

Interlevel Causation

Synonyms

Definition

Interlevel causation refers to the causal influence of an entity at one level on an entity at another. Top-down causation is said to occur when items at higher levels influence items at lower levels. Bottom-up causation is said to occur when items at lower levels influence items at higher levels. On some readings of the terms *level* and *causal influence*, the idea of interlevel causation is metaphysically mysterious. On another reading, interlevel causation is a metaphysically central and utterly commonplace feature of sciences that integrate the reductive pursuit of lower-level mechanisms with the discovery of higher level causes and effects in systems biology.

Characteristics

1. The Allure of Inter-level Causal Discourse

In the quest for explanation scientists propose and test causal claims (e.g., neurotransmitter release in neurons causes muscles to contract). Each of the putative causes is independent of its effect and precedes it in time. Manipulating the cause changes the effect. But sometimes explanation appeals not to something preceding and independent of the affected entity, but to the activities or operations of its components (as ionic diffusion explains the action potential). This explanatory relationship between components and the containing mechanism gives rise to the thought that they are causally related as well. Because the mechanism is at a higher level than its components [see [mechanisms](#), [multilevel](#)] the apparent causal relation between components and the system is bottom-up.

Bottom-up causation seems unproblematic, given the grand tradition of success in reductively explaining the behavior of systems in terms of the behavior of underlying components. Such explanations are especially important in explaining why two mechanisms respond differently (a neuron and a muscle cell) to the same causal input. Such explanations are commonly expressed in causal language: the operation of the part caused the behavior of the whole. The rise of systems biology, in which researchers sometimes appeal to the activities of the system to explain the operation of its parts, suggests to some that causation can also work top-down: interactions between the system/mechanism and its environment change the mechanism, and this in turn causes the parts of the mechanism to change (Noble 2006). Such explanations are clearly relevant to explaining why two essentially identical mechanisms (e.g., a cell *in vivo* and one in culture) behave differently depending on the causal context in which they operate.

2. Conceptualizing Interlevel Causation

Some people find the notion of interlevel causation mysterious and metaphysically suspect. Others find it utterly commonplace and unproblematic. What generates these different responses are different understandings of the terms *level* and *causal influence*.

Levels. Biologists use the term level in many ways (Craver 2007). On most uses, interlevel causation is unproblematic. Levels are sometimes defined by the *sciences, theories, and techniques* used to study them, as in the level of cell biology or the electrophysiological level. If one understands levels this way, interlevel causation is unproblematic. Causal influence among biological components is unaffected by who studies the components or by how they study and describe them. Levels are also sometimes defined by *size*, with bigger things at higher levels than smaller things. In that case, interlevel causation takes place whenever large and small things interact, e.g., when planets attract molecules into atmospheres or viruses infect elephants. Such interactions are again commonplace but uninteresting, as size differences, though relevant to how and whether many things interact, place no absolute restriction on whether two things can possibly influence one another. On these two understandings of *level*, there is no need to posit a separate relation, interlevel causation, where one relation, causation, would do just as well.

The issue of interlevel causation arises with levels within mechanisms. Levels of mechanisms are related as part to whole, where the behavior of the whole is what the mechanism does, and the behavior of the part is an activity or operation within the mechanism. For example, the circadian mechanism as a whole regulates the circadian functions of the organism, including sleep-wake cycles. In mammals, the responsible mechanism is located in the suprachiasmatic nucleus, which maintains endogenous rhythms and is required to maintain circadian functions in other organs. The endogenous rhythm is maintained within individual cells through a feedback loop involving the transcription and translation of genes into proteins which then inhibit subsequent transcription until they are degraded (Dunlap et al. 2004) The genes and proteins are parts of the mechanism, and through their activities realize a special kind of part-whole relationship in which the parts do work and are organized together in such a way that the mechanism as a whole exhibits its distinctive behavior. In levels of mechanisms, interlevel causation refers to the causal influence of a component on the behavior of a mechanism as a whole (bottom-up) or the causal influence of the behavior of the mechanism as a whole on the behavior of a component (top-down).

Causal Influence. The idea of causal influence can be understood in two ways: as causal relevance or as causal power. Roughly, a variable X is causally relevant to another variable Y if (under restrictions we neglect) one can change the value of Y by changing the value of X (see Woodward 2004). The value of Y depends on the value of X, and so the value of X makes a difference to the value of Y. Light is relevant to sleep/wake cycles in the sense that the duration of the sleep-wake cycle depends on environmental light/dark cycles. Controlled experiments test the causal relevance of one variable upon another by systematically varying the putative cause variable while fixing everything else in the system and measuring the difference, if any, that this variation produces in the effect variable.

Experiments of this sort are often used to investigate dependency relations between entities at different levels of mechanisms. Sometimes, one intervenes to alter, inhibit, or stimulate a component in a mechanism in order to investigate its contribution to the behavior of the mechanism as a whole. Genetic manipulations that produce mutations in genes, knock them out, or alter their regulation are often used to understand the role that genes play in higher-level mechanisms. Sometimes, one intervenes by activating a mechanism or changing its context and detects the consequences of that change on the activities and operations of its components.

Cognitive neuroscientists, for example, engage subjects in cognitive tasks while measuring indicators of neuronal activity in component regions of the brain. Such experimental methods are used to establish that a component is causally relevant to (makes a difference to) the behavior of the mechanism as a whole.

If one understands interlevel causation as a matter of causal relevance, it is not metaphysically mysterious. It is merely the commonplace fact that a mechanism sometimes behaves differently when one manipulates its parts, and that the parts sometimes behave differently when the mechanism is put in different contexts. This is a crucial background idea for much of the experimental work in biology. When causal influence is understood as the exercise of causal powers, however, the notion of interlevel causation becomes potentially problematic. On this construal, causal influence involves the direct exertion by an agent of a causal power over a patient. It is difficult to make this notion precise, but the idea is intuitively a matter of the agent's breaking, igniting, making, pushing, pulling, or repelling the patient. In some cases, a conserved quantity or a mark is transmitted from the agent to the patient (see Salmon 1984). When billiard balls collide, they exchange momentum. Animals consume energy when they eat. Other forms of causal power are not clearly cases of transmission but rather involve well-defined activities such as transcription, translation, or gravitational and magnetic attraction. Causal relevance between two variables merely indicates that some such causal powers link them.

Two features commonly associated with the notion of causal powers make it mysterious how it applies to phenomena at distinct levels of mechanisms. First, the agent and patient in such an interaction must be wholly distinct. Second, the agent and its relevant properties must exist or be present before the change is induced in the patient. Third, the agent and the patient must come into contact with one another, either directly or indirectly via wave-like proxy, if the causal influence is to be propagated from one to the other. These features of causal powers (rather than causal relevance) do not appear to hold when the agent and patient are at different mechanistic levels, which relate parts to wholes. The part and the whole are not wholly distinct. Any change introduced into the part is, ipso facto, a change to the whole. And there would appear to be no time for the causal influence to propagate from the change in the part to the change in the whole (violating the idea that causes must precede their effects) (see Kim 2005). These considerations largely account for visceral reaction of some against interlevel causation. One might simply deny that these features apply to all instances of causal production, but one must then say what a causal power amounts to beyond the notion of causal relevance.

Interlevel Causation: A Hybrid View

Appeals to interlevel causation, when causal influence is understood as the exertion of causal powers, is better understood on a hybrid view according to which the difference that things at one level make to the things at other levels can be analyzed in terms of two metaphysical relations: the exertion of causal powers among the components in a mechanism, and the constitution relationship between the behaviors of all of the organized components and the behavior of the mechanism as a whole. On this model, there is no causal power propagated between the part and the whole or between the whole and the part. Rather, the causal powers are propagated among the components in a mechanism which, taken together, constitute the higher level behavior in a hierarchy of mechanisms. In short, putative interlevel exertions of causal

powers are in fact hybrids of intralevel exertions of causal powers and interlevel constitutive relations (Craver and Bechtel 2007).

On this hybrid model, cases of bottom-up causation can be analyzed without loss of content as mechanistically-mediated effects. A change introduced into a component has downstream consequences for the behaviors of the other components which changes either the output of the mechanism as a whole or, alternatively, constitutes a change in how the mechanism as a whole behaves. Cases of top-down causation can also be analyzed without loss of content as mechanistically mediated effects. By changing the mechanistic context in which a given component behaves, one changes its inputs and outputs. When one alters the behavior of the mechanism as a whole, one changes the mechanistic context of the component. As a result, it changes how the component behaves. This is not the mysterious exertion of a higher-level power, but the ramification of causal powers through a mechanism.

The hybrid view is illustrated by recent developments in circadian rhythm research. The oscillatory period generated by the transcriptional feedback loops in individual cells in the suprachiasmatic nucleus is highly variable. The regular circadian behavior of the organism depends upon the interaction between cells through which they synchronize their behavior (Welsh et al. 2010). The interaction between cells is causally relevant to the behavior within cells, but the causal powers are all intralevel—the dispersal of a peptide between cells and the intracellular processes involved in the generation of the peptide and its uptake by recipient cells. Causal powers are exerted at different levels, but their efficacy on other levels is mediated by the constitution relation between levels.

Cross-references

Mechanism, multilevel

References

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