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# THE CONFIRMATION OF NON-CAUSAL DEPENDENCIES: ON KISTLER'S "CONSTITUTION AND CAUSATION IN MECHANISMS"

## La confirmación de las dependencias no causales: Acerca de "Constitution and Causation in Mechanisms" de Kistler

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**Abstract**

Kistler's "Constitution and Causation in Mechanisms" raises important issues regarding Craver, Glennan, and Povich's "Matched Interlevel Experiments" approach to determining relations of non-causal dependence. Is this theory meant to be an account of actual scientific practice or merely a set of joint sufficiency conditions for determining relations of non-causal dependence? Has the manipulability approach to relations of non-causal dependence overlooked an important scientific source of evidence for relations of non-causal dependence? This paper draws attention to these questions.

**Key words:** Abduction; Matched Interlevel Experiments; Mutual Manipulability; Non-Causal Dependence.

**Resumen**

El trabajo de Kistler, "Constitution and Causation in Mechanisms", plantea cuestiones importantes respecto al enfoque de los "experimentos internivel emparejados" de Craver, Glennan y Povich para determinar relaciones de dependencia no causal. ¿Se supone que esta teoría constituye una explicación de la práctica científica real o simplemente un grupo de condiciones de suficiencia conjunta para determinar relaciones de dependencia no causal? ¿Acaso el enfoque de la manipulabilidad para las relaciones de dependencia no causal ha pasado por alto una importante fuente científica de evidencia para las relaciones de dependencia no causal? Este artículo aborda estas preguntas.

**Palabras clave:** Abducción; Experimentos internivel emparejados; Manipulabilidad mutua; Dependencia no causal.

## 1. Introduction

Craver (2007) proposed to show how an interventionist method for inferring causal dependencies—made famous among philosophers through

Woodward (2003)—might be extended to provide an interventionist method for inferring non-causal dependences. A familiar example of such a non-causal dependence is the dependence of the rising phase of the action potential (an S Ψ-ing) on an influx of sodium ions into the axon (a set of $x_i$'s $\varphi_i$-ing). Simplifying, Craver's methodological proposal was that to establish that an S Ψ-ing non-causally depends on a set of $x_i$'s $\varphi_i$-ing, it is sufficient to combine a manipulation of the S Ψ-ing and a detection of a change in some $x_i$'s $\varphi_i$-ing and a manipulation of an $x_i$'s $\varphi_i$-ing with a detection of a change in S Ψ-ing.[1] This is often referred to as a "mutual manipulability" theory.

A central problem for the mutual manipulability theory has been that Woodward's conditions on ideal interventions—the relevant kinds of manipulations and measurements—were unsatisfiable. Thus, the theory has been widely recognized as vacuous.[2] Attempts to address this problem lead Craver, Glennan, and Povich (CGP) to propose what they now call the "matched interlevel experiments" (MIE) approach.[3] This is where Kistler's critique enters this picture.[4] Most notably, he proposes that CGP embrace an untenable nominalism about higher level S's Ψ-ing.

This brief discussion has two goals. First, it draws attention to a lack of clarity regarding the aims of the interventionist approach. Sometimes the approach appears to be a theoretical account of actual scientific practice, and sometimes it appears to be a mere set of joint sufficiency conditions for establishing the existence of non-causal dependency. This lack of clarity is found in both the interventionist literature and in Kistler's discussion. The second goal is to suggest an account of how scientists confirm hypotheses of non-causal dependence, namely, a theory of abductive confirmation.

The paper will discuss these goals in reverse order. Section 1 will point to cases of scientific experimentation on non-causal dependencies that fall outside the scope of the interventionist proposals developed so far. Many scientists rely on abductive inferences to confirm the existence of non-causal dependencies. They manipulate an object, measure its behavior, and then postulate undetected, unmeasured things that non-

[1] This statement is a simplification in at least three respects. There is no discussion of 1) Craver's concepts of activation and inhibition, 2) the details about so-called "ideal interventions," and 3) whether the account is supposed to provide necessary and sufficient conditions for non-causal dependency or merely sufficient conditions. Each of these topics has played a significant role in the literature to date, but is tangential to the issues discussed here.

[2] See, for example, Romero (2015) and Baumgartner and Gebharter (2016).

[3] Other attempts leading up to the CGP approach include Krickel (2018), Harinen (2018), Baumgartner, Casini, and Krickel (2020), and Prychitko (2021).

[4] Kistler (2025).

causally explain what underlies the connection between the manipulation and the measurement. This is the method Horace Barlow adopted in his account of simultaneous contrast. This is the method adopted by Alan Hodgkin and Andrew Huxley in their experimental work on the action potential.[5] Section 2 will turn to Kistler's critique of CGP. Kistler's charge of nominalism regarding S's Ψ-ing is an objection to MIE insofar as MIE is meant to be an account of actual scientific practice. But it is irrelevant insofar as interventionism is only meant to be a logically possible set of joint sufficiency conditions for determining non-causal dependencies.

It should be noted at the outset that the aims of this brief discussion are not to tweak one or another condition on an interventionist account of the confirmation of non-causal dependencies. There is already an active literature on this. Nor is the aim to provide a fully fleshed-out alternative to interventionist accounts.[6] Instead, the aim is to clarify the aims of the interventionist approach and suggest a new avenue of investigation regarding the confirmation of relations of non-causal dependence.

## 2. The Abductive Road Not Taken

Early in *Explaining the Brain*, Craver proposes that fuzzy dice are not part of the driving mechanism of a car.[7] Suppose that this is correct and that each of us knows that this is correct. But step back for a moment. Does a pairing of top-down and bottom-up experiments have anything to do with this knowledge? Probably not. Moreover, most of us believe that the pistons of an internal combustion engine are part of the driving mechanism of the car. Is this because each of us knows about the pairing of top-down and bottom-up scientific experiments that confirm the role of the pistons in the mechanism of the engine? Probably not. Maybe such experiments have never even been performed.

But, if a combination of top-down and bottom-up experiments does not justify our belief that the fuzzy dice are not part of the driving mechanism of the car or our belief that pistons are part of the driving mechanism of the car, then what does? In short, it is our well-confirmed background beliefs. Each of us knows enough about fuzzy dice and how automobile engines work to know that fuzzy dice are not part of the mechanism of the car, whereas the pistons are.

[5] See, for example, Aizawa and Headley (2022) and Aizawa (2025).

[6] For something more closely approximating this, see Aizawa and Headley (2022) and Aizawa (2025).

[7] Craver (2007, p. 4). Harinen (2018) uses an example of the caliper brakes on a bicycle. Harinen's example would serve present purposes just as well.

But this is just common sense. It is a toy example, if you will. This point raises another issue. How is it that *scientists* determine what is non-causally dependent on what? As Kistler comments, "Much effort has been spent on trying to provide an analysis of the empirical criteria scientists use for judging that activity $\Phi_i$ of part $X_i$ of organism $S$ is *constitutive* of $S$'s mechanism $\Psi$." (Kistler, 2025, p. 573). Craver also sometimes suggests that he wants a method that scientists use.[8] Suppose philosophers of science want to know how scientists justify claims of non-causal dependence. A reasonable place to look is in the experimental scientific literature.

Consider a very simple example of how one scientist tried to justify a hypothesis of non-causal dependence. In the 19th century, the French chemist Michel-Eugène Chevreul reported on extensive studies of simultaneous contrast. To take a simple case of the phenomenon, a gray box will appear darker against a white background than against a black background. (See Figure 1.) A subject experiencing simultaneous contrast is an S $\Psi$-ing. In 1953, Horace Barlow reported frog ganglion cells that have ON-center, OFF-surround response properties. That is, the cells fired more vigorously when illuminated in their centers, but less vigorously when illuminated in their periphery. (See Figure 2.) These ganglion cells are $x_i$'s $\varphi_i$ -ing. In his report, Barlow connected simultaneous contrast and the activity of these ganglion cells:

> Simultaneous contrast effects are presumably caused by an inhibitory mechanism similar to the one described; a white spot surrounded by black looks brighter (more impulses) than a white spot of the same intensity surrounded by grey, because the grey falls on the inhibitory fringe of the ganglion cells, and so inhibits their discharge (causes fewer impulses) (Barlow, 1953, p. 85). (See Figure 3.)

Barlow believed that ganglion cells were at least part of the mechanism of simultaneous contrast.

[8] See, for example, Craver (2007, p. vii). Kaplan (2012) and Prychitko (2021) provide examples of scientists trying to confirm hypotheses about non-causal dependence. Why? A reasonable conjecture is that they mean for the examples to illustrate scientists using the interventionist approach.

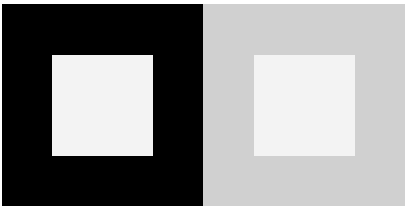


Figure 1. Simultaneous Contrast.

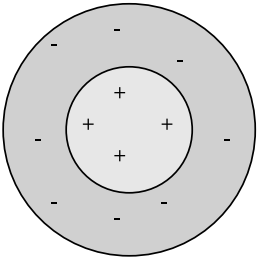


Figure 2. ON-center, OFF-surround cell.

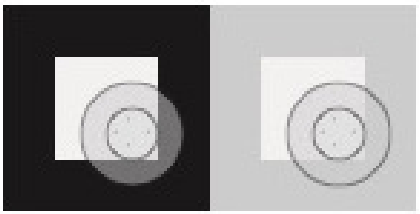


Figure 3. Barlow's ganglion cell explanation of simultaneous contrast.

But what reason did Barlow have for believing this? He did not, for example, perform top-down and bottom up experiments on frogs perceiving simultaneous contrast. His experimental work revealed the response properties of ganglion cells, but that did not link ganglion cells to simultaneous contrast. For this linking step, Barlow relied on his background knowledge to forge the explanation. He believed that ganglion cells were the mechanism of simultaneous contrast, because ganglion cells would explain simultaneous contrast. To a first approximation, he believed that ganglion cells were the mechanism of simultaneous contrast by way of an inference to the best explanation, where the best explanation was a non-causal explanation.[9]

[9] For a better approximation to what Barlow was doing—why one should think

One might object that this was just a one-off illustration or that Barlow did not really *know* that ganglion cells were the mechanism of simultaneous contrast. But this is to overlook the vast tracts of science that work in much the same way. Consider, for just one more example, Hodgkin and Huxley's first experiment in Hodgkin and Huxley (1952). In this experiment, Hodgkin and Huxley voltage clamped an axon in a sodium-containing solution, then in a sodium-free solution, then again in a sodium-containing solution. These three test runs lead to an inward current, then an outward current, then an inward current. (See Figure 4.) The underlying mechanism is that after voltage clamp, the membrane changes permeability, allowing sodium to move down its concentration gradient, either into the cell or out of the cell.

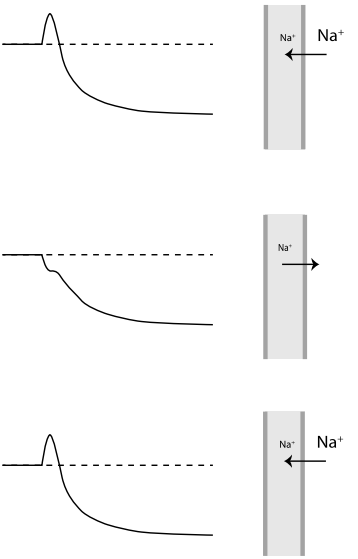



Figure 4. Hodgkin and Huxley's explanation of the results of their first experiment in Hodgkin and Huxley (1952). The axon voltage clamped with normal amounts of sodium in the extracellular medium (top and bottom panels) generates an initial inward current, shown as a little "hill" on the left side of the current trace. The axon voltage clamped in a sodium-free extracellular medium (middle panel) generates an initial outward current, shown as a little "dip" on the left side of the current trace.

In this experiment, Hodgkin and Huxley did not use either top-down or bottom-up techniques. They voltage clamped the axon and then measured a current in the axon. It was an intralevel experiment. In this

that Barlow was doing abduction, rather than inference to the best explanation—see Aizawa (2025). Peirce is often read as believing that abductive inferences do not provide confirmation; they only serve to introduce (pursuit-worthy) hypotheses. See, for example, Cabrera (2023) for this view and numerous references to the literature. Aizawa (2025) argues that scientists do not use abduction just to introduce hypotheses; they often use it to confirm hypotheses.

experiment, the axonal currents were *S* Ψ-ings that they took to be non-causally dependent on the movements of sodium ions, an $x_i$ $\varphi_i$-ing. To forge this explanation, Hodgkin and Huxley relied on background knowledge about intracellular and extracellular sodium concentrations and that sodium is a positively charged ion. In other words, to a first approximation, they believed in the change in membrane permeability because of an inference to the best non-causal explanation.

Note that it is logically possible for scientists to use both abductive reasons and interventionist reasons as evidence for non-causal dependency. The two methods can, logically speaking, be complementary. Complementary roles for "abductionism" and interventionism does not, of course, negate the importance of abduction. More importantly, this logical point does not speak to the issue of the empirical criteria scientists use for judging that activity $\varphi_i$ of part $x_i$ of organism *S* is constitutive of *S*'s mechanism Ψ. As a historical point, Hodgkin and Huxley never used interlevel experiments, since they were, in some sense, impossible. Given mid-twentieth-century electrophysiological technology, Hodgkin and Huxley could not directly measure sodium ion fluxes. Scientists may one day develop technology to manipulate and measure these things directly, so that interventionist interlevel experiments may not be strictly speaking nomologically impossible. Nevertheless, in point of historical fact, the empirical criteria Hodgkin and Huxley used never included interlevel experiments.

## 3. The Interventionist Road

Let me now connect the foregoing scientific examples to the details of Kistler's critique of CGP.[10] First of all, like many of the interventionists, Kistler does not univocally state the goal of the interventionist project. As noted above, at one point refers to the empirical criteria scientists use (Kistler, 2025, p. 573). This suggests that the project is one of describing or characterizing some actual scientific practice. At another point, however, he claims that "The task is to spell out a conceptually coherent experimental criterion for constitution, i.e., a sufficient condition for one variable $\Phi_i$ being a constituent of variable Ψ, where all variables are both observable and at least in principle experimentally manipulable." (Kistler, 2025). This, by contrast, suggests that the project is a matter of abstract theorizing.

[10] Kistler (2025), of course, includes a lot that is not discussed here, namely, a critique of Baumgartner, Casini, and Krickel (2020) and Kistler's own positive proposal for an interventionist approach to non-causal dependence.

The distinction is central to evaluating Kistler's critique. Kistler proposes that MIE offers a nominalist conception of mechanisms that dispenses with *S*'s Ψ-ing, but that this does not fit scientific practice: "This nominalist conception of mechanisms is eliminativist for activities of all levels except the fundamental. Such mechanistic nihilism is ontologically parsimonious but it accounts neither for scientific practice nor for scientific models" (Kistler, 2025), and "Mechanistic nihilism matches neither scientific practice nor scientific theory" (Kistler, 2025). Kistler does not argue that MIE is a conceptually incoherent experimental criterion for constitution. So, one can see how crucial it is to Kistler's project that he correctly assesses what MIE is meant to do. If MIE is meant to describe actual scientific practice, then Kistler at least has a plausible case. But, if MIE is merely meant to provide a conceptually coherent experimental criterion for constitution, his observation regarding actual scientific practice is beside the point.

### *3.1. Two possible CGP replies*

#### *3.1.1. Embodied and disembodied processes*

One might doubt that Kistler's charge of nominalism is fair to CGP. Here is why. Kistler proposes that MIE eliminates higher level entities and activities, such as S Ψ-ing, in favor of causal chains of lower level entities and activities, $x_1$ $\phi_1$-ing, $x_2$ $\phi_2$-ing, … $x_n$ $\phi_n$-ing. The idea of MIE is then to establish non-causal dependence by establishing the existence of two sub-chains, one from $x_1$ $\phi_1$-ing to $x_i$ $\phi_i$-ing and the other from $x_i$ $\phi_i$-ing to $x_n$ $\phi_n$-ing. This is done by manipulating S $\Psi_{input}$-ing and then measuring $x_i$ $\phi_i$-ing and manipulating $x_i$ $\phi_i$-ing and then measuring S $\Psi_{output}$-ing. This is how Kistler interprets CGP's claims that "constitutive relevance is causal betweenness" (Craver, Glennan & Povich, 2021, p. 8807) and "constitutive relevance […] amounts to a kind of causal mediation" (Craver, Glennan & Povich, 2021, p. 8821).

At one point, CGP introduce a distinction between embodied and disembodied processes. The embodied cases appear to be the familiar cases in which $x_1$ $\phi_1$-ing, $x_2$ $\phi_2$-ing, … $x_n$ $\phi_n$-ing explains S Ψ-ing. The disembodied cases are relatively unfamiliar instances in which there is supposed to be no S that does the Ψ-ing. In their own words, CGP propose that "some mechanisms, like erosion on a riverbank or the Rayleigh scattering that makes the sky blue, are not embodied in entities; they are not mechanisms by which an entity acts […] In such cases there is no determinate entity S that ψs […] there is only ψ-ing" (Craver, Glennan & Povich, 2021, p. 8811). In this passage, CGP are eliminating S. Then, two paragraphs later, they

write, "The higher-level activity, ψ-ing, just is an organized collection of $X_i$ $\varphi_i$-ing." (Craver, Glennan & Povich, 2021, p. 8812). In this passage, CGP appears to be eliminating the Ψ-ing. There is no Ψ-ing; there are just $x_i$ $\phi_i$-ings. This is some textual basis for Kistler's thinking that CGP are indeed mechanistic nominalists.

CGP might use this point to reply to Kistler. CGP might emphasize that there are supposed to be two kinds of cases. There is the familiar embodied S Ψ-ing and the newly recognized disembodied Ψ-ing. So, CGP might claim, Kistler is misinterpreting the view. They might continue, "Look, we also write about levels in our new view: 'There is a level of input–output relations (i.e., a "phenomenal" level), and there is a level of the component parts and activities in virtue of which that input–output relationship is maintained.' (Craver, Glennan & Povich, 2021, p. 8813)" How can Kistler think that we are mechanistic nominalists when we write about higher and lower levels?

Suppose we take there to be a *bona fide* distinction between embodied and disembodied mechanisms.[11] Concede for the sake of argument—as Kistler may be willing to do—that CGP can handle the disembodied cases. Kistler's objection still indicates that MIE does not provide an account of how scientists confirm hypotheses about non-causal dependence in the originally investigated cases of embodied Ψ-ing—the many cases of S Ψ-ing.

*3.1.2. Joint sufficiency conditions that scientists use*

Earlier, we noted that if MIE is merely meant to provide a conceptually coherent experimental criterion for constitution, Kistler's observation regarding actual scientific practice is beside the point. But that may not be the best interpretation of what CGP intend as the goal for MIE. CGP explicitly formulated MIE as a set of joint sufficiency conditions for one variable $\phi_i$ being a constituent of variable Ψ.[12] The worry one might have about this interpretation is that it threatens to undermine the value of MIE as a bit of philosophy of science. Why would MIE be relevant to the philosophy of science, if MIE is not used in science? Maybe analytic epistemologists would care about mere joint sufficiency conditions, but why would philosophers of science care? What would such joint sufficiency conditions tell us about science?

Perhaps the more generous interpretation of what CGP have in mind—one that has never been explicitly formulated in the literature—is

[11] In correspondence, Kistler expressed doubts about this distinction.

[12] Craver, Glennan, and Povich (2021, p. 8822).

that they are offering joint sufficiency conditions *that scientists actually use*. Unfortunately for GCP, this reply simply returns them to Kistler's objection that the nominalism built into the conditions does not capture actual scientific practice. Thus, Kistler's main point still stands: for the many cases of the embodied mechanisms that were originally of interest, MIE is not the method scientists use. It is not the method scientists use because MIE is designed for a scientific ontology that scientists do not embrace.

## 4. Conclusion

This discussion note has had two goals. The first was to draw attention to a lack of clarity regarding the aims of the interventionist approach. Most notably, the approach might be interpreted as a theoretical account of actual scientific practice, or it might be interpreted as merely a set of joint sufficiency conditions for establishing the existence of non-causal dependency. We have seen how clarity on this issue is not only important to appreciating what MIE is up to but also essential to evaluating Kistler's critique. The second goal was to suggest an alternative to the interventionist account, a theory of abductive confirmation. Skeptics will not be swayed by two briefly presented examples, but there is much more one might say about the cases.

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