity compared to the optimal dose. Figure 2 b) shows the trade-off between expected utility and harm relative to the optimal dosage $a_{\text{CATE}} = \arg\max_a \mathbb{E}[Y_a]$. For almost-optimal doses we observe a steep harm gradient, with small improvements in symptom severity requiring large increases in harm. For example, choosing a lower dose than a_{CATE} one can reduce harm by 90% while only reducing the treatment effect by 2%. This demonstrates that ignoring harm while maximizing expected utility will often result in extreme harm values (an example of Goodhart's law [91]).

Finally, we consider identifying safe doses using a reasonable factual objective function (risk-aversion) and describe how this can result in harm following distributional shifts. Consider the risk-averse objective $\mathbb{E}[J|a] = \mathbb{E}[Y_a] - \beta \mathsf{Var}[Y_a]$. As $\mathsf{Var}[Y_a]$ increases with dose, this objective also selects lower harm doses in our example. However, consider a distributional shift where (11) gains an additional noise term, $y = f(a, \theta_1, \theta_2, \varepsilon, \eta) = \theta_1 a + \theta_2 f(a) + \varepsilon + \eta^Y(10 - 0.5a)$ where $\eta^Y \sim \mathcal{N}(0, 1)$, e.g. describing a shift where untreated patients ($A = 0$) have a high variation in outcome Y which decreases as the dose increases. It is simple to check that, for $a = \arg\max_a \mathbb{E}[U|a]$ and $a' = \arg\max_a \mathbb{E}[J|a]$, $E[U|a] > E[U|a']$ and $E[h|a] < E[h|a'] \; \forall \, \beta > 0$ in this shifted environment. Hence all risk averse agents are needlessly harmful in the shifted environment (Definition 6).

7 Conclusion

One of the core assumptions of statistical learning theory is that human preferences can be expressed solely in terms of states of the world, while the underlying dynamical (i.e. causal) relations between these states can be ignored. This assumption allows goals to be expressed as factual objective functions and optimized on data without needing to learn causal models or perform counterfactual inference. According to this view, all behaviours we care about can be achieved using the first two levels of Pearl's hierarchy [68]. In this article we have argued against this view. We proposed the first statistical definition of harm, and showed that agents optimizing factual objective functions are incapable of avoiding harmful actions in general. Arguably the ability to avoid causing harm is a key requirement for deploying safe and ethical AI [12], and our results add weight to arguments that advanced AI systems will be severely limited if they are unable to perform causal and counterfactual reasoning [67]. In Appendix K we discuss some related works that propose solutions to ethical and safety problems using counterfactual inference, namely counterfactual fairness [48] and path-specific objectives [26]. While our theoretical results have been derived in the framework of structural causal models for the sake of generality, there is a growing body of work developing deep learning models that are capable of supporting counterfactual reasoning in complex domains including medical imaging [65], visual question answering [61], and vision-and-language navigation in robotics [64] and text generation [54], and we discuss these in Appendix K.

References

[1] Jason Abrevaya, Yu-Chin Hsu, and Robert P Lieli. Estimating conditional average treatment effects. *Journal of Business & Economic Statistics*, 33(4):485–505, 2015.

[2] Peter Martey Addo, Dominique Guegan, and Bertrand Hassani. Credit risk analysis using machine and deep learning models. *Risks*, 6(2):38, 2018.

[3] Julia Angwin, Jeff Larson, Surya Mattu, and Lauren Kirchner. Machine bias. *ProPublica, May*, 23(2016):139–159, 2016.

[4] Isaac Asimov. *I, robot*, volume 1. Spectra, 2004.

[5] Chen Avin, Ilya Shpitser, and Judea Pearl. Identifiability of path-specific effects. 2005.

[6] Alexander Balke and Judea Pearl. Counterfactual probabilities: Computational methods, bounds and applications. In *Uncertainty Proceedings 1994*, pages 46–54. Elsevier, 1994.

[7] Elias Bareinboim, Juan D Correa, Duligur Ibeling, and Thomas Icard. On pearl's hierarchy and the foundations of causal inference. *ACM Special Volume in Honor of Judea Pearl (provisional title)*, 2:4, 2020.

[8] Sander Beckers, Hana Chockler, and Joseph Halpern. An analysis of harm using causal models and default utilities. *(forthcoming)*, 2022.

[9] Daniel Belanche, Luis V Casaló, and Carlos Flavián. Artificial intelligence in fintech: understanding robo-advisors adoption among customers. *Industrial Management & Data Systems*, 2019.

[10] James O Berger. *Statistical decision theory and Bayesian analysis*. Springer Science & Business Media, 2013.

[11] Ioana Bica, Ahmed M Alaa, Craig Lambert, and Mihaela Van Der Schaar. From real-world patient data to individualized treatment effects using machine learning: Current and future methods to address underlying challenges. *Clinical Pharmacology & Therapeutics*, 109(1):87–100, 2021.

[12] Nick Bostrom and Eliezer Yudkowsky. The ethics of artificial intelligence. *The Cambridge handbook of artificial intelligence*, 1:316–334, 2014.

[13] Ben Bradley. Doing away with harm 1. *Philosophy and Phenomenological Research*, 85(2):390–412, 2012.

[14] Lars Buesing, Theophane Weber, Yori Zwols, Sebastien Racaniere, Arthur Guez, Jean-Baptiste Lespiau, and Nicolas Heess. Woulda, coulda, shoulda: Counterfactually-guided policy search. *arXiv preprint arXiv:1811.06272*, 2018.

[15] Erik Carlson, Jens Johansson, and Olle Risberg. Causal accounts of harming. *Pacific Philosophical Quarterly*, 2021.

[16] Paul Christiano, Jan Leike, Tom B Brown, Miljan Martic, Shane Legg, and Dario Amodei. Deep reinforcement learning from human preferences. *arXiv preprint arXiv:1706.03741*, 2017.

[17] Juan Correa and Elias Bareinboim. A calculus for stochastic interventions: Causal effect identification and surrogate experiments. In *Proceedings of the AAAI Conference on Artificial Intelligence*, volume 34, pages 10093–10100, 2020.

[18] Alessio Crippa and Nicola Orsini. Dose-response meta-analysis of differences in means. *BMC medical research methodology*, 16(1):1–10, 2016.

[19] Andrew J Cutler, Ronald N Marcus, Sterling A Hardy, Amy O'Donnell, William H Carson, and Robert D McQuade. The efficacy and safety of lower doses of aripiprazole for the treatment of patients with acute exacerbation of schizophrenia. *CNS spectrums*, 11(9):691–702, 2006.

[20] A Philip Dawid. Influence diagrams for causal modelling and inference. *International Statistical Review*, 70(2):161–189, 2002.

[21] Jeffrey De Fauw, Joseph R Ledsam, Bernardino Romera-Paredes, Stanislav Nikolov, Nenad Tomasev, Sam Blackwell, Harry Askham, Xavier Glorot, Brendan O'Donoghue, Daniel Visentin, et al. Clinically applicable deep learning for diagnosis and referral in retinal disease. *Nature medicine*, 24(9):1342–1350, 2018.

[22] Loic Desquilbet and François Mariotti. Dose-response analyses using restricted cubic spline functions in public health research. *Statistics in medicine*, 29(9):1037–1057, 2010.

[23] Kai Epstude and Neal J Roese. The functional theory of counterfactual thinking. *Personality and social psychology review*, 12(2):168–192, 2008.

[24] Tom Everitt, Marcus Hutter, Ramana Kumar, and Victoria Krakovna. Reward tampering problems and solutions in reinforcement learning: A causal influence diagram perspective. *Synthese*, pages 1–33, 2021.

[25] Tom Everitt, Ramana Kumar, Victoria Krakovna, and Shane Legg. Modeling agi safety frameworks with causal influence diagrams. *arXiv preprint arXiv:1906.08663*, 2019.

[26] Sebastian Farquhar, Ryan Carey, and Tom Everitt. Path-specific objectives for safer agent incentives. *arXiv preprint arXiv:2204.10018*, 2022.

[27] FDA. Benefit-risk assessment in drug regulatory decision-making. *Draft PDUFA VI Implementation Plan (FY 2018–2022)< https://www. fda.gov/downloads/For Industry/ UserFees/ PrescriptionDrugUserFee/ UCM60*, 2885, 2018.

[28] Joel Feinberg. Wrongful life and the counterfactual element in harming. *Social Philosophy and Policy*, 4(1):145–178, 1986.

[29] Joel Feinberg. *Harm to others*, volume 1. Oxford University Press on Demand, 1987.

[30] Peter C Fishburn. *The foundations of expected utility*, volume 31. Springer Science & Business Media, 2013.

[31] Andrew Forney, Judea Pearl, and Elias Bareinboim. Counterfactual data-fusion for online reinforcement learners. In *International Conference on Machine Learning*, pages 1156–1164. PMLR, 2017.

[32] Tobias Gerstenberg, Noah D Goodman, David A Lagnado, and Joshua B Tenenbaum. A counterfactual simulation model of causal judgments for physical events. *Psychological Review*, 128(5):936, 2021.

[33] Joseph Halpern and Max Kleiman-Weiner. Towards formal definitions of blameworthiness, intention, and moral responsibility. In *Proceedings of the AAAI Conference on Artificial Intelligence*, volume 32, 2018.

[34] Joseph Y Halpern. *Actual causality*. MiT Press, 2016.

[35] Matthew Hanser. The metaphysics of harm. *Philosophy and Phenomenological Research*, 77(2):421–450, 2008.

[36] Elizabeth Harman. Harming as causing harm. In *Harming future persons*, pages 137–154. Springer, 2009.

[37] Trevor J Hastie and Robert J Tibshirani. *Generalized additive models*. Routledge, 2017.

[38] David Heckerman and Ross Shachter. A decision-based view of causality. In *Uncertainty Proceedings 1994*, pages 302–310. Elsevier, 1994.

[39] James J Heckman. Randomization and social policy evaluation. *Evaluating welfare and training programs*, 1:201–30, 1992.

[40] Ronald A Howard and James E Matheson. Risk-sensitive markov decision processes. *Management science*, 18(7):356–369, 1972.

[41] Guido W Imbens and Donald B Rubin. *Causal inference in statistics, social, and biomedical sciences*. Cambridge University Press, 2015.

[42] Kristin Johnson, Frank Pasquale, and Jennifer Chapman. Artificial intelligence, machine learning, and bias in finance: toward responsible innovation. *Fordham L. Rev.*, 88:499, 2019.

[43] John M Kane, William H Carson, Anutosh R Saha, Robert D McQuade, Gary G Ingenito, Dan L Zimbroff, and Mirza W Ali. Efficacy and safety of aripiprazole and haloperidol versus placebo in patients with schizophrenia and schizoaffective disorder. *Journal of Clinical Psychiatry*, 63(9):763–771, 2002.

[44] S Kay, Lewis A Opler, Abraham Fiszbein, PM Ramirez, MGA Opler, and L White. Positive and negative syndrome scale. *GROUP*, 1:4, 1992.

[45] Danielle Leah Kehl and Samuel Ari Kessler. Algorithms in the criminal justice system: Assessing the use of risk assessments in sentencing. *Responsive Communities Initiative, Berkman Klein Center for Internet & Society, Harvard Law School*, 2017.

[46] Max Kleiman-Weiner, Tobias Gerstenberg, Sydney Levine, and Joshua B Tenenbaum. Inference of intention and permissibility in moral decision making. In *CogSci*, 2015.

[47] Justin Klocksiem. A defense of the counterfactual comparative account of harm. *American Philosophical Quarterly*, 49(4):285–300, 2012.

[48] Matt J Kusner, Joshua Loftus, Chris Russell, and Ricardo Silva. Counterfactual fairness. In I. Guyon, U. V. Luxburg, S. Bengio, H. Wallach, R. Fergus, S. Vishwanathan, and R. Garnett, editors, *Advances in Neural Information Processing Systems*, volume 30. Curran Associates, Inc., 2017.

[49] David A Lagnado and Tobias Gerstenberg. Causation in legal and moral reasoning. *Oxford handbook of causal reasoning*, pages 565–602, 2017.

[50] David A Lagnado, Tobias Gerstenberg, and Ro'i Zultan. Causal responsibility and counterfactuals. *Cognitive science*, 37(6):1036–1073, 2013.

[51] Jan Leike, David Krueger, Tom Everitt, Miljan Martic, Vishal Maini, and Shane Legg. Scalable agent alignment via reward modeling: a research direction. *arXiv preprint arXiv:1811.07871*, 2018.

[52] Dawen Liang, Laurent Charlin, and David M Blei. Causal inference for recommendation. In *Causation: Foundation to Application, Workshop at UAI. AUAI*, 2016.

[53] Albert C Lin. The unifying role of harm in environmental law, 2006 wis. *L. Rev*, 897:904, 2006.

[54] Nishtha Madaan, Inkit Padhi, Naveen Panwar, and Diptikalyan Saha. Generate your counterfactuals: Towards controlled counterfactual generation for text. In *Proceedings of the AAAI Conference on Artificial Intelligence*, volume 35, pages 13516–13524, 2021.

[55] Prashan Madumal, Tim Miller, Liz Sonenberg, and Frank Vetere. Explainable reinforcement learning through a causal lens. In *Proceedings of the AAAI Conference on Artificial Intelligence*, volume 34, pages 2493–2500, 2020.

[56] Harry M Markowitz. *Portfolio selection*. Yale university press, 1968.

[57] Joseph P McEvoy, David G Daniel, William H Carson Jr, Robert D McQuade, and Ronald N Marcus. A randomized, double-blind, placebo-controlled, study of the efficacy and safety of aripiprazole 10, 15 or 20 mg/day for the treatment of patients with acute exacerbations of schizophrenia. *Journal of psychiatric research*, 41(11):895–905, 2007.

[58] John Stuart Mill. *On liberty and the subjection of women*. Penguin UK, 2006.

[59] Oskar Morgenstern and John Von Neumann. *Theory of games and economic behavior*. Princeton university press, 1953.

[60] Ashvin Nair, Bob McGrew, Marcin Andrychowicz, Wojciech Zaremba, and Pieter Abbeel. Overcoming exploration in reinforcement learning with demonstrations. In *2018 IEEE International Conference on Robotics and Automation (ICRA)*, pages 6292–6299. IEEE, 2018.

[61] Yulei Niu, Kaihua Tang, Hanwang Zhang, Zhiwu Lu, Xian-Sheng Hua, and Ji-Rong Wen. Counterfactual vqa: A cause-effect look at language bias. In *Proceedings of the IEEE/CVF Conference on Computer Vision and Pattern Recognition*, pages 12700–12710, 2021.

[62] Alastair Norcross. Harming in context. *Philosophical Studies*, 123(1-2):149–173, 2005.

[63] Ziad Obermeyer, Brian Powers, Christine Vogeli, and Sendhil Mullainathan. Dissecting racial bias in an algorithm used to manage the health of populations. *Science*, 366(6464):447–453, 2019.

[64] Amin Parvaneh, Ehsan Abbasnejad, Damien Teney, Javen Qinfeng Shi, and Anton van den Hengel. Counterfactual vision-and-language navigation: Unravelling the unseen. *Advances in Neural Information Processing Systems*, 33:5296–5307, 2020.

[65] Nick Pawlowski, Daniel Coelho de Castro, and Ben Glocker. Deep structural causal models for tractable counterfactual inference. *Advances in Neural Information Processing Systems*, 33:857–869, 2020.

[66] Judea Pearl. *Causality*. Cambridge university press, 2009.

[67] Judea Pearl. Theoretical impediments to machine learning with seven sparks from the causal revolution. *arXiv preprint arXiv:1801.04016*, 2018.

[68] Judea Pearl. The seven tools of causal inference, with reflections on machine learning. *Communications of the ACM*, 62(3):54–60, 2019.

[69] Jonas Peters, Dominik Janzing, and Bernhard Schölkopf. *Elements of causal inference: foundations and learning algorithms*. The MIT Press, 2017.

[70] Steven G Potkin, Anutosh R Saha, Mary J Kujawa, William H Carson, Mirza Ali, Elyse Stock, Joseph Stringfellow, Gary Ingenito, and Stephen R Marder. Aripiprazole, an antipsychotic with a novel mechanism of action, and risperidone vs placebo in patients with schizophrenia and schizoaffective disorder. *Archives of general psychiatry*, 60(7):681–690, 2003.

[71] Duncan Purves. Harming as making worse off. *Philosophical Studies*, 176(10):2629–2656, 2019.

[72] Jonathan G Richens, Ciarán M Lee, and Saurabh Johri. Improving the accuracy of medical diagnosis with causal machine learning. *Nature communications*, 11(1):1–9, 2020.

[73] W David Ross. *Foundations of ethics*. Read Books Ltd, 2011.

[74] Leonard J Savage. *The foundations of statistics*. Courier Corporation, 1972.

[75] Bernhard Schölkopf, Francesco Locatello, Stefan Bauer, Nan Rosemary Ke, Nal Kalchbrenner, Anirudh Goyal, and Yoshua Bengio. Toward causal representation learning. *Proceedings of the IEEE*, 109(5):612–634, 2021.

[76] Wilko Schwarting, Javier Alonso-Mora, and Daniela Rus. Planning and decision-making for autonomous vehicles. *Annual Review of Control, Robotics, and Autonomous Systems*, 1:187–210, 2018.

[77] Uri Shalit, Fredrik D Johansson, and David Sontag. Estimating individual treatment effect: generalization bounds and algorithms. In *International Conference on Machine Learning*, pages 3076–3085. PMLR, 2017.

[78] Ilya Shpitser and Judea Pearl. Identification of conditional interventional distributions. *arXiv preprint arXiv:1206.6876*, 2012.

[79] Ilya Shpitser and Judea Pearl. What counterfactuals can be tested. *arXiv preprint arXiv:1206.5294*, 2012.

[80] Cedric M Smith. Origin and uses of primum non nocere—above all, do no harm! *The Journal of Clinical Pharmacology*, 45(4):371–377, 2005.

[81] Daniel K Sokol. "first do no harm" revisited. *Bmj*, 347, 2013.

[82] Richard S Sutton and Andrew G Barto. *Reinforcement learning: An introduction*. MIT press, 2018.

[83] Jin Tian and Judea Pearl. Probabilities of causation: Bounds and identification. *Annals of Mathematics and Artificial Intelligence*, 28(1):287–313, 2000.

[84] Eric J Topol. High-performance medicine: the convergence of human and artificial intelligence. *Nature medicine*, 25(1):44–56, 2019.

[85] Erick H Turner, Daniel Knoepflmacher, and Lee Shapley. Publication bias in antipsychotic trials: an analysis of efficacy comparing the published literature to the us food and drug administration database. *PLoS medicine*, 9(3):e1001189, 2012.

[86] Sandra Wachter, Brent Mittelstadt, and Chris Russell. Counterfactual explanations without opening the black box: Automated decisions and the gdpr. *Harv. JL & Tech.*, 31:841, 2017.

[87] Michael R Waldmann and Jörn H Dieterich. Throwing a bomb on a person versus throwing a person on a bomb: Intervention myopia in moral intuitions. *Psychological science*, 18(3):247–253, 2007.

[88] Richard W Wright. Causation in tort law. *Calif. L. Rev.*, 73:1735, 1985.

[89] Liuyi Yao, Sheng Li, Yaliang Li, Mengdi Huai, Jing Gao, and Aidong Zhang. Representation learning for treatment effect estimation from observational data. *Advances in Neural Information Processing Systems*, 31, 2018.

[90] Aleš Završnik. Algorithmic justice: Algorithms and big data in criminal justice settings. *European Journal of Criminology*, 18(5):623–642, 2021.

[91] Simon Zhuang and Dylan Hadfield-Menell. Consequences of misaligned ai. *arXiv preprint arXiv:2102.03896*, 2021.