Chapter 6: Arguments from Correlation to Causation

Abstract: The contested notion of cause is centrally important for evaluating evidential reasoning in law and science, especially in so many cases where a causal conclusion if drawn from statistical correlations. Three current examples of arguing from correlation to causation that are based on scientific reasoning and that are of broad public concern as health issues are described and evaluated in chapter 6. The first one concerns the question of whether eating chocolate makes people smarter. The second one concerns a correlation between weather patterns in the southern Pacific and flu pandemics. The third one concerns the question of whether ingestion of copper causes Alzheimer's disease. This chapter shows how to improve the existing argumentation tools to enable us to judge whether a given instance is a reasonable argument or not and how to adjudicate cases where an allegation of drawing a hasty conclusion or even committing a fallacy is made.

This chapter argues that arguments from correlation to causation have to be studied at two levels. One is the inferential level of the argumentation scheme representing the structure of the argument as leading to a conclusion of a certain type based on premises of a certain type. The other is the dialectical level, which concerns the context of use of the argument. It will be shown that arguments from correlation to cause are initially scientific arguments used to collect evidence and draw conclusions in an investigation, but then they are also used both by scientists and non-scientists for all kinds of purposes. For example they may be used in medicine to give advice on treatment decisions or to set public policies.

In section 1, three current examples of arguing from correlation to causation that are based on scientific reasoning and that are of broad public concern as health issues are described. The first one concerns the question about nutrition, the second one is about weather patterns and flu pandemics, and the third one is about whether ingestion of copper causes Alzheimer's disease. The chapter is built around analyzing the arguments from correlation to cause in these (and two other) examples. The main tool that has been used in the literature for this purpose is the argumentation scheme representing the form of argument from correlation to cause. In section 2 some accounts are given of this argumentation scheme in informal logic textbooks, and the notions of correlation are defined for the purposes of this chapter. Needless to say, the notion of causation is highly controversial, so these definitions are provisional. It is also shown how CAS uses this scheme. In section 3 nine conditions for drawing a scientific inference from causation to cause, called the Bradford Hill Criteria, are explained and summarized. Section 4 explains how heuristic devices are used in order to argue from correlation to cause in cases where the ultimate endpoint of a causal investigation may be difficult or impossible to measure. For example if a cancer treatment is shown to shrink tumors, the shrinking of the tumors may be taken as a surrogate marker to stand in for the ultimate endpoint, which is the extension of life. Section 5 compares how the argumentation in the three examples stand up to analysis, and ranges the arguments in an ordering of strength and weakness. Section 6 shows how the need to examine arguments from correlation to cause a dialectical level that takes context into account, as shown by the examples in sections 7 and 8. Sections 7 and 8 present two more examples. One is the classic case of proving the causal link between smoking and lung cancer. The other is the currently controversial issue of the causal link between playing football and brain damage. Section 9 shows how to improve the current method of evaluating arguments from correlation to cause by reformulating the set of critical questions matching the argumentation scheme for argument from correlation to cause. Section 10 offers a solution to the problem of analyzing the *post hoc* fallacy.

1. Three Current Examples

The first of the three examples is called the chocolate example. Messerli (2012) conducted a statistical survey to support the hypothesis that chocolate consumption can improve cognitive function, and can even be effective in slowing down the reduction of cognitive performance that occurs with aging. He found there was a correlation between a country's level of chocolate consumption and cognitive function of the population. His starting point was the following assumption: "the total number of Nobel laureates per capita could serve as a surrogate endpoint reflecting the proportion with superior cognitive function and thereby give us some measure of the overall cognitive function of a given country" (Messerli, 2012, 1562). One basis for the study was the list of countries ranked in order of Nobel laureates per capita found on Wikipedia, and another was a Swiss source that had data available on chocolate consumption in 23 countries.

It was found that there was a close significant linear correlation between chocolate consumption per capita and the number of Nobel laureates per 10 million persons in these countries (Messerli, 2012, 1563). Messerli added the qualification (1563) that even such a statistically strong correlation as this one does not prove causation, but he argued that it seems likely that "chocolate intake provides the abundant and fertile ground needed for the sprouting of Nobel laureates"(1563). He also considered the possibility of reverse causation, referring to the possibility that persons with superior cognitive function are more aware of health benefits of dark chocolate. But he decided to exclude this hypothesis (1563) because it seems unlikely that receiving the Nobel Prize "would in itself increase chocolate intake countrywide". He also had some theory-based biochemical basis to support his conclusion about the causal link: substances called flavonoids, present in cocoa, are known to improve blood flow in the brain, leading to improved cognitive performance in rats and humans.

The second example, called the birds example, can be described as follows. It was found by scientists that the four most recent human influenza pandemics, which took place in the years 1918, 1957, 1968, and 2009, occurred just after La Niña events that brought cool water to the surface in the South Pacific. La Niña is the cold counterpart to El Niño. The two events make up the weather variation that is called the El Niño southern oscillation. The correlation between these La Niña events and flu pandemics might suggest that there could be a causal link between them. But more likely it would suggest the conclusion that the two events happening one after the other in these four instances is merely a coincidence, and also the conclusion that to think otherwise would be an instance of the *post hoc* fallacy.

Nevertheless consideration of some other circumstances suggests that there could be three intervening variables connecting the two events. First, pandemics are caused by exposure to people of novel strains of a virus to which they have not developed immunity. Second, such a flu virus can be introduced to humans by animals, for example birds or pigs. Third, patterns of flight stopovers during bird migrations are affected by El Niño and La Niña events. And fourth, a change such as a La Niña event could easily cause birds to mingle together that do not otherwise mix. These intervening connections easily suggest how a virus causing a pandemic could be transmitted from birds to humans after a La Niña event.

Shaman and Lipsitch (2013) suggested that in light of this evidence a causal connection can be drawn: "We hypothesize that La Niña conditions bring divergent influenza subtypes together in some parts of the world and favor the reassortment of influenza through simultaneous multiple infection of individual hosts and the generation of novel pandemic strains". They tested this

hypothesis by using evidence from influenza population genetics, virus prevalence in various host species, and avian migration patterns. They concluded (2013, 3690) that their findings indicate "a possible association between the emergence of pandemic influenza" during southern oscillation events such as La Niña. But they were careful to add that whether the association between the two events they found is causal or merely coincidental has not yet been established. They also added the conclusion that "the most plausible biological explanation" for the association between the four La Niña events and the four pandemic influenza events involves climate change shifts in bird migration patterns.

The third example is called the copper example. Many news media reports concern a study in the proceedings of the *National Academy of Sciences* that drew the conclusion that ingestion of copper can lead to plaque buildup in the brain causing Alzheimer's disease (Singh et al., 2013). Alzheimer's disease is caused by the accumulation of a protein in the brain called amyloid beta. The study purported to show that the ingestion of copper has a cumulative effect of impairing the systems by which amyloid beta is removed from the brain. Copper is necessary for human health, and is found in meat, fruits and vegetables, as well as drinking water that often flows through copper pipes. Lead researcher Dr. Rashid Deane was reported in a University of Rochester Public Release as saying: "It is clear that, over time, copper's cumulative effect is to impair the systems by which amyloid beta is removed from the brain".[1] In the experiment conducted by Deane and his colleagues, copper-laced drinking water was given to mice for three months. Their theory is that the copper made its ways into the walls of capillaries that protect the brain from toxins, ultimately breaking down the blood brain barrier that prevents harmful substances from entering the brain. The resulting buildup of plaque was described as a "one-two punch" which both stimulates the production of amyloid beta and inhibits its clearance.[2] The research concluded that there is a causal connection between too much copper consumption and the creation of "logjams of the protein that the brain's waste disposal system cannot clear", the plaque buildup taken to be the cause of Alzheimer's disease.

An especially interesting aspect of this research finding is that other experts strongly disagreed with it. Christopher Exley, a professor of bioinorganic chemistry at Keele University, published a chapter (Exley et al., 2012) claiming that his team's research on brain tissues reached the opposite conclusion. Exley commented in a news interview on this research[3] by flatly disagreeing with its conclusion: "Research including our own shows the opposite, that copper prevents amyloid from forming the type of structures seen in the plaques". He based this claim on a chapter written by him and his colleagues in *Nature*. He said that according to everything his research group knows, based on their work with brain tissues, if anything, copper would be protective against Alzheimer's.

2. Current Versions of the Argumentation Scheme

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[1] James Gallagher, Copper Linked to Alzheimer's Disease, BBC News, Accessed August 21, 2013: http://www.bbc.co.uk/news/health-23755037

[2] Mark Michaud, University of Rochester Public Release date Aug. 19, 2013. Copper Identified as Culprit in Alzheimer's Disease, Accessed Aug. 21, 2013. http://www.eurekalert.org/pub_releases/2013-08/uorm-cia081413.php

[3] Deccan Chronicle (no author given). Copper linked to Alzheimer's? New Research Fuels Debate: http://www.deccanchronicle.com/130820/lifestyle-health-and-well-being/article/copper-linked-alzheimers-new-research-fuels-debate Accessed Wednesday, Aug 21, 2013

Johnson and Blair (1983, 121) deal with fallacies such as the *post hoc* fallacy under the general category of arguments that make a causal claim. They define a more general kind of fallacy they call the fallacy of questionable cause, which has three identifying conditions. The first is that a causal claim appears in a proponent's argument. The second is that the proponent argues for the causal claim, but fails to provide adequate support for it. The third is that there are grounds for questioning the acceptability of the claim. This approach has two aspects that are especially interesting to note. One is that it links the failure of a causal argument with critical questions that express doubt about the acceptability of the causal claim, based on lack of supporting evidence. The other is it sees cases of fallacies of arguing to support a causal claim as being weak and questionable sorts of arguments that could in principle be supported by evidence, but where in fact insufficient evidence is given to support the claim. This approach suggests that what is objectionable about such arguments, when they are said to be fallacious, or logically defective, is not their inherent wrongness, but the overlooking of factors that do not address critical questions that can be raised.

Govier (2005, 343) also does not see arguing from correlation to cause as fallacious, and to emphasize the point, she renames the error as the fallacy of objectionable cause. She reconfigures the form of inference corresponding to this fallacy as an argument with three premises and a conclusion of the following form, where *A* and *B* represent events.

A occurred.

B occurred.

We can plausibly connect *A* to *B* in a causal relationship.

Therefore *A* caused *B*.

Her diagnosis of the problem with this kind of argument is that there is no basis given for ruling out alternative factors that might explain the connection between *A* and *B* as being something other than causal in nature.

Groarke and Tindale (2004, 305) reconfigure the *post hoc* fallacy by emphasizing that arguments from correlation to cause need to be evaluated in light of critical questions or counter-arguments that reveal how the argument might violate conditions for good causal reasoning. On their account, such critical questions and potential counterarguments can be built into additional premises that needed to be added to the simple argument from correlation and causation. They propose (2004, 303) the following scheme for general causal reasoning, where *X* and *Y* are causal variables for events of the kind that fit into a causal relation.

Premise 1: *X* is correlated with *Y*.

Premise 2: the correlation between *X* and *Y* is not due to chance.

Premise 3: the correlation between *X* and *Y* is not due to some mutual cause *Z*.

Premise 4: *Y* is not the cause of *X*.

Conclusion: *X* causes *Y*.

They also emphasize that the two events *X* and *Y* need to be regularly connected by showing that there is a general theory (normally based on the work of scientists) who have shown there is some deeper understanding of the chain of events connecting *X* and *Y*.

Pinto (1995) provided a useful summary of the textbook treatments of the *post hoc* fallacy stressing that a careful distinction needs to be drawn between particular events and types of events that figure in causal generalizations. Pinto (1995, 309) gave the example of a child bouncing a rubber ball against her bedroom wall when a hairline crack immediately appeared in the plaster of the wall in the exact spot where the ball struck. In this case, we have two particular events, the child's bouncing the rubber ball against her bedroom wall and the appearing of the

crack in the plaster immediately afterwards. Statements about types of events frequently make up generalizations. For example, the statement that smoking causes lung cancer is a generalization that applies not just to one particular case where an individual person smoked and later developed lung cancer.

Next we need to define the notions of correlation and causation as they will be used in conjunction with argumentation schemes. A *correlation* between two (or more) events is an instance (or number of them) where both (or all) events occur. Correlation is a statistical notion, because the number of cases in which events occur can be counted, and these numbers can then be used as the basis for statistical calculations. Notice that on this definition a correlation can be between two events, or types of events or it can be among a sequence of events, or types of events. For example there could be a correlation between weather events in the South Pacific and flu pandemics. But there can also be a correlation among a sequence of events such as weather events in the South Pacific, bird migration patterns and flu pandemics.

Causation, as the term is used in this chapter is a practical notion, not a purely statistical notion. For the purposes of argumentation schemes, event *A* is said to cause another event *B* where the occurrence of *B* can be inferred as a normal outcome of the occurring of *A* based on the evidence in a case. On this definition, causation is a field-dependent notion. A *field* is a stable environment that can be presumed to be constant, but not completely identical, from one case to another. Causation, when defined in this way, is a defeasible evidential relation between events. The statement that *A* causes *B* means that *B* can be inferred from the evidence in a constellation of factors including *A*. A *cause* is defined as a set of conditions that are individually necessary for the occurrence of an event, and taken together are sufficient (*ceteris paribus*) for the occurrence of that event. Consider the event that a particular warehouse burned down, where there is evidence that just before the fire Bernie, a known arsonist, was seen leaving the location of the warehouse by a witness, who also noticed that Bernie was carrying a container that smelled like gasoline. In this case, the police formulate a hypothesis on which to base their investigations. This hypothesis is the statement that Bernie set the fire. The cause of the fire, according to this hypothesis, was Bernie's use of gasoline to start the fire. Such a case can be structured as an argument graph in formal argumentation systems, where the events are propositions and the causal inferences are argument nodes.

Schemes are now being incorporated into software systems that can be used for argument identification, analysis, diagramming, evaluation and construction (argument invention), for example CAS[4]. A user can also select argumentation schemes from a menu and use them to analyze and evaluate arguments, as well as to search through the database for new arguments to prove a claim. CAS is a mathematical model of argumentation (Gordon, 2010) that has an Open Source argument mapping graphical user interface available at no cost to users. The current version of CAS is a web application with a three-tiered architecture consisting of a database, an applicable logic and a graphical user interface. The previous version of CAS, a desktop application called the Carneades Editor, is still available. The current version has a catalogue of argumentation schemes including such forms of argument as argument from expert opinion, argument from testimony, argument from analogy, argument from precedent, practical reasoning, and argument from correlation to cause.

CAS models critical questions by drawing a distinction between two kinds of premises in an argumentation scheme, assumptions and exceptions. The premises of the scheme that are explicitly stated are treated as assumptions, meaning that they are taken to hold unless they are

[4] The current version of the CAS editor can be downloaded from http://carneades.github.com/

challenged, but if they are challenged the arguer has to back up the premise with some evidence, or the argument fails. But there are other assumptions in addition to the ordinary stated ones. The kind of premise that represents an exception is taken to remain acceptable even when the question is posed. The current version of the scheme for argument from correlation to cause is given below.

id: correlation-to-cause
strict: false
direction: pro
conclusion: Event *E1* causes event *E2*.
premises:

- Events *E1* and *E2* are correlated.

assumptions:

- There exists a theory explaining how event *E1* causes event *E2*.

exceptions:

- Event *E3* causes events *E1* and *E2*.

The ordinary premise, the statement that *E1* and *E2* are correlated, is taken to hold, but if questioned, it is no longer accepted. The same criterion applies to the assumption that there exists a theory explaining how event *E1* causes event *E2*. But the exception is treated differently. Merely asking the question is not enough to make the premise no longer accepted. A specific event *E3* has to be cited in order for that to happen. In other words, we could say that with assumptions, the burden of proof is on the proponent of the argument whereas with exceptions the burden of proof is on the opponent.

The version of the scheme given in (Walton, 1996, p. 142) is very simple. It has only one premise and one conclusion.

Premise: There is a positive correlation between *A* and *B.*

Conclusion: Therefore *A* causes *B.*

However, the same scheme can be more fully expressed as a form of argument with two premises.

Premise 1: If there is a positive correlation between *A* and *B* then *A* causes *B.*

Premise 2: There is a positive correlation between *A* and *B.*

Conclusion: Therefore *A* causes *B.*

This version of the scheme has the so-called DMP (defeasible *modus ponens*) format. This form of argument is treated in (Walton, 1996) as defeasible and presumptive, meaning that commitment to the premise only gives a reason for commitment to the conclusion that may default when one of the appropriate critical questions is asked.

Matching the argument from correlation to cause is the following set of seven critical questions (Walton, 1996, 142-143).

CQ1. Is there a positive correlation between *E1* and *E2*?
CQ2. Are there a significant number of instances of the positive correlation between *E1* and *E2*?
CQ3. Is there good evidence that the causal relationship goes from *E1* to *E2* and not just from *E2* to *E1*?
CQ4. Can it be ruled out that the correlation between *E1* and *E2* is accounted for by some third factor *E3* (a common cause) that causes both *E1* and *E2*?

CQ5. If there are intervening variables, can it be shown that the causal relationship between *E1* and *E2* is indirect (mediated through other causes)?
CQ6. If the correlation fails to hold outside a certain range of cases, then can the limits of the range be clearly indicated?
CQ7. Can it be shown that the increase or change in *E2* is not solely due to the way *E2* is defined, the way entities are classified as belonging to of *E2*, or changing standards, over time, in the way *E2* is defined or classified?

If the proponent puts forward an argument that fits the scheme for argument from correlation to cause, and the respondent accepts the premises of the argument, then she is also taken to accept the conclusion unless she can ask a critical question or provide a counter-argument.

As an example of critical question 3, we can consider this sort of case (Freedman, 2010, 59). Many studies have shown that people who exercise more tend to be generally more healthy. The conclusion suggested is that exercise as a way to improve your health. But the question is which way the causal relationship goes. Is the exercise the cause of the good health, or is a person's good health a causal factor in making him or her more likely to exercise?

As an example of critical question 4, we can consider this sort of case (Freedman, 2010, 57). On the grounds of the correlation between lack of sleep and obesity, it has been claimed that if someone starts getting more sleep they will lose weight. But there could be intermediate factors, rather than sleep levels, that produce, or are involved in the outcome of obesity. It may be that those who sleep less tend to be those who exercise less, eat less healthy foods, have a hormone disorder, or are depressed. It could be any of these other factors, or some combination of them that affects obesity. So it may be that once these other variables are taken into account, the connection between lack of sleep and obesity is merely incidental. Hence jumping to the conclusion that the lack of sleep is causing the obesity needs to be recognized as an instance of *post hoc* reasoning that is questionable at best.

Broadly speaking, what is common to these accounts of the argumentation scheme for argument from correlation to cause is that the scheme can be seen as taking a simple form with only one or two premises, or it can be seen as a more complex scheme that features a range of additional premises. There is variation on how many critical questions or additional premises there should be. The main theoretical difference among the various accounts of the scheme is whether these additional features are seen as critical questions or as additional premises. CAS deals with this difference in a systematic way by treating the critical questions as additional premises. On this way of managing the critical questions, you can see the scheme as having two additional kinds of premises, called assumptions and exceptions, in addition to the regular premises.

3. The Bradford Hill Criteria

The following nine conditions for drawing a scientific inference from causation to causality (Susser, 1977; Doll, 1992) are called the Bradford Hill Criteria (Hill, 1965).

1. **Temporality**. The cause is supposed to precede the effect.
2. **Strength and Association**. The causal conclusion is derived by observations of a statistical correlation between a pair of events. The strength of the correlation can be measured

numerically. The principle of measurement of the causal inference is that the stronger is the correlation between the two events, the stronger is the inference that the one causes the other.
3. **Dose-response Gradient**. There is expected to be a relationship between cause (the dose given, in a clinical case) and the effect (the reaction of the patient). The relationship may be expected to have minimal and maximum thresholds.
4. **Consistency**. The likelihood of the causal hypothesis holding is increased with its giving consistent results in a wider range of circumstances.
5. **Theoretical Possibility**. There should be a greater acceptance of an association between two events as causal when there is a theoretical scientific basis linking the two events.
6. **Specificity**. There can be multiple causes of a given event, and one suspected cause can be stronger than another if it better explains the occurrence of the event. Also a causal hypothesis can be strongest when there is no plausible competing explanation for the event.
7. **Evidence**. Research based on experiments, where other variables can be held stable to prevent them from interfering with results, will make a causal inference more plausible.
8. **Analogy**. A causal hypothesis that has been identified can be held more strongly when other supposed causes analogous to it have been identified and eliminated from the investigation.
9. **Coherence**. Coherence is defined as an alignment between the findings of scientific experiments in which variables are controlled, and independent everyday evidence based on common knowledge of events we are familiar with in our practical experiences.

Clearly there are a number of close similarities between the set of critical questions matching this scheme for argument from correlation cause and the Bradford Hill Criteria. Critical question 2 concerned the significance of the number of instances of the correlation, while Bradford Hill Criterion 2 takes this consideration further by stating the principle of measurement that when the correlation is stronger, the inference to the causal conclusion also becomes stronger. Bradford Hill Criterion 3 states another aspect relating to measurement of degree: when the cause is stronger the effect is expected to be stronger as well. Critical question 3, which concerns the directionality of the two events, relates to Bradford Hill Criterion 1, stating that the cause is supposed to precede the effect.

One factor that is suggested to be very important by the examples introduced above and analyzed below is the Bradford Criterion 5, which states that the causal inference is stronger when there is a theoretical scientific basis linking the two events. This particular factor was stressed in the scheme for argument from correlation to cause currently in the category of schemes in CAS (see above). Hence it is a recommendation that a critical question corresponding to this factor could be added to the list of seven critical questions of (Walton, 1996), the leading list that occurs elsewhere in the literature including (Walton, Reed and Macagno, 2008).

4. Surrogate Markers

Reasoning from cause to effect is typical in scientific investigations and clinical trials. For example in an investigation to determine whether a particular medication is effective for reducing heart attack or stroke, a clinical trial is carried out in which the effects of giving one group of patients the medication are compared with the effects of giving a placebo to another group of patients. The effect or so-called ultimate endpoint of such a causal investigation is the death of the patient by heart attack or stroke, for example. The causal factor being studied may

be the taking of a particular medication. The purpose of the investigation is to delay the morbid outcome of shortening of life expectancy by heart attack or stroke.

Typically, however, there may be practical reasons why it is difficult or costly to measure the ultimate endpoint, and for this reason something called a surrogate marker is used to substitute for that endpoint. In clinical trials, a surrogate marker, sometimes also called a proxy measurement, is a measure of the effect of a certain treatment that is taken by inference to cause the ultimate clinical endpoint, but the inferential step from the cause to the endpoint is defeasible, and can be subject to critical questioning. For example, in clinical trials to study the causes of events such as heart failure and stroke, vascular disease is the most common cause (Cohn, 2004). Vascular disease progresses through a mechanism that includes inflammation, plaque formation and thrombosis, and new technology makes it possible to track the progression of the sequence by using surrogate markers: "the availability of reliable markers for the disease might ultimately allow disease progression to replace endpoint events as a guide to the risk of disease and its responses to therapy" (Cohn, 2004, 20). Such surrogate markers are used for practical reasons, to achieve results more rapidly and at less cost.

Surrogate markers are divided into two subtypes by Cohn (2004, 20), structural and functional surrogate markers. Cohn uses the diagnosis of cardiovascular disease as an example. Structural abnormalities of the arteries or the heart are considered to be structural surrogate markers of cardiovascular disease. Measurements of carotid artery wall thickness and left ventricular mass identify these two structural surrogate markers. They are taken to indicate cardiovascular disease of a kind that would be expected to worsen over the course of time. Functional surrogate markers are described as imperfect markers for the structure of cardiovascular disease. The presence of some of them may be expected to reduce the risk of the morbid event, "but with others it is less certain that the response of the surrogate is a prerequisite for the benefit on the disease process."(Cohn, 2004, 20). Examples of functional surrogate markers for cardiovascular disease are blood pressure and arterial wall compliance or stiffness.

These descriptions of this example raise some questions about the inferential link that should properly be required between the surrogate marker and the endpoint. What is the nature of this inferential link? And how strong does it have to be in order for something to be considered a valid surrogate of an endpoint? Is it enough that there should be a correlation between the two events? Or should it be required that the surrogate marker causes the endpoint? And if the latter, how strong does the causal relationship need to be? Could it be merely a contributory cause, or is some stronger type of causal connection required? For example, there might be a correlation between high blood pressure and cardiovascular endpoints such as heart attack or stroke, but it might be questionable to say that either of these two indicators by themselves can be taken as surrogate markers of fatal cardiovascular events such as heart attack or stroke. High blood pressure might be a causal factor in heart attack and stroke, but that might not be a solid enough reason to treat it as a surrogate marker of these outcomes for purposes of clinical investigations.

It would appear that the connection between the two points in the sequence of reasoning must be stronger. But what requirements precisely should be used to define the criteria for a surrogate marker, and to determine how strong the inference needs to be to the final outcome in order for something to properly qualify as a surrogate marker? One such criterion might be that to properly justify some factor being used as a surrogate marker, the effect of carrying out the event taken to be the surrogate must cause the effect taken to be the endpoint. Another might be the sort of causal link whereby a significant increase in the presence of the surrogate marker is needed to increase the likelihood of the event taken to be the endpoint.

Consider the example where the endpoint is death from heart failure but the surrogate marker is increasing cholesterol levels. Increase an cholesterol level increases the likelihood of heart failure, but on the other hand there are many people who have normal cholesterol levels who go on to have heart failure, and also many who have high cholesterol levels who do not. So if a clinical trial shows that taking a particular drug has the outcome of reducing cholesterol, that is an interesting finding, but it does not show that taking this drug will prevent heart failure. On this basis, it would not be logically justified to treat high cholesterol as a surrogate marker of heart failure. There are cases where something was initially considered to be a surrogate marker for a potentially deadly endpoint, but where this hypothesis had to be retracted.

The drug bevacizumab, which goes by the trade name of Avastin, is an antibody that promotes shrinkage of cancerous tumors by slowing the growth of new blood vessels. Avastin was approved for treating several kinds of cancers by the FDA in 2004, and was approved for treatment of breast cancer in 2011. Although the drug did shrink cancer tumors, its approval by the FDA for the treatment of breast cancer was revoked on the grounds that there was no evidence that it extended life or quality-of-life (Couzin-Frankel et al., 2011). Moreover, there was evidence that it caused severe high blood pressure and hemorrhaging. In this case the surrogate marker was the shrinking of a cancer tumor, and the ultimate endpoint was extended life for the cancer patient. Here we have an instance of defeasible reasoning. Because Avastin was shown to shrink tumors, and tumor shrinkage was taken to be a surrogate marker of extension of life, it was concluded that Avastin would extend life. When further evidence came in showing that this surrogate marker did not connect up in the right way with the ultimate endpoint, the causal reasoning had to be retracted.

The Avastin example, and the other examples studied in the chapter, especially illustrating the dangers of the use of surrogate markers and drawing inferences from correlation to cause, show how important the critical questions are in analyzing and evaluating this type of argumentation. It would appear that is typical of statistical evidence used to argue from correlation because it depends on the use of such surrogate markers. What is typically concealed, or at least not strongly enough emphasized in such cases is that the proper evaluation of the argument from correlation to cause depends crucially on an inference from a surrogate marker to the ultimate conclusion that is taken to be the endpoint of the inference. The structure of such a causal inference as illustrated by the Avastin example, is shown in figure 6.1.

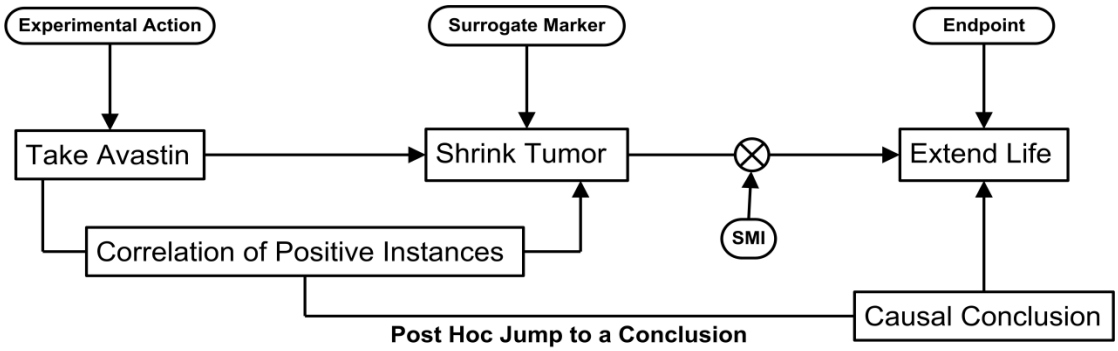

Figure 6.1: Structure of the Inference from a Surrogate Marker to a Causal Conclusion

In this example the experimental action was the recommendation to take the drug Avastin as a treatment for cancer, based on the experimental finding that taking Avastin had been shown in

the past to shrink tumors. But tumor shrinkage was taken as a surrogate marker for the intended effect of treatment which is to extend the patient's life. Further research showed, however, that the shrinking of the tumor in such cases did not have the desired effect of extending life.

The implicit but questionable inference in such a case as shown in figure 6.1 is what is called the SMI, or surrogate marker inference, referring to the inference from the surrogate marker to the endpoint. Relating to the SMI, two critical questions concerning the use of surrogate markers, shown to be important in the examples studied in the chapter, need to be added to the existing set. The first question is whether the argument from correlation to cause as stated goes to the ultimate conclusion or whether instead it goes to a surrogate marker. The second question to be asked is when the surrogate marker has been identified. This question asks whether the inference from the surrogate marker to the conclusion is justified.

5. Comparison of Examples

The argumentation in the chocolate example relates to the critical questions from the list of (Walton, 1996) in interesting ways. The first two critical questions are answered, because according to Mersserli's data, the statistical correlation between consumption of chocolate and Nobel Prize winners in countries where the Nobel Prize had been awarded were quite high. Pertaining to the third critical question, it was noted in the description of the chocolate example above that Messerli considered the possibility of reverse causation, but decided to exclude it. This consideration relates to the third critical question, which asks about the causal relationship going the other way.

Consideration of some of the other critical questions emerged during a discussion of the chocolate example with other scientists reported in the BBC News[5]. It was pointed out that Switzerland had the highest chocolate consumption per person and also the highest number of Nobel laureates of all the countries. However, Sweden appeared to be a counterexample to the causal relationship. Although it had a very high number of Nobel laureates, its people consume much less chocolate than the average of the other countries. Two explanations for this anomaly were offered by Mersserli. One was that since the Nobel Prize is evaluated in Sweden, "the Swedes might have a slightly patriotic bias". Let's break this sequence of argumentation down into two stages to see how CAS models its structure.

The pro argument shown at the top of figure 6.2 has two premises that are highly plausible. One is that there is a high correlation between chocolate consumption and Nobel Prize winners in countries. As noted above, Messerli found data that showed that this correlation was extremely high. The other is a proposition that winning a Nobel Prize can be taken as a surrogate marker of superior cognitive function. This too is a proposition that is highly plausible. Therefore in CAS the audience would accept these two propositions, and hence they are shown in figure 6.2 in green boxes. Assuming the top argument fits the scheme for argument from correlation to cause, the argument is defeasibly valid. On this basis, the conclusion that consumption of chocolate causes superior cognitive function is automatically calculated by CAS as accepted. Hence it is shown in a green box in figure 6.2.

[5] Charlotte Prichard, Does Chocolate Make You Clever?, *BBC News*, Nov. 19, 2012: http://www.bbc.co.uk/news/magazine-20356613

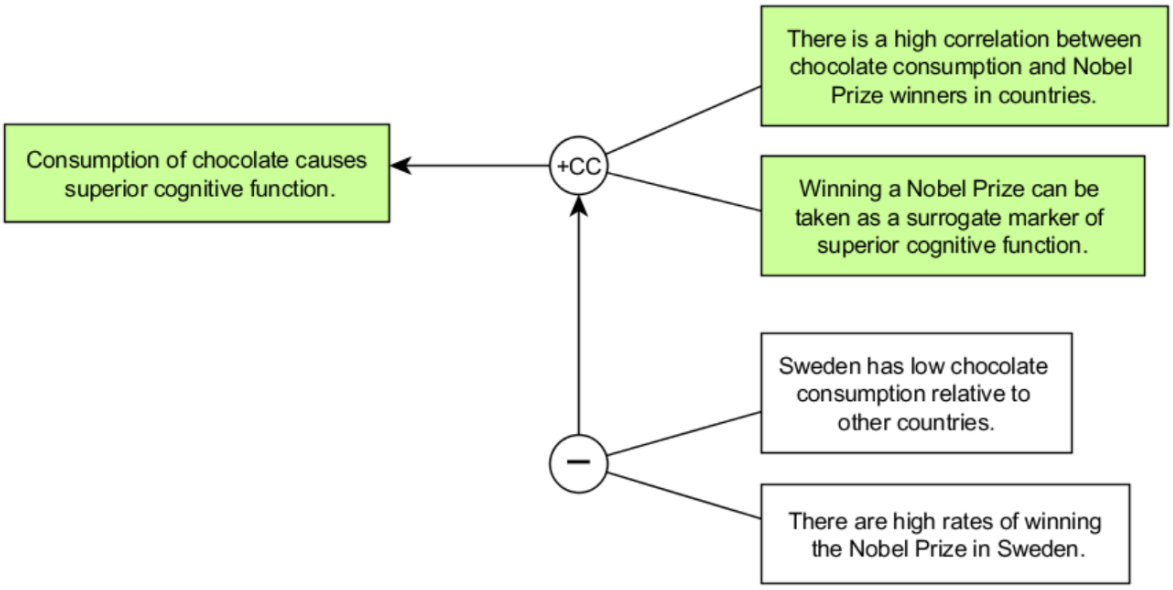

Figure 6.2: First Stage of the Chocolate Argument

However, now let's examine the con argument at the bottom of figure 6.2. This argument puts forward the counterexample of the case of Sweden, a country that has low chocolate consumption but high rates of winning the Nobel Prize. This argument would undercut the argument from correlation to cause if both its premises were to be accepted. What would happen in this instance?

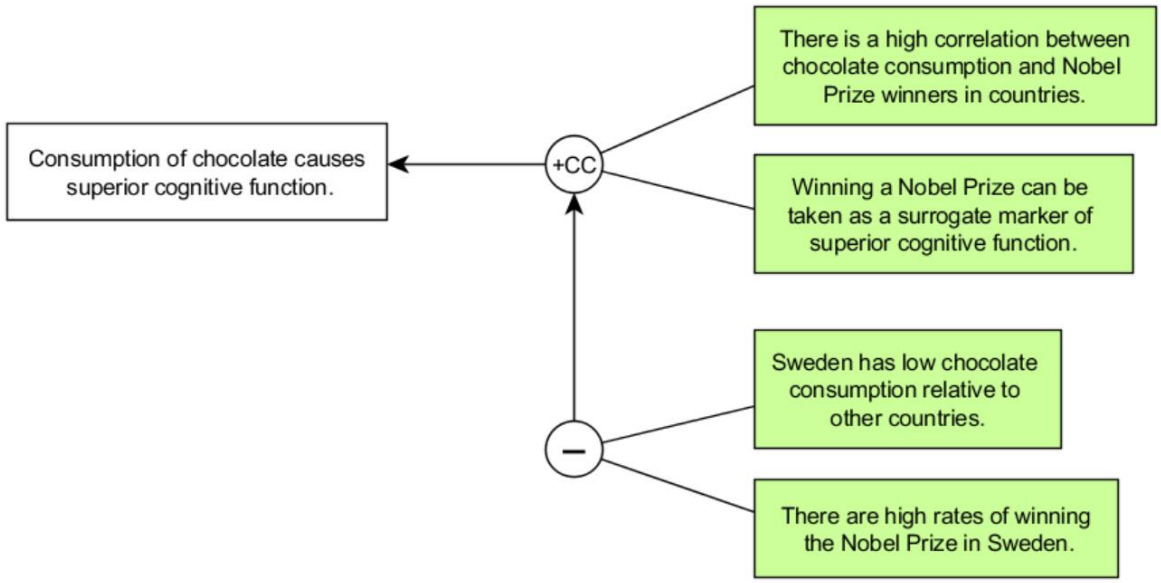

Figure 6.3: Second Stage of the Chocolate Argument

If both premises in the lower argument were to be accepted, and it is accepted that the lower argument licenses the transfer of acceptance from its premises to its conclusion, the lower argument would function as an undercutter to the argument from correlation to cause shown above it in figure 6.2. What happens now? What happens is that the conclusion of the argument, the statement that consumption of chocolate causes superior cognitive function, is no longer accepted. This situation is shown in figure 6.3.

The argument at the bottom cites the exception of the case of Sweden, and its node is shown in a grey box containing a minus sign. Assuming that both premises of this argument are plausible, the argument acts as an undercutting rebuttal of the previous pro argument. So represented, it attacks and defeats the previous argument. Hence on balance, the conclusion of the argument, the statement that consumption of chocolate causes superior cognitive function, is no longer taken by CAS to be accepted. Hence it is now shown in a white box.

In this instance the low chocolate consumption in Sweden is being used as a rebuttal to the argument from correlation to causation. The argument has two stages. Since the pro argument shown in figure 6.2 at the top has both premises accepted, and since it is a defeasibly valid argument, its conclusion is shown in a grey box. But once the rebutting argument at the bottom is put forward, as shown in figure 6.3, the conclusion at the left now appears in a white box, showing that it is no longer accepted.

Now let's look at figure 6.4, which shows the third stage of the chocolate argument. This argument has the same premises and conclusions as the one shown in figure 6.3, except that three new arguments have been added. The first argument has the premise that the Swedes might have a bias. This premise is shown as being supported by two further arguments. This statement is shown as a con argument attacking the con argument above it.

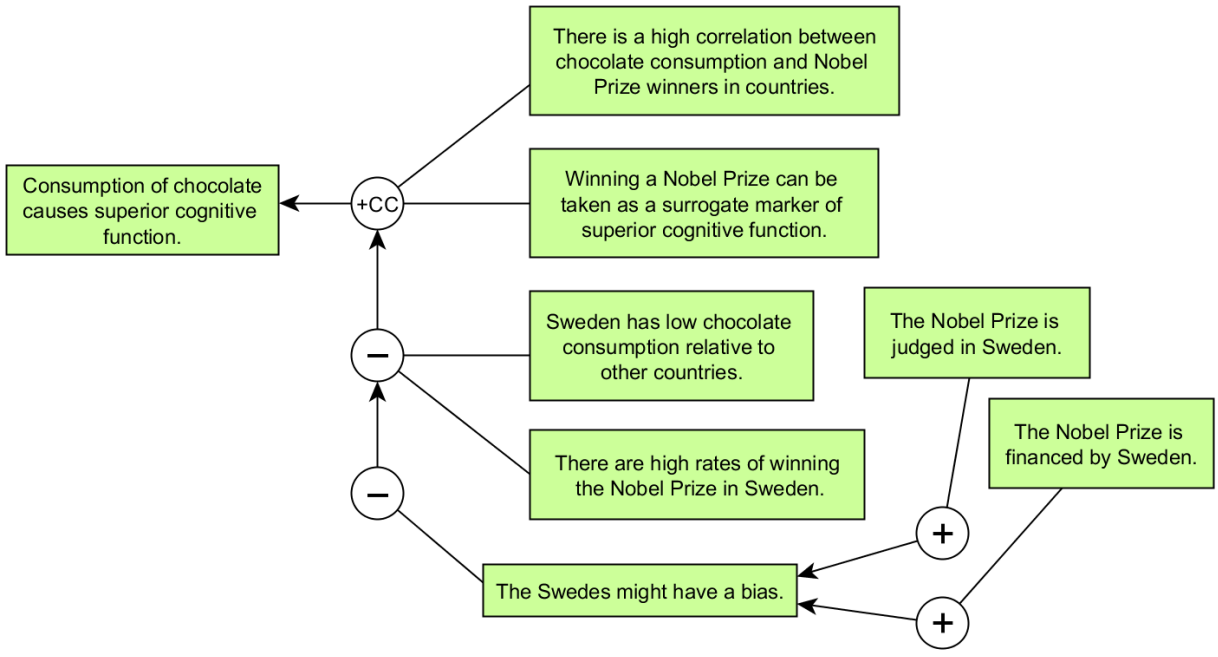

Figure 6.4: Third Stage of the Chocolate Argument

An exception in CAS is taken to be a premise that holds only if it is supported by backup evidence. In this instance the statement that the Swedes might have a bias is supported by the statement that the Nobel Prize is judged and financed in Sweden. This evidence is represented in figure 6.4 as two separate arguments. Therefore CAS evaluates the argument by ruling that the exception defeats the con argument. In this case what is displayed is another undercutting of a rebuttal. The undercutter defeats the Swedish argument counter-example rebuttal, which no longer proves that the ultimate conclusion is false. And for this reason the original argument displayed at the top of figure 6.4 is sufficient to warrant the acceptance of the ultimate conclusion that consumption of chocolate causes superior cognitive function, even though the premises of its undercutter are accepted.

There are two critical questions that should also be considered in evaluating this argument. First, there is the question of how we know whether the Nobel Prize winners are enthusiastic eaters of chocolate. Presumably, the basis for this assumption is that the Nobel Prize winners can be treated as a statistical sample that are comparable in regard to their chocolate eating habits with other people in their respective countries. This might not be so. For example, it might be true, as a matter of fact, that Nobel Prize winners eat less chocolate than other people in their countries. Still, it seems a reasonable statistical assumption that the Nobel Prize winners are on par with the others in their countries when it comes to eating chocolate. The second critical question is whether being a Nobel Prize winner can be taken as a surrogate marker for possessing the property of having a high cognitive function. However, I don't think the audience would question this assumption, because of the high regard we have for the award of the Nobel Prize, taken to be a prize that is only awarded to the brightest thinkers. This assumption could certainly be critically questioned, but it seems persuasive in the absence of evidence to the contrary.

Next, let's consider the birds example. At first sight, using the correlation between the four La Niña events and the four most recent flu pandemics to suggest the conclusion that there is a causal connection between these two events seems ridiculous. There are only four events involved, it is hard to see how there could be any causal connection between weather events in the Pacific and these four events. The best conclusion is that it is simply a coincidence. Once attention has been drawn to the three intervening variables connecting the two events however, the hypothesis that there is a causal connection between the two events becomes much more plausible. First we need to be aware that flu viruses of the kinds that caused the pandemics can be transmitted from birds or other animals to humans. Second, we need to see how these weather events influence patterns of flight stopovers during bird migrations, and how that could lead to birds mingling together that would not otherwise mix. Once these intervening causal variables are known, it becomes apparent that there could well be a sequence of events causally connecting weather events in the South Pacific with flu pandemics (answering CQ5).

Shaman and Lipsitch also supported their hypothesis that the La Niña conditions can favor the spreading of influenza by testing it using evidence from population genetics and bird migration patterns. So in addition to filling in the causal missing links between the two events, they also presented some theoretical backing connecting the two events and supporting their hypothesis. Note however that they were modest in describing their causal conjecture, expressing that their conclusion is based on argument to the best explanation. Their hypothesis was that the association between the four La Niña events and the four influenza events is the most plausible biological explanation of the correlation between the two sequences of events.

For these reasons, the argument from correlation to causation in the birds example can be evaluated as more plausible than the argument from correlation to causation in the chocolate

example. The reason is that the argument and a former example answers more critical questions, and is less open to some worrisome critical questions than the argument in the latter example.

The argument from correlation to causation in the copper example was also based on experimental results. Deane and his colleagues conducted an experiment on the results of feeding drinking water containing copper to mice for three months. They also offered a scientific theory joining the ingestion of copper with the kind of plaque buildup that leads to Alzheimer's disease. On this theory Alzheimer's disease is caused by the accumulation of amyloid beta in the brain. They offered a scientific theory explaining how copper can make its way into the walls of the capillaries that protect the brain from toxins, ultimately breaking down the blood brain barrier. They colorfully describe this procedure as a one-two punch that both stimulates the production of amyloid beta and prevents the brain from clearing it away.

If we were to stop the description of the argument from correlation to causation at this point, it would seem to be based on the right kind of scientific evidence, and right sort of explanation of the sequence of events linking the two variables. Hence we would probably evaluate the argument as being at least as plausible as those in the chocolate example and the birds example. But as we go on to examine additional evidence in the copper example in section 6, we will need to recall that another expert strongly disagreed with the conclusion of Deane and his colleagues.

6. The Dialectical Context of an Argument

In the copper example we have a typical battle of the scientific experts, a common phenomenon in legal argumentation in trials (Walton and Zhang, 2013). How should we proceed in a case where the conclusion claimed by one expert contradicts the conclusion put forward by another? There is another scheme that comes into play in such cases. The scheme for argument from expert opinion takes the following form (Walton, Reed and Macagno, 2008, 310).

Major Premise: Source *E* is an expert in subject domain *S* containing proposition *A*.
Minor Premise: *E* asserts that proposition *A* is true (false).
Conclusion: *A* is true (false).

This scheme, like the scheme for argument from correlation to cause, can be formulated in a conditional form of a defeasible modus ponens argument (DMP). The conditional version can be formulated as follows (Reed and Walton, 2003, 201).

Conditional Premise: If Source *E* is an expert in subject domain *S* containing proposition *A*, and *E* asserts that proposition *A* is true (false) then *A* is true (false).
Major Premise: Source *E* is an expert in subject domain *S* containing proposition *A*
Minor Premise: *E* asserts that proposition *A* is true (false).
Conclusion: *A* is true (false).

There are six basic critical questions (Walton, Reed and Macagno, 2008, 310) matching this scheme.

Expertise Question: How credible is *E* as an expert source?
Field Question: Is *E* an expert in the field *F* that *A* is in?
Opinion Question: What did *E* assert that implies *A*?
Trustworthiness Question: Is *E* personally reliable as a source?
Consistency Question: Is *A* consistent with what other experts assert?
Backup Evidence Question: Is *E*'s assertion based on evidence?

The consistency question is the one on point in the copper example. This critical question is treated as an exception in CAS. In order to defeat the argument from expert opinion, a critic needs to cite the opinion of an opposed expert.

The structure of this situation can also be modeled as a rebuttal as shown in figure 6.5.

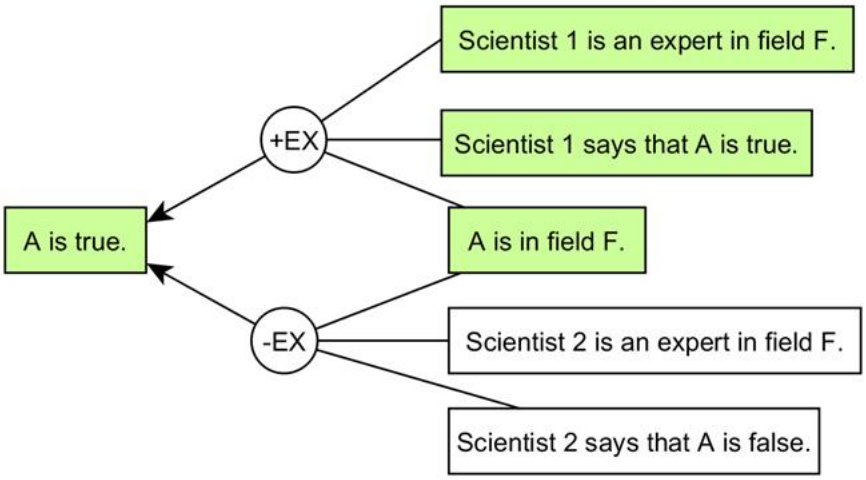

Figure 6.5: Battle of the Experts Stage 1

The problem is how to deal with this kind of case. The argument at the top of figure 6.5 is a pro argument from expert opinion supporting the conclusion that proposition *A* should be accepted. The argument at the bottom is a con argument from expert opinion supporting the conclusion that *A* should not be accepted. Just considering the top argument as having premises that are accepted, as shown in figure 6.5, the conclusion is also automatically accepted by the system.

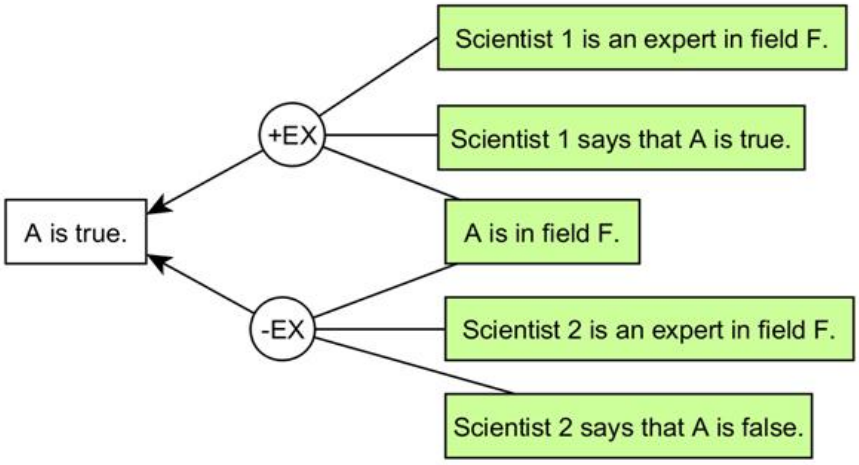

Figure 6.6: Battle of the Experts Stage 2

But once we consider both arguments as having accepted premises, as shown in figure 6.6, the conclusion that *A* is true is no longer accepted. Unless we know that the one argument is stronger than the other, or unless further arguments can be brought in on one side or the other to break the deadlock, the solution is to suspend judgment. The proper conclusion to draw is that research on the question of whether the argument from correlation to cause in the copper example put forward by Deane as a hypothesis is in dispute. Since Deane admits himself that it is only a provisional hypothesis which needs to be subject to further testing and investigation, this hypothesis of a deadlock seems to be the right solution in this instance

But there is another aspect of this kind of situation to be considered. How is a physician supposed to advise his or her patient on the question of whether ingestion of copper should be avoided in order to prevent or treat Alzheimer's disease? Or how is a person to proceed who has Alzheimer's disease and is worried about it to change his or her diet in light of the scientific knowledge about causation of Alzheimer's disease by ingestion of food or water containing copper? Such a person cannot normally go out and test the scientific evidence. The best he or she can do is to examine the scientific literature, collect the findings supported by the experts, and judge what conclusion on the subject is best to move ahead with.

To deal with the problem of the battle of the experts the argumentation approach needs to take into account the transmission of scientific expertise to those outside the particular field in which a scientific claim lies (Walton and Zhang, 2013). As well as the argumentation scheme, the argumentation approach takes into account as well the setting in which an argument or some related speech act was put forward in a context of dialogue. With the kinds of arguments being considered here, it is important to differentiate between two such contexts of argument use. In the first setting, the argument is being used in a scientific context where a research paper published in a science journal is reporting the results of a correlation based on statistical findings and possibly experimental evidence, and conclusions are drawn. In the second setting, the conclusions that were drawn are being reported as a scientific finding that is of interest to those who might wish to apply it to solve some problem, normally a problem about what to do. For example, typically such scientific findings are used by physicians to give advice to patients on what course of action to take. But scientific findings that are especially interesting to a large number of people will be reported in the popular media, and described in a manner that their readers will be able to understand, and will find interesting.

For this reason, for those of us interested in studying examples of arguments of this sort, and even studying biases and logical shortcomings in them, and trying to find some basis for evaluating arguments from correlation and causation as strong or weak, understanding the shift from the one setting to the other is of vital importance.

There is pressure on those who report in the popular news media to present information in a way that makes it interesting and exciting by creating a favorable impression. To do this, the popular report may ignore important qualifications expressed by the scientific researcher, for example mention of side effects, or limitations on the conclusion posed by the need for further scientific investigations. However, it has also been noted that there is pressure on the scientists themselves to get their papers published by making their findings appear exciting. For example, scientific journals have a strong preference for publishing positive findings, as opposed to negative findings to the effect that an expected result did not occur (Freedman, 2004, 110). As Freedman (2004, 11) reported, there is an intense pressure to produce publishable results that are positive and exciting, while at the same time there is a principle that the more novel and exciting an idea is, the less likely it is to be right (Ioannidis, 2005). Hence there are good reasons to

accept the proposition that the biases and other shortcomings of scientific arguments from correlation and causation in such matters as nutrition and health are not exclusively due to misreporting of scientific research by the media.

7. Contesting the Causal Link between Smoking and Lung Cancer

The kinds of examples studied so far are instances of the traditional *post hoc* fallacy, where a premature leap is made from a correlation based on scientific evidence that has been collected to a causal conclusion. But there are also interesting cases of the opposite problem to be found. In these cases, the scientific evidence supporting the existence of a causal link between two events is played down by denying or minimizing it using opposed scientific evidence. Instead of arguments supporting the causal link between *A* and *B*, there are counterarguments attacking the hypothesis of a causal link between *A* and *B*. The most famous case of this sort is the campaign of the tobacco industry to argue against the hypothesis of a causal link between smoking and lung cancer (and other causes of premature morbidity).

By the 1960s there was a growing body of scientific evidence showing a causal link between tobacco and harm from cancer, and there was an especially strong causal link established by many scientific studies between smoking and dying from lung cancer. An organization called the Council for Tobacco Research, funded by the tobacco industry, supported scientific research purporting to show that the link between cancer and cigarettes was "merely statistical". The opposed scientific evidence funded and presented by this organization claim to show that it would be premature to accept the causal theory linking smoking and cancer (Proctor, 1995, 1060. Nowadays this tobacco industry research is ridiculed as biased science of the kind promoted by public relations firms. But at the time it exerted a powerful influence on public opinion concerning the effects of smoking on cancer.

During this period there was a great public debate marshaling scientific findings on both sides. Tobacco industry-supported research argued that there might be a genetic explanation showing a personality type that leads people to smoke and at the same time predisposes them towards lung cancer (Proctor 1995, 107). This argument corresponds to the critical question of argument from correlation to cause asking whether there might be a common cause linking the two events that are supposed to be causally connected. Asking this critical question is a way of casting doubt on the hypothesis, in this instance, that smoking causes lung cancer. Instead, it is suggested that there is some third variable causing both the smoking and lung cancer.

The asking of this in this case can also be modeled as a counter-argument. The counter-argument can be expressed as follows.

- Scientific evidence shows that people who smoke and develop lung cancer are predisposed to both smoke and get this disease by reason of their heredity.
- Therefore there is a better explanation of the statistical correlation between smoking and lung cancer than the causal link theory.
- This better explanation is the genetic explanation that there is a common cause linking smoking and lung cancer.
- This genetic explanation is supported by scientific research.
- Therefore it is doubtful that smoking causes lung cancer.

This counterargument can be modeled using the argumentation scheme for inference to the best explanation. In figure 6.7, the conclusion, the statement that smoking causes lung cancer, is shown at the left. The argument from correlation to cause is shown at the top, indicated by the +CC notation in the argument node. The plus sign indicates it is a pro-argument. This argument is shown as being attacked by a counterargument that has the form of inference to the best explanation, indicated by the notation -IB in the argument node. The minus sign indicates it is a con argument.

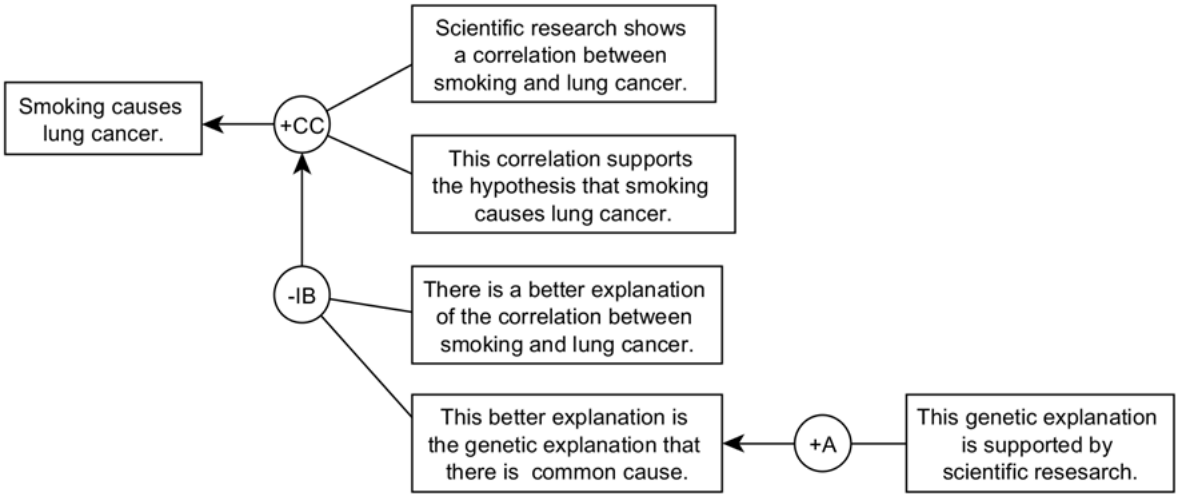

Figure 6.7: Counter-argument to the Causal Lung Cancer Argument

What is illustrated in figure 6.7 is an argument from correlation to cause being put into question by an undercutter that performs the role of posing a critical question that casts the original argument into doubt. Moreover, the undercutting argument is successful because one of its premises is backed up by supporting evidence, expressed in the statement that the genetic explanation is supported by scientific research.

What is especially interesting to note in this case is that the argument is not purely a scientific one. Is this a use of scientific evidence for the purpose of public relations? As Proctor, (1995, 107) explained, there is a legal and public relations value of such studies.

> If differential cancer susceptibilities could ever be established, one could plausibly argue that people who come down with the disease have a least partly their own heredity to blame. Perhaps many of those who smoke are invulnerable, after all, only about one in five smokers ever gets lung cancer - why doesn't everyone?

In this case then we can see that the mounting evidence of the link between smoking and lung cancer based on argument from correlation to cause became stronger and stronger, threatening the interests of the tobacco companies. They fought back by funding their own scientific research that not only critically questioned the argument from correlation to cause, but actually counterattacked it by financing their own scientific studies, and using these to mount counterarguments.

8. Contesting the Causal Link between Football and Brain Damage

The following case concerned the scientific question of whether playing football causes brain damage. A *Frontline* program first aired in 2013, *League of Denial: The NFL's Concussion Crisis*, chaired by Mark Fainaru-Wada and Steve Fainaru, chronicled the story of how this causal connection first came to be suspected by clinical investigators, and how it played out in the subsequent inquiry. At that time both men were employees of the entertainment and sports programming network (ESPN), a media empire operating seven 24-hour sports channels, a website with more than 37 million visitors every month, and a radio network of more than 400 stations. The huge fan base for professional football in the USA made the NFL an extremely rich and powerful organization. In the same year that the *Frontline* program appeared, a book was published (Fainaru-Wada and Fainaru, 2013) that gave a detailed and carefully documented account of the events and scientific investigations making up the story.

The best place to begin is with Mike Webster. "Iron Mike" was a famous football player for the Pittsburgh Steelers when they won four Super Bowls in the 1970s. He was famous for his aggressive style of play and his ability to take extreme punishment, take the blows and absorb the pain, when colliding with other players. After he retired, his health deteriorated through states of depression and into progressively serious dementia. Webster's family told how his life spiraled out of control. He was unable to remember things, unable to get his thoughts together, he was an angry and confused man who threatened to commit suicide, and in the end wound up living in his truck, separated from his family (Fainaru-Wada and Fainaru, 2013, 3). Eventually he decided to sue the NFL, arguing that his injuries were caused by playing football. When the NFL's physicians eventually agreed with Webster's doctors that his injuries were due to football, they agreed to pay him a monthly check.

When Webster died at the age of 50 in 2002, Pittsburgh Medical Examiner, Dr. Bennet Omalu performed the autopsy. When he first examined Webster's brain, it appeared to be in a normal condition. Fortunately however, he made the decision to preserve the brain, and after further investigations found that it was filled with the protein characteristic of CTE (chronic traumatic encephalopathy). CTE is a progressive degenerative disease found in individuals with a history of multiple concussions or other forms of serious head injury. It can be definitively diagnosed only by pathological examination of the brain (after death).

What is interesting from our point of view in this book is the opposition to Dr. Omalu's findings and how it was strongly backed by scientific and medical experts. When Dr. Omalu published his findings, the NFL tried to have the journal retract his article. The journal refused, but the NFL continued to attack Dr. Omalu's research and his credentials. The NFL published its own medical articles claiming that football is safe and denying the connection between brain injuries at football. Next, NFL founded a Mild Traumatic Brain Injury Committee in the 1990s chaired by a medical doctor. This powerful and well-financed committee continued to advocate the position that there was no causal connection between football and brain injuries. The committee was chaired by several successive leaders, and was at a later stage chaired by a neurologist who went so far as to make the claim that the findings of Dr. Omalu were wrong.

The next stage in the evolution of the case was that Boston University formed a research team to look at the impact of football injuries on the brain. They recruited Dr. Anne McKee, a pathologist, a specialist in studying Alzheimer's disease. She started to collect the brains of former football players and study them. She found evidence of CTE in almost all of the brain she studied. When the researchers made this discovery, they thought the NFL executives would

support their findings and try to make football safer. Instead what happened is that the NFL used its scientific and media power to attack the research. The NFL had co-opted an influential medical journal to publish a series of papers that denied concussions were due to football. Several of these papers were rejected by peer reviewers and later even disavowed by some of their own authors (Fainaru-Wada and Fainaru, 2013, 6).

Gradually a number of cases came to be publicized (Fainaru-Wada and Fainaru, 2013, 7). One well-known NFL player killed himself by drinking antifreeze. Two players fired handguns into their chests, and a famous linebacker shot himself in the guest room of his beach house. These football players were public figures who were well known to be good citizens and widely admired people, in contrast with their degeneration into almost unrecognizable states. Eventually, nearly 6,000 retired players and their families sued the NFL for negligence and fraud, arguing that the NFL had propagated its own industry funded and falsified research in order to conceal the causal link between football and brain damage.

After Boston University researchers had published their findings, congressional hearings compared the NFL to the tobacco industry's marshaling of scientific evidence for so many years supporting the claim that there was no causal link between smoking and lung cancer. To broadly summarize the sequence of events in the case, the NFL had spent over twenty years marshaling a series of their own scientific studies designed to systematically attack the causal connection between football and brain damage. A neurosurgeon connected to the NFL claim that children were more likely to sustain brain injuries from riding a bike or falling down (Fainaru-Wada and Fainaru, 2013, 7). Just as in the case of the tobacco industry the NFL had supported their arguments funding their own opposed expert scientific findings.

In the smoking and football examples, it is more than just a case of critically questioning an expert opinion by posing the critical question that other experts do not agree with a claim made by a particular expert. In these cases, there is a systematic disagreement between the two sides, each representing a group of scientific experts. The opinions of each group may be internally consistent, but as a whole constellation of expert opinions, they each disagree with each other. Not only that, we can see that the arguments put forward by the second group of experts have been specifically designed to attack the arguments put forward by the first group of experts.

In these kinds of cases, there is a large mass of evidence on both sides. To analyze such a case using argumentation methods, you have to collect the arguments on the one side, and show how they are connected to the arguments on the other side. An examination has to be made of how each argument either supports or attacks other arguments put forward by the other side, and you also have to collect and examine all the evidence that has been put forward by experts that both sides agree to. Examining either of these cases in detail is a project beyond the resources of this book. Analyzing them using argumentation methods of the kind described and illustrated in the book can be a project for future research. But both cases are especially interesting for the purposes of studying argument from correlation and causation because both illustrate the problem of biased scientific evidence employed for public relations purposes to promote the interests of the group with something to gain. This kind of strategy uses argument from expert opinion to attack a prior argument from correlation to cause.

9. Reformulating the Critical Questions

A recommendation for modifying the set of critical questions matching this scheme for argument from correlation to cause concerns Bradford Criterion 5, which states that the causal

inference is stronger when there is a theoretical scientific basis linking the two events. This particular factor was stressed in CAS. It is proposed that the following critical question be added to the existing list of seven: is there a theoretical scientific basis linking the two events *E1* and *E2*? There are also some other critical questions to be considered.

The structure of the inference from a surrogate marker to a causal conclusion shown in figure 6.5 suggests other critical questions. The first is whether the argument from correlation to cause as stated goes to the ultimate conclusion or whether instead it goes to a surrogate marker. The second question is to be asked when the surrogate marker has been identified. The third question asks whether the inference from the surrogate marker to the conclusion is justified. It could be argued that consideration of the SMI is already included in the existing critical question 7, and so the three new critical questions could be seen as subquestions of the old critical question 7. However because of the importance of the SMI, as demonstrated in the examples treated in this chapter, it is concluded that these new questions should be added to the existing set.

Adding these three new critical questions suggests revising the set of critical questions matching the scheme for argument from correlation to causation as follows.

CQ1. Is there a positive correlation between *E1* and *E2*?
CQ2. Are there a significant number of instances of the positive correlation between *E1* and *E2*?
CQ3. Is there good evidence that the causal relationship goes from *E1* to *E2* and not just from *E2* to *E1*?
CQ4. Can it be ruled out that the correlation between *E1* and *E2* is accounted for by some third factor *E3* (a common cause) that causes both *E1* and *E2*?
CQ5. If there are intervening variables, can it be shown that the causal relationship between *E1* and *E2* is indirect (mediated through other causes)?
CQ6. If the correlation fails to hold outside a certain range of cases, then can the limits of the range be clearly indicated?
CQ7. Can it be shown that the increase or change in *E2* is not solely due to the way *E2* is defined, the way entities are classified as belonging to *E2*, or changing standards, over time, in the way *E2* is defined or classified?
CQ8. Is there a theoretical scientific basis linking the two events *E1* and *E2*?
CQ9. Does the argument from correlation to cause as stated go to the ultimate conclusion or to a surrogate marker?
CQ10. If a surrogate marker has been identified, is the inference from the surrogate marker to the conclusion justified?

As each critical question is asked, and replied to appropriately by the arguer, the argument from correlation to cause is strengthened. As suggested by the three textbook accounts of Johnson and Blair (1983), Govier (2005) and Groarke and Tindale (2004), the supporting evidence gained by answering the questions appropriately makes the argument less weak and less open to the *post hoc* objection. The approach suggested by this chapter is that arguments from correlation to cause should be judged on a dialectical continuum, so that as more and more critical questions concerning the hypothesis that there is a causal relation between the two events are answered appropriately, support for the hypothesis becomes stronger and stronger. As the pro evidence outweighs the con evidence and answers critical questions, the strength of the argument from correlation to causation increases. Alternatively, it can decrease as critical questions are not answered, or as counter-arguments are brought against the argument.

More precise details of how this dialectical evaluation procedure works can be shown by explaining how CAS manages the burden of proof between the arguer and the critical questioner in a dialogue sequence (Walton and Gordon, 2011). Argument from correlation to causation is evaluated by using a set of critical questions matching the scheme in a dialectical procedure where a burden of proof is shifted back and forth between the proponent and the questioner. The problem with modeling this procedure using an argument diagram of the kind shown in figures 6.2, 6.3, and 6.4 is that some critical questions shift the initiative back to the proponent, while others only do so when evidence to back up the question is given. In CAS, the former types of critical questions are treated as assumptions while the latter one are treated as exceptions. The ordinary premises of a scheme are taken to hold in CAS, but if questioned they are taken to be no longer accepted until they are supported by some evidence. But there are two additional kinds of premises that have been designed to model the two different kinds of critical questions. Assumptions, like the ordinary premises, only need to be backed up if they have been questioned, while exceptions are assumed not to hold until evidence is given showing that they do.

It should be noted that the list of ten critical questions can be used to guide a causal inquiry as well as to evaluate arguments from correlation to causation. A causal inquiry might typically start from a correlation between two events or types of events that is puzzling because there is yet no explanation of the connection, if any, between the events. The list of critical questions can aid an investigation to move forward by searching for experimental evidence or other kinds of evidence that might link the two events together. This can be done either by finding intervening events that connect them, or by bringing a scientific theory to bear on experimental evidence that shows a much more convincing kind of connection than one that can be established by statistical evidence of correlation alone. Next, the causal inquiry might move to a stage where a scientific explanation is offered that reveals the underlying connection, a physical, chemical or biochemical connection for example, between the two events. At the next step, inference to the best explanation can be applied to the evidence in the case, and alternative explanations to the most plausible one may be rejected, again based on the evidence known at that point as the inquiry proceeds. To fill out this suggestion, more needs to be known about the opening and closing stages of such an inquiry, and how the evidential reasoning in the stage between them moves forward to the stage of proof or disproof of the hypothesis. Such a model of evidential reasoning in an inquiry is outlined in chapter 7.

10. The *Post Hoc* Fallacy

The problem posed by arguing from correlation to causation is that it has traditionally been associated in logic with the fallacy of *post hoc ergo propter hoc*, but is often a reasonable form of argument, under the right conditions. It is not only one of the most prominent forms of argument in medicine and other applied sciences, but it is also a kind of reasoning we constantly depend on in everyday life, as the examples given in section 1 suggest. The problem is generally that, as shown in this chapter, there is a continuum of instances of this type of argument from the very weak, to the basically reasonable but questionable, to the fallacious. We have limited the scope of this chapter to five main examples, but there are other examples at both ends of the continuum that could be studied in future research. Let's consider just one example at the weak end and one at the strong end. According to a BBC News Report[6] a study by Barclays Capital

[6] http://www.bbc.co.uk/news/business-16494013

linked skyscrapers with impending financial crashes, citing the building of the Empire state building just before the Great Depression and the building of the Burj Khalifa just before the large recession in Dubai. Although an impressive set of examples of correlations between these two events was cited in the report, there appears to be no other evidence to suggest that the building of skyscrapers causes financial crashes. As an example at the strong end, consider the correlation between smoking and lung cancer. At one time in history the existence of a causal link of this nature was strongly denied, especially by tobacco company research, as shown in section 8, but now the evidence makes this argument convincingly strong.

Also there is a variation of context in examples that needs to be taken into account. A very weak argument from correlation and causation might well be reasonable as a form of argument used to suggest a hypothesis at a very early discovery stage of an investigation. However the very same argument used at a later stage of the investigation, could well be inadequately supported, or even fallacious, if it pressed too strongly for a causal conclusion while overlooking critical questions that need to be asked at that point. The dialectical complexity of the situation is compounded when authors of a research report make a claim for a causal conclusion based on experimental or statistical findings about correlation between the two events in question, and this conclusion is accepted and advocated by parties external to the scientific research. The typical kind of example studied above has two stages. The first is that of a scientific paper arguing from correlation and causation as the basis for inferring a causal conclusion. The second is that of a news report in the media or other public source of information, or other academic journals, reporting the causal conclusion and describing the evidence that led the scientific investigators to it, based on a correlation. Both instances can be classified as cases of arguing from a correlation to a causal conclusion, but the standards of proof and the methods of argumentation used in each instance are different.

Some traditional logical fallacies have been associated with heuristics, or fast and frugal rules of thumb that are commonly used, and also are very useful means of quickly jumping to a tentative conclusion that may later need to be retracted or revised as more calculative methods are brought into play (Walton and Gordon, 2009). The examples studied in this chapter show that argument from correlation to causation fits this category very well. At the first stage of the typical sequence of argumentation of this sort, a surprising correlation is found. The question at this stage is whether it is merely a coincidence or whether there may be some causal connection between the two events at issue. The conjecture of a possible causal connection between the two events at this point should be treated as a very weak argument that may be presumptively acceptable as a means to move forward towards further collection of evidence, but that should be seen as inherently open to critical questioning. At the next points in the sequence, some further evidence connecting the two events may be found. For example, there may be other events intervening between the two events that connect them together as a longer sequence. Or there may be some scientific evidence, or a scientific theory, that tends to confirm the connection between the two events, creating a more plausible hypothesis that they are causally connected.

Given this procedure of evaluation, figure 6.1 can be used to illustrate how the *post hoc* fallacy can be explained. As shown along the bottom of figure 6.1, there is a tendency to jump from a correlation to a causal conclusion. This kind of inferential jump is extremely common in everyday reasoning, as well as in scientific reasoning such as medical reasoning. It is hard to resist it, and easy to take advantage of it. As soon as we see a correlation between two events, or even one event happening after another one, using the causal heuristic we jump to the conclusion that the one event might cause the other. Depending on how strong we take the argument from

correlation to cause to be, succumbing to the heuristic to accept the conclusion that the one event causes the other can be a good guess, unfounded speculation, or even a fallacious argument. The fallacy, or the error of *post hoc* reasoning, essentially occurs when the arguer jumps ahead on the basis of a correlation to conclude that a causal connection exists, without taking the critical questions into account. In the example shown in figure 6.1, such a presumptive leap to a causal conclusion would not be justified unless it is taken into account to what extent the SMI can be justified. So the kind of reasoning shown in figure 6.1 can be generalized to offer a theoretical explanation of how the *post hoc* fallacy works, an explanation that confirms and extends the approach to arguments from correlation to cause taken by Johnson and Blair (1983), Govier (2005) and Groarke and Tindale (2004). An argument from correlation to cause can be weak, but that is OK if it was only put forward as a hypothesis not yet fully tested. But if questioned, the questioning and answering procedure must fulfill the requirements of burden of proof appropriate for the discussion.

If the arguer persists too aggressively or thoughtlessly by overlooking appropriate critical questions, or even by trying to prevent the questioner from asking them, or evading the burden of answering them, then this is a kind of case where an accusation of committing the *post hoc* fallacy can be brought into play. The smoking and the football examples have shown that the *post hoc* fallacy does not consist exclusively in the error of using a heuristic to jump too quickly to conclude that a causal connection exists based on a correlation. In some cases, the fallacy consists of a strategic maneuver designed to forestall the drawing of a reasonable inference from correlation to causation by means of employing a systematic tactic of attempting to prevent critical questions from being asked. Some significant similarities between the tactics used in the football case and the smoking case were noted by Fainaru-Wada and Fainaru (21013, 280). In both cases powerful companies had used their power and vast financial resources to discredit scientists they disagreed with. They tried to downplay work of scientists who had opposed expert opinions. They used inappropriate kinds of critical questions to attack these opposed expert opinion arguments. They put forward expert opinion arguments of their own based on scientific research that served their interests and that they had financially supported.

These cases show evidence of systematic public relations campaigns, supported by the use of scientific evidence, to try to shift the burden of proof back to the side who has claimed that a causal conclusion can be inferred from a correlation. The variant of the *post hoc* fallacy illustrated by these examples does not represent the error of jumping too quickly to a conclusion. It is just the opposite. It represents a strategy of pushing back too hard against a well-supported argument from correlation to cause by using all kinds dubious tactics to try to persuade the audience (the public) without giving the argument a fair hearing. For the purposes of this chapter, these cases are merely examples of a certain kind of argumentation strategy used to rebut arguments from correlation to causation. However, from a point of view of argumentation logic, they represent patterns of argument that we need to be aware of, and that we need to study further.

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