

## Causal Capacities, Causal Laws and Probabilistic Causality\*

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Cartwright claims that the three-place theory of probabilistic causation does not represent laws concerning causal capacities. So the theory of probabilistic causation is not metaphysically informative but merely a statistically methodological theory. I argue that the theory of probabilistic causation is a metaphysical theory of causation concerning causal laws, and provides information about causal capacities.

【key words】 Cartwright, causal capacities, causal laws, ceteris paribus condition, probabilistic causal relations, the theory of probabilistic causation

### 1. Introduction

Cartwright (1988, 1989, 1995) poses a problem for the theory of probabilistic causation articulated by herself (1983), Skyrms (1980) and Eells (1983, 1991). Cartwright claims that the theory of probabilistic causation cannot tell us anything about *singular causes* carrying *causal capacities*, and does not represent *laws* concerning *causal capacities*. So the theory of probabilistic causation is not a metaphysical theory about laws representing causal capacities but merely a methodological theory or principle. In this paper, I shall argue that, contrary to Cartwright's argument, the theory of probabilistic causation is a metaphysical theory,

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and provides information about causal capacities.

I shall first briefly introduce the theory of probabilistic causation called "the three-place theory of probabilistic causation": a theory relating a causal factor  $X$ , an effect factor  $Y$  and a population  $P$  within which  $X$  is some kind cause of  $Y$ . Second, I shall introduce Cartwright's argument against the three place theory of probabilistic causation: three-place probabilistic causal relations do not represent *laws* concerning *causal capacities*, and is not metaphysically informative. Third, I shall argue that three-place probabilistic causal relations are causally lawful relations, so that the theory of probabilistic causation is a metaphysical theory. Fourth, I shall argue that the modality (causal tendency or potentiality) three-place probabilistic causal relations take on represents what causal capacities are intended to represent.

## 2. Three-Place Theory of Probabilistic Causation

The basic model of probabilistic causation represents causal significance of a factor  $X$  for a factor  $Y$  in terms of a two-place relation between a factor  $X$  and a factor  $Y$ , i.e.,  $Pr(Y/X) > Pr(Y/-X)$ . This two-place model of probabilistic causation is too simple to represent the relation of causal relevance between  $X$  and  $Y$  rigorously. Consider a causal statement, Smoking raises the probability of getting lung cancer. Suppose that there are Martians who have a physiologically different structure from Earthians (i.e., human beings). Relative to the population of Martians, smoking may have no positive causal significance for lung cancer, whereas, relative to the population of Earthians, smoking has positive causal significance for lung cancer. Thus, the relation of causal relevance between  $X$  and  $Y$  depends on which population the relation of causal relevance between  $X$  and  $Y$  is relative to. What are populations? A population  $P$  is a token

population that exemplifies a population type, or kind  $Q$ . So a token population exemplifies many different population types. The relation of causal relevance between  $X$  and  $Y$  depends on which population type  $Q$  a population  $P$ , within which  $X$  is a cause of  $Y$ , exemplifies. As we saw a moment ago, smoking raises the probability of getting lung cancer relative to a token population  $P$  exemplifying a population type  $Q$  (e.g., human beings) (Eells, 1991, p.25.). The relativity of causal relations to a population  $P$  of  $Q$  will be again discussed in section 4.

Another core idea of the three-place theory of probabilistic causation is that a causal factor  $X$  always raises, lowers, or is equal to, the probability of an effect factor  $Y$  in every homogenous background causal context, or subpopulation (Eells, 1991, 2.2.). In other words, the relation of causal relevance between  $X$  and  $Y$  is contextually unanimous, or constant. Otherwise,  $X$  is mixed for  $Y$ . What are background causal contexts? In a word, all of the factors *causally independent of a causal factor  $X$  and causally relevant to an effect factor  $Y$*  are held fixed as causal background contexts. In order to assess a causal factor  $X$ 's causal significance for an effect factor  $Y$ , we need to hold fixed all of the factors  $\{F_1, \dots, F_n\}$  causally independent<sup>1)</sup> of the causal factor  $X$  and causally relevant to the effect factor  $Y$ . Then, the factors  $\{F_1, \dots, F_n\}$  need to be positively or negatively held fixed. Since there are  $n$  number of the factors, there are  $2^n$  ways for them to be held fixed. That is, there are  $2^n$  maximal conjunctions. For example, if there is only one factor  $F_1$  causally independent of  $X$  and causally relevant to  $Y$ , then  $F_1$  is positively and negatively held fixed as follows:

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- 1) The independence of  $\{F_1, \dots, F_n\}$  from  $X$  means that  $\{F_1, \dots, F_n\}$  should not include factors to which  $X$  is causally relevant. For example, suppose that  $F_1$  is the intermediary causal factor between  $X$  and  $Y$ . Then, if  $F_1$  is negatively held fixed as a causal context, then we cannot correctly assess the causal significance of  $X$  for  $Y$  in the transitive relation from  $X$  to  $F_1$  to  $Y$ .

$Pr(Y/ X \& F_1)$ ,  $Pr(Y/ \neg X \& F_1)$  and  $Pr(Y/ X \& \neg F_1)$ ,  $Pr(Y/ \neg X \& \neg F_1)$ . Suppose that a genetic factor  $F_1$  is causally independent of smoking and is causally relevant to lung cancer. We are interested in causal significance of smoking for lung cancer. Then, the genetic factor being held fixed positively and negatively, the causal significance of smoking  $X$  for lung cancer  $Y$  is assessed as follows:  $Pr(Y/ X \& F_1) >, =, < Pr(Y/ \neg X \& F_1)$  and  $Pr(Y/ X \& \neg F_1) >, =, < Pr(Y/ \neg X \& \neg F_1)$ . Of  $2^n$  maximal conjunctions, let  $K_1, \dots, K_m$  be conjunctions that have non-zero probability both in conjunction with  $X$  and in conjunction with  $\neg X$ : for  $i = 1, \dots, m$ ,  $Pr(Y/ X \& K_i) > 0$  and  $Pr(Y/ \neg X \& K_i) > 0$ . These  $K_i$ s are causal background contexts, relative to the assessment of  $X$ 's causal significance for  $Y$ . So, *in every homogeneous<sup>2)</sup> background causal context (subpopulation)  $K_i$  ( $i = 1, 2, \dots, m$ )*,  $X$  is positive, neutral or negative causal factor for  $Y$  if and only if, *for each  $i$* ,  $Pr(Y/ X \& K_i) >, =, < Pr(Y/ \neg X \& K_i)$ . This inequality or equality must hold for *each* of the background contexts  $K_i$ , which is called the condition of contextual unanimity (Eells 1991, 2.3.). Otherwise,  $X$  is mixed for  $Y$ .<sup>3)</sup>

In sum, according to the (three-place) theory of probabilistic causation, a factor  $X$  is positively, negatively or neutrally causally relevant to another factor  $Y$  in a population  $P$  of  $Q$  if and only if the probability of  $Y$  in the presence of  $X$  is, within the population  $P$  of  $Q$ , greater, less than, or equal to, the probability of  $Y$  in the absence of  $X$  in all homogeneous causal contexts. Otherwise,  $X$  is causally mixed for  $Y$ .

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2) A causal context is homogeneous in the sense that the conditionalization of  $X$  on the causal context does not make any difference to the probability of  $Y$  in the presence of  $X$  and the probability of  $Y$  in the absence of  $X$ .

3) For example, if  $Pr(Y/ X \& F) > Pr(Y/ \neg X \& F)$  and  $Pr(Y/ X \& \neg F) < Pr(Y/ \neg X \& \neg F)$ , then  $X$  causally interacts, or is mixed, with  $F$  with regard to  $Y$ .

### 3. Cartwrights Argument against the Theory of Probabilistic Causation

Cartwright claims that the theory of probabilistic causation is not metaphysically informative as follows:

How exactly then should we characterize the test populations? I think that if you want populations in which an increase of probability of Y on X is both necessary and sufficient for X causes Y, you had better pick populations in which *if there is an increase in probability, there is no other account possible than that some Xs are causing Ys and conversely that if Xs can cause Ys, their doing so will result in an increase in probability*. But that is not informative, one may object. Thats true, but not surprising. What can one expect from a general methodological principle? ... But the hunt for general methodological principles that are both effective and generally informative is the hunt for illusion. (Cartwright, 1995, p.186.)

In the above passage, Cartwright distinguishes between pragmatic, or methodological viewpoint and metaphysical viewpoint. The explication of probabilistic causal relations relative to populations is effective or valuable from the pragmatic or methodological point of view, but it is not informative from the metaphysical point of view. For the probabilistic causal relation between X and Y depends on which population it is relative to. If so, what is the causal truth that is not relative to populations? Cartwright claims that it is an unattainable ideal of the ultimate form of a causal knowledge. (Cartwright and Dupre, 1988, p.531)<sup>4)</sup> What is the ultimate form of a causal knowledge?

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4) But without an unattainable ideal of the ultimate form of causal knowledge, this [probabilistic causation] provides no reason to deny that, in the population as a whole, smoking is as good a cause of cancer as anything is of anything else. (Cartwright, [1979], 1983, p.21.)

According to Cartwright, it seems not to be causal effects relative to populations but an uniform causal truth intrinsic across all populations. It seems that only the causal truth<sup>5)</sup> is informative. In order see what the causal truth is, let us look at the following passage.

This (the theory of probabilistic causation) teaches us something important about the correct form for the causal claims that can be based on these general statistical methods: they are always claims about net causal upshots, and not claims about causal laws and causal capacities.(Cartwright and Dupre, 1988, p.534.)

According to this passage, the ultimate causal truth is associated with claims about causal laws and causal capacities. If so, then can theory of probabilistic causation indeed not tell anything about causal laws and causal capacities? And is the theory of probabilistic causation merely a statistical method?

I shall pose three problems with Cartwrights understanding of the theory of probabilistic causation. First, Cartwright misleadingly assimilates the metaphysical theory of probabilistic causation into a methodological theory. Second, it should be noticed that probabilistic causal relations<sup>6)</sup> are claims about causal laws, and the theory of probabilistic causation is a metaphysical theory of causation. Third, contrary to Cartwright argument, the theory of probabilistic causation can represent causal capacities in question. In this section, I shall discuss the first problem. In the following sections, I shall in turn discuss the second problem and the third problem.

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5) We will see that the ultimate causal truth refers to causal capacities carried by singular causes.

6) From now on, I shall substitute probabilistic causal relations for three-place probabilistic causal relations in the three-place theory of probabilistic causation.

We need to note that the theory of probabilistic causation aims to *explicate the concept of causal relations* in terms of probabilistic relations, relative to all homogeneous background contexts.<sup>7)</sup> The probabilistic causal relations in the theory of probabilistic causation are far from simply statistical relations. Statistical methodologies, Cartwright claims, aim to discover causal relations from empirical data and causal background knowledge. But the theory of probabilistic causation is different from such statistical methodologies as causal modeling, path analysis, Bayesian data analysis and so on. The theory of probabilistic causation explicates the notion of causal relations that the statistical methodologies aim to discover from analyses of data. The theory of probabilistic causation concerns not the heuristic program for the discovery of causal relations but a conceptual program for the explication of causal relations. Cartwright should be aware that the theory of probabilistic causation never explicates causal relevance in terms of a statistical net, or average value. Dupre's theory of probabilistic causation (Dupre, 1984) explicates causal relations in terms of the averaging strategy across all of the causal contexts. But it cannot tell statistical correlation from causal relations (i.e., causal laws). This is the most critical problem with the averaging strategy. But the theory of probabilistic causation explicates the relation of causal relevance between *X* and *Y* holding for every homogenous causal context. So the theory of probabilistic causation ascertains whether the relation of causal relevance between *X* and *Y* is unanimous in every homogeneous causal context. If it is not unanimous for every homogeneous causal

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7) The explication of causal relevance in terms of probabilistic relations is circular as indicated in section 1. For *X*'s causal role for *Y* is assessed by other causally relevant factors. But the circularity is not vicious. For *X*'s causal role for *Y* is assessed by the causal roles of factors other than *X* for *Y*.

context, then  $X$  is mixed for  $Y$ . The theory of probabilistic causation aims to explicate true causal relations holding for every homogenous causal context, and is intended to represent causal laws. This is why the theory of probabilistic causation is a metaphysical theory of causation.

In the section 4, I shall clarify that probabilistic causal relations are intended to represent causal laws, and the theory of probabilistic causation is a metaphysical theory of causation. In the section 5, I shall think about the nature of causal capacities and what laws about causal capacities amount to, and will argue that the theory of probabilistic causation is metaphysically informative.

#### 4. Ceteris Paribus Condition, Causal Laws and Probabilistic Causal Relations

Let us first think about what causal laws are. Cartwright makes a distinction between laws of association and causal laws. *Laws of association* concern how often two qualities or quantities are co-associated. They are causally neutral and provide no account of *what makes things happen*. (Cartwright, [1979], 1983, p.21.) On the other hand, *causal laws* have the word *cause*, e.g., Smoking causes lung cancer or Forces causes change in motion. Cartwright claims that causal laws not only underlie laws of association but also are not reducible to laws of association. Cartwright ([1979]1983, p.25.) introduces CC<sup>8)</sup> ("C causes E if and only if C increases the probability of E in every situation

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8) An anonymous referee claims that CC reflects the methodological feature of the theory of probabilistic causation. Of course, if we focus on the way in which probabilistic causal relations represent causal laws, then the theory of probabilistic causation has the methodological feature. But we need to focus on what probabilistic causal relations are intended to represent.



which is otherwise causally homogeneous with respect to  $E$ ) as a connection between laws of association and causal laws. Cartwright (1989, 1995) clearly considers probabilistic causal relations as causal laws. So probabilistic causal relations *are intended to represent causal laws*. In general, causal laws are intended to represent the way causal structure is, which is a metaphysical project. If so, then the theory of probabilistic causation must be a metaphysical theory. By employing *ceteris paribus condition* (CPC), I shall clarify that probabilistic causal relations are causally lawful relations, and establish that the theory of probabilistic causation is a metaphysical theory.

*Ceteris paribus* is in general understood as all other things being equal. A strict law might say that  $X$  events always cause  $Y$  events, or that  $X$  events have such and such objective chance of causing  $Y$  events. (S. Schiffer, 1991). Then, the *ceteris paribus condition* is established by *ceteris paribus* being added to the strict law;  $X$  events cause  $Y$  events *ceteris paribus*, or *all other things being equal*,  $X$  events have such and such objective chance of causing  $Y$  events. In general, CPC plays a crucial role in rendering scientific laws lawlike. Consider, for example, Snells law refined by CPC as follows:

Snells law:

*Refined Snells Law: For any two media which are optically isotropic, at an interface between dielectrics there is a refracted ray in the second medium, lying in the plane of incidence, making an angle  $\theta_2$  with the normal, such that:  $\sin \theta / \sin \theta_1 = n_2/n_1$ .*

The difference between unrefined Snells law and refined Snells law rests on whether or not the italic modifier is added. In the refined Snells law, the italic modifier plays a role of *ceteris paribus condition*. Without the italic modifier, Snells law is a crude statistical law.<sup>9)</sup>

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9) With regard to Snells law, Cartwright (1983) would like to pose a problem

Humphreys (1989, p.130.) clarifies four applications of CPC in the philosophy of science:

- (1) Any factor which has not been explicitly included in the causal claim, law, or model is assumed to be irrelevant.
- (2) All relevant or unknown factors not explicitly included in the causal claim, law, or model are assumed to be constant.
- (3) All relevant or unknown factors not explicitly included in the causal claim, law, or model are assumed, on average, to have no effect.
- (4) All relevant factors are not explicitly included in the causal claim, law, or model, whether known or unknown, are assumed to be absent (have value zero).

Considering this clarification of CPC (in particular, (2)), we can see that probabilistic causal relations represent causally lawful relations. The relativity of the probabilistic causal relation between  $X$  and  $Y$  to a population  $P$  of  $Q$  and the relativity of the probabilistic causal relation between  $X$  and  $Y$  to each homogeneous causal context in  $P$  of  $Q$  play the same role of CPC in the theory of probabilistic causation as follows: *All of the factors causally independent of  $X$  and causally relevant to  $Y$  being held fixed as homogeneous causal contexts in  $P$  of  $Q$ ,  $X$  always raises, lowers or does not change, the probability of  $Y$ , assuming that the relation of causal relevance between  $X$  and  $Y$  is relative to  $P$  of  $Q$ .* The italicized parts render the relation between  $X$  and  $Y$  *invariant, or uniformly causally relevant*. Otherwise,  $X$  is causally mixed for  $Y$ . Invariant relationships are "those relationships which will remain stable or continue to hold under some class of changes or interventions or

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with CPC such that CPC ultimately renders every statistical law non-statistical. Later, Cartwright (1989, 1995) also claims that laws about capacities are not bound to CPC, which makes a difference between laws about capacities and the other laws.

permutations in initial or background conditions." (Woodward, 1994, p.355-356.) Invariance represents physically lawful necessity. Invariance relationships are relative to background conditions, or contexts (e.g. *ceteris paribus* conditions). So a relationship is not invariant if the relationship does not remain constant when disrupted by changes in background conditions. So, in the theory of probabilistic causation, homogeneous background causal contexts in a population  $P$  of  $Q$  are boundary conditions, which render the relation between  $X$  and  $Y$  causally invariant, or lawful. The boundary conditions are homogeneous with the invariant causal relationship in the sense that, in the boundary conditions, the invariant relationship remains constant.

I have clarified the way in which probabilistic causal relations represent causal laws. Cartwright admits that probabilistic causal relations are causally lawful relations. If so, Cartwright should also admit that the theory of probabilistic causation is a metaphysical theory of causation. If Cartwright would like to maintain that the theory of probabilistic causation is a kind of methodological theory in question, then she at least misleadingly should not associate it with statistical correlation about net value, or average causal significance. Cartwright says, Eells is not interested in claims of capacities but only in context-dependent causal laws. (1995, p.183.) Cartwright (1989, 1995) claims that (three-place) probabilistic causal relations are "context-dependency causal laws": Probabilistic causal relations depend on which population  $P$  of  $Q$  they are relative to. So Cartwright thinks that probabilistic causal relations represent causal capacities *partially or locally*. If so, then Cartwright does not say that probabilistic causal relations does not represent causal capacities at all. In fact, Cartwright (1995, p.185.) would like to emphasize that her theory does a better job in representing causal capacities carried by singular causes than the theory of probabilistic causation. This is the reason why Cartwright

claims that the theory of probabilistic causation is not metaphysically informative. In the following section, I shall first think about the nature of causal capacities and what laws about causal capacities amount to. I shall argue that the modality (i.e., potentiality, or causal tendency) probabilistic causal relations take on represents what causal capacities are intended to represent. An upshot is as follows: The modality depends on the fact that causal relations are explicated in terms of probabilistic relations relative to a population  $P$  of  $Q$ . And the probabilistic relations depend on the hypothetical relative frequency relation relative to a population  $P$  of  $Q$ . Thus, causal capacities concerning singular causes are explicable in terms of such a modality as tendency, or propensity concerning singular causes.

## 5. Causal Capacities, Tendencies and Two Types of Causal Laws

Cartwright explicates causal capacities in terms of Mills concept of tendency.

We might, indeed, guard our expression ... by saying that the body moves in [the prescribed] manner unless prevented, or except in so far as prevented, by some counteracting cause, but the body does not only move in that manner unless counteracted, it tends to move in that manner even when counteracted. ... These facts are correctly indicated by the expression tendency. All laws of causation, in consequence of their liability to be counteracted, required to be stated in words affirmative of tendencies only, and not of actual results. (J.S.Mill, *System of Logic*, p.444-5. in Cartwright (1989) p.177.)

Cartwright (Ibid., p.178.) derives two different laws from Mills account of tendency. First, tendency laws represent only *regularities*<sup>10)</sup>

Tendency laws are bound to *ceteris paribus* condition or counterfactual. Second, laws about tendencies represent *tendency* itself<sup>11)</sup> Only laws about tendencies represent fixed capacities. (Ibid., p.178.) According to Cartwright, the theory of probabilistic causation concerns only tendency laws. Cartwright claims that even though probabilistic causal relations are tendency laws in terms of *ceteris paribus* condition and counterfactual, they are not laws about tendencies. For laws about tendencies alone represent tendencies (i.e., capacities) uniformly moved despite certain restriction or counteractions. Let us recapitulate Cartwrights accounts of causal capacities and laws about causal capacities: First, causal capacities are tendencies in the Mills sense. The tendencies are unconditionally or persistently invariant. Second, laws about causal capacities are unconditional laws in the sense that they are not bound to such a restriction as *ceteris paribus* condition. Third, causal capacities are ascribed to singular causes. A question arises about the first two points: What does it mean to say that laws about capacities represent persistently invariant capacities without any boundary condition?

Cartwright does not introduce any particular example to demonstrate unconditional laws about capacities, except that laws about capacities

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10) That (*ceteris paribus*) means they would hold if all disturbing causes were absent. But that will not do. That was Mills own view, and one that I support as well. Even if these regularities did hold *ceteris paribus*or, other thing being equalthat would have no bearing on the far more common case where other things are not equal. (Ibid., p.177.).

11) More important for my thesis, however, is not the fact that laws which are nearly true, albeit for particular situations and finite periods, are not fundamental, but rather that fundamental laws are not true, nor nearly true, nor true for the most part. That is because fundamental laws are laws about distinct atomic causes and their separate effects; but when causes occur in nature they occur, not separately, but in combination. (Ibid., p.175.).

are analogous to laws about tendencies in the Mills sense. Cartwright merely addresses what causal capacities and laws about causal capacities should be like. At this point, let us think about whether there is any significant distinction between tendency laws (e.g., causal laws) and laws about capacities. Let us look at the following passage.

They (causal laws including probabilistic causal laws) describe what would happen were the situation like that. But by their very nature they do not describe what would happen were the situation different. ... They are derivative, like all regularly laws. Regularities, whether causal or associational, arise only as a consequence of the operation of a capacity. (1995, pp.154-155.)

Cartwright claims that causal laws do not *represent what would happen if the situation were different*. If so, laws about capacities are supposed to represent what would happen if the situation were different. And this is the way in which laws about capacities are supposed to represent the invariance of causal capacities across every situation. If so, then I do not see why probabilistic causal relations cannot fully represent causal capacities. In the theory of probabilistic causation, probabilistic causal relations represent different causal tendencies, depending on which population  $P$  of  $Q$  they are relative to. This is exactly *what probabilistic causal relations would be if they were in different situations*: Depending on which each population  $P$  of  $Q$  a probabilistic causal relation is relative to, the probabilistic causal relation represents the relation of positive, negative, neutral or mixed causal relevance. I think that the four types of causal relevance represent the operation of causal capacities exhaustively. In order to render this point more vivid, let us think about the third point of causal capacities: Causal capacities are ascribed to singular causes. Cartwright says that, as a mixed, or dual causal factor turns out, both probabilistic causal relations and the

claims about causal capacities do not fare better. (1989, pp.163-164., 1995, p.185.) But the claims about causal capacities (that is, her theory) do a better job in representing causal capacities. Cartwright believes that claims about causal capacities cannot be fully represented by probabilistic relations, for her theory alone reveals causal capacities carried by singular causes. Cartwright (1989, Ch.3) claims that singular causal cases should be invoked in the account of general causal facts. If we examine Cartwright accounts of singular causal cases, then we can see that Cartwright considers singular causal cases as continuous causal processes concerning singular causes or relative frequencies of individuals. (Cartwright 1989, pp.101-102.) The causal processes concerning singular causes indicate not actual causal processes but potential causal processes represented as counterfactuals.<sup>12)</sup> (It is needless to say that relative frequencies represent tendency or potentiality.) So individuals causal processes or relative frequencies of individuals represent such a modality as potentiality or tendency. At this point, let us look at the difference between type-level causation and token-level causation. Type-level, or general causal relations are the relations between properties, or factors, e.g., the relation between the *property* of smoking and the *property* of lung cancer. Type-level causation represents causal tendency. On the other hand, token-level, or singular causal relations between events at a particular time and place, e.g., the causal relation between Simpsons smoking at  $t_0$  and his getting lung cancer at  $t_1$ . So the situations concerning singular causation require actual events to stand in causal relations at a

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12) Each test population of individuals for the law  $X$  causes  $Y$  must be homogenous with respect to some complete set of  $Y$ s causes (other than  $X$ ). However, *some individuals* may have been causally influenced and altered by  $X$  itself; just *these individuals* should be reassigned to populations according to the value they would have had in the absence of  $X$ s influence. (Cartwright, 1989, p.96.)

particular time and location. But the situations concerning singular causation introduced by Cartwright are not associated with the situations concerning actual causal relations. Cartwright associates singular causal cases with *tendencies of singular causal cases expressed by counterfactuals*. As far as Cartwright is concerned not with actualized singular causal cases but with causal capacities of individuals, the theory of probabilistic causation can explicate causal capacities in the situations introduced by Cartwright, without invoking singular causation. The theory of probabilistic causation concerns individuals or singular cases (i.e., a population of individuals or a collection of singular cases) and aims to represent tendencies or propensities carried by individuals or singular cases. If capacities are ascribed to *individuals, or a group of individuals*, then the theory of probabilistic causation provides a formal generalization of capacities being ascribed to singular causal cases. Let us look at the way in which causal capacities are explicable in terms of the core idea of the three-place theory of probabilistic causation.

Consider again the causal claim Smoking causes lung cancer. Assuming that indeterminism holds for the world, a question arises about how the causal claim represents the invariant causal tendency of smoking for lung cancer. If the relation between smoking and lung cancer is indeterministic, then smoking does not necessitate lung cancer. Smoking has indeterministic causal tendency for lung cancer. The causal tendency refers to *potentiality*, or the potentiality of actual causal relations. In the theory of probabilistic causation, the modality of probability is intended to represent the modality of indeterministic causal tendency: The conditional probability of  $Y$  given  $X$  (i.e.,  $Pr(Y/X)$ ) is intended to represent the causal tendency of  $X$  for  $Y$ . The modality of probability of  $Y$  given  $X$  comes from the *hypothetical* relative frequency of  $Y$  given  $X$  in a *hypothetical* population  $P$  of  $Q$ .<sup>13</sup> Why



are causal capacities of singular causes explicable in term of the hypothetical relative frequency of  $Y$  on  $X$  in a *hypothetical* population  $P$  of  $Q$ ? As we saw a moment ago, Cartwright refers to singular causal causes not as actual singular causal cases but as potential singular causal cases. Also, Cartwright associates capacities not with the singular causal case but singular causal cases, or a collection of singular causal cases. So the causal capacity of smoking for lung cancer at the type level depends on the causal capacities ascribed to singular causal cases. The casual capacity of smoking for lung cancer represents the operation of the causal capacities ascribed to singular causal cases. So the causal capacity of smoking for lung cancer is explicable if we show that the causal capacity of smoking for lung cancer represents the causal capacities ascribed to singular causal cases, or a collection of singular causal cases.

Consider, for example, the probability of a coin coming up with a head when it is tossed. A population  $P$  is 20 trials on which a coin is tossed. I toss a coin in two ways. First, I first toss a dice. So if the dice comes up with one or six, then I toss a coin. Otherwise, I do not toss a coin. Let this kind of population be a population type  $Q$ . Second, I first toss a dice. If the dice comes up with two, three, four or five, then I toss a coin. Otherwise, I do not toss a coin. Let this kind of population be a population type  $Q^*$ . So the population  $P$  can exemplify either of the two kinds of a coin toss:  $Q$  and  $Q^*$ . Then, it is clear that the probability of a coin coming up with a head given a coin toss is different, depending on which of the two population types a population  $P$  exemplifies. Suppose that a coin is tossed *infinitely*, and a population  $P$  is *infinite* trials on which a coin is tossed. Suppose that

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13) I shall not introduce a detailed formal proof of how  $\Pr(Y/X)$  is interpretable as  $HRF(Y/X)$  in  $P$  of  $Q$  ( $HRF$ : hypothetical relative frequency) since it needs another long discussion.

the population  $P$  is hypothetical. Then, according to the probability interpretation of the hypothetical relative frequency, the probability of a coin coming up with a head given a coin toss comes from the *hypothetically infinite frequency* of a coin coming up with a head given a coin tossing in a *hypothetical* population of infinite trials  $P$  of  $Q$  or  $P$  of  $Q^*$ . Of course, the probability of a coin coming up with a head given a coin toss is different, depending on which of the two population types a population  $P$  exemplifies. Let us consider the probability of lung cancer given smoking. Let  $P$  be a hypothetical population of infinite number of individuals. The probability of lung cancer ( $Y$ ) given smoking ( $X$ ) depends on the *hypothetical relative frequency* of lung cancer on smoking in a *hypothetical* population  $P$  of  $Q$  (for example, a hypothetical population  $P$  of human beings population type  $Q$ ). Thus, the probability of  $Y$  given  $X$  represents the causal capacity (i.e., invariant causal tendency or causal potentiality) of  $X$  to bring about  $Y$  relative to  $P$  being of  $Q$ . This is the way in which the probabilistic causal significance of  $X$  for  $Y$  formally represents that *individuals* in  $P$  of  $Q$  carry the causal capacity of  $X$  for  $Y$ .

Against this explication of the modality of capacities in terms of the modality of probability, Cartwright would emphasize that capacities are something concrete, or real without modalities. But I do not see what capacities without modalities might be like and how capacities without modalities might be measured. For there is the most critical problem with capacities without modalities: How might it be possible to consider laws about capacities if capacities do not take on any modality? Finally, Cartwright should notice that the fact of singular causal events carrying capacities does not logically imply that singular causal events actualize the causal capacities and vice versa.

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## 인과의 힘, 인과 법칙 그리고 확률적 인과성

김 준 성

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초기 카트라이트(Cartwright), 스킴즈 (Skyrms), 엘스(Eells)에 의해 발전된 확률적 인과 이론의 기본 개념은 다음과 같다. 한가지 요소 X와 인과적으로 아무런 관계가 없는, 그리고 다른 요소 Y와 인과적 관련성을 가진 모든 요소들을 배경 요소들로서 (즉,  $K_i$  ( $i = 1, \dots, n$ )) 고정시켰을 때,  $K_i$ 가 주어진 상태에서 X가 Y의 확률을 높인다면, X는 Y의 원인 요소가 된다. 그때, 항상 한가지 속성을 가진 집단 (population) (예를 들어 인간들의 집단) 안에서 X는 Y의 원인 요소가 된다. 후기 카트라이트(Cartwright)는 이런 인과 관계에 대한 확률적 해명이 인과의 힘(causal capacities)을 충분히 해명하지 못하기 때문에 확률적 인과 이론이 형이상학적으로 빈곤한 이론이라고 주장한다. 나는 우선 확률적 인과 이론이 통계적 방법에 관한 이론과는 다르다는 것을 해명한다. 둘째, 확률적 인과 이론이 제한적 배경 조건하에서 인과 법칙을 해명하려는 형이상학적 이론임을 밝히고자 한다. 셋째, 확률적 인과 이론이 카트라이트가 주장하는 인과의 힘을 해명할 수 있다고 주장한다. 카트라이트가 제시하는 인과의 힘은 한 속성을 가진 집단에 의존하는 가설적 상대 빈도 수와 다르지 않으며, 따라서 인과의 힘은 확률적 인과 이론이 보여주려는 인과적 성향과 다르지 않다.