Arguing about Causes in Law: A Semi-formal Framework for Causal Arguments

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Abstract. Disputes over causes play a central role in legal argumentation and liability attribution. Legal approaches to causation often struggle to capture cause-in-fact in complex situations, e.g. overdetermination, preemption, omission. In this paper, we first assess three current theories of causation (but-for, NESS, 'actual causation') to illustrate their strengths and weaknesses in capturing cause-in-fact. Secondly, we introduce a semi-formal framework for modelling causal arguments through strict and defeasible rules. Thirdly, the framework is applied to the *Althen* vaccine injury case. And lastly, we discuss the need for new criteria based on a common causal argumentation framework and propose ideas on how to integrate the current theories of causation to assess the strength of causal arguments, while also acknowledging the tension between evidence-based and policy-based causal analysis in law.

Keywords: argumentation, causation, evidence, law, legal reasoning

1 Introduction

Disputes over causes play a central role in legal argumentation. In particular, lawyers and judges are interested in explanations of causal links from the outcome to its causes and how these explanations can be linked to the evidence and parties involved in the case. Questions such as – who injured the victim or what caused the fire in the house – are commonplace in legal discourse. However, the theoretical foundation for causal argument assessment in law is lacking. Researchers [24] have struggled for decades to explain some of the complex theoretical phenomena that come with reasoning from the effects to causes. For instance, how to establish a causal link in cases of inaction, or what is the cause in situations where two (or more) parties had a partial role in some overall causal action? The issue of causation in law is both practical, with the limited guidance provided to the decision makers who need to balance the opposing arguments in establishing causal links, and theoretical, with the competing causal theories unable to accurately capture causes in complex situations.

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This study investigates how people reason and argue about causes in legal cases. In particular, we analyse the language and arguments used by the medical expert witnesses and judges in a vaccine injury case. To assess and understand causal arguments presented in the case, we first consider two issues of complex causal phenomena, overdetermination and preemption, and how the current causal theories explain the phenomena. Namely, we analyse the *but-for counterfactual test* [12], Hart and Honoré's [11] *necessity and sufficiency test*, and Halpern and Pearl's [10] structural *'actual causation' test*. After evaluating the strengths and weaknesses of these theories, we present our causal framework, which is designed to model arguments about causation in a legal case. In our framework, causal models encompass various strengths of belief in causal links and represent the structure of the causal arguments as observed in the arguments. We abstract the arguments through a semi-formal framework that employs defeasible logic as well as introduce basic notions of causal relations that are linked to evidential arguments in the cases. By modelling and assessing legal arguments about causation, we seek to better understand which underlying causal structures are present in the legal arguments.

Our causal framework[4] contributes to the current developments in legal reasoning and argumentation research in at least two ways. Firstly, by systematically examining causation in legal arguments, we are able to uncover implicit premises and argumentation steps in expert witness testimonies, allowing for an explicit, structured comparison between the opposing views. Secondly, we model the causal links found in the arguments through a new semi-formal framework and propose an assessment of the causal links based on the current theories of causation. Our strategy in using the causal theories is not to choose one over the other in all possible cases. Rather it is to choose the most efficient approach for the scenario at hand. For instance, if the simple but-for test is able to correctly capture the cause in a case, then a full formal analysis by the 'actual causation' theory is not needed. In the same vein, a clear reasoning path in complex cases, where there can be multiple causes interacting over a long period of time, is essential for building sound arguments for parties in the case. Furthermore, a domain of medical causation often involves highly specialised evidence presented by the experts, leaving the judges with the increased burden of assessing the arguments. A potential development of the proposed framework is to extend it into causal argument schemes that include some of the core reasoning patterns for the most common causal issues (multiple causes, preemption, omission) and to pose critical questions for assessing them. While the current analysis is mostly concerned with the initial steps of legal decision making, i.e. establishing the cause-in-fact based on the evidence available, we are also interested in the role of normative reasoning in restricting or broadening the causal test for liability attribution.

[4] Different versions of the framework have been presented at the Evidence and Decision Making Workshop, ICAIL, 2017, London and AICOL Workshop, JURIX, 2017. This paper presents the latest developments of the framework to date.

The paper is presented in three main sections. We start by laying out the background and running case examples of the study, as well as introducing and assessing the causal theories. In section 3, we introduce our semi-formal framework and model causal arguments of the *Althen* vaccine injury case. We conclude the paper by discussing the need for new criteria for assessing causal arguments in law and presenting an early proposal for extending the framework.

2 Background

Causation is one of the fundamental concepts of human reasoning, it allows us to connect events in a meaningful way. In this study we investigate two aspects of causation: the logical structure of cause-in-fact and argumentation strategies used to discuss causes in law. We first introduce the dominant philosophical and formal theories that define ways causal links can be established based on various forms of logic. We present three approaches in order of increasing complexity: the *but-for test*, Hart and Honoré's *NESS test*, and Halpern and Pearl's *'actual causation'* theory. To illustrate the differences between these approaches we consider three classic causal scenarios: direct causation, overdetermination and preemption. Secondly, we show how these causal theories can be used to assess arguments about causation.

What is important to note is that our semi-formal framework does not restrict the use to only one causal theories, rather the idea is to have the freedom to choose between them for the best-fit for the scenario at hand. For instance, if the link between the medication and recovery can be established by a simple but-for test, there is no need to apply the more elaborate NESS or 'actual causation' tests. Thirdly, we introduce the case study of the *Althen* vaccine injury case. We consider the argumentation strategies used in the case, focusing on the language used by the medical expert witnesses and the judge.

2.1 Examples

We start by introducing three classical examples found in the causation literature [24]. Direct causation is an instance where the link between the event and the effect is uninterrupted; overdetermination where there are two or more causes for the same effect; and preemption where one event preempts the following event in causing the effect.

Simple (Direct) Causation. In a simple (direct) causal scenario there is only one cause and one effect. Think of a scenario where Anne gets a headache and takes an aspirin (TA) [19]. Just a few minutes later, she already feels the effects of the pill: the headache is gone (HG). The effect of the aspirin pill was direct and uninterrupted. While simple causal events happen often, there are many other conditions that might come into play complicating the original scenario. Moreover, in legal discussions it is usually the causation of the more complex nature that raises issues for disputes.

Fig. 1. Direct causation

Overdetermination. The multiple causes, or the overdetermination, effect occurs when there is more than one cause sufficient for the outcome. The classical example chosen here is that of a firing squad, where two soldiers (S1 and S2) are instructed to aim and shoot at the same prisoner to carry out the death penalty. Assuming that both shooters are accurate and shoot at the same time, they both cause the death of the prisoner (PD). However, each of their shots alone would have been sufficient for killing the prisoner. Who caused the death of the prisoner in this scenario? Different causal theories lead to conflicting conclusions. Multiple causes also appear in many medical situations. For instance, when two (or more) alternative treatments independently cure the illness, but these are applied together causing the patient to recover.

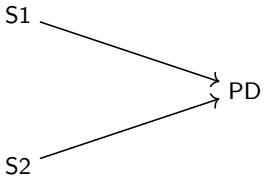

Fig. 2. Overdetermination

Pre-emption. Preemption is a special instance of overdetermination cases, as it also concerns conditions that are independently sufficient for the outcome. The key difference here is in the order of events. While in overdetermination we assume that the actions happened simultaneously or at least in a way that

the agents are not able to change their actions before the effect takes place, then in preemption scenarios there is an order of events such that one action preempts the other. We will use the example of rock throwing [9] to illustrate this phenomenon. Suzy and Billy play a game where they both throw rocks at a bottle. Suzy throws (ST) and her rock hits (SH) and shatters the bottle (BS), while Billy's throw (BS) comes right after and does not (BH) shatter the bottle, since the bottle has already been shattered. The graphical order of the lines in Figure 3 reflects this showing all possible events and their interactions. However, Billy's rock would have hit the bottle if Suzy's rock had not reached the bottle first. While we might intuitively have an idea about the cause-in-fact in this case, causal theories have struggled to accommodate these relations by providing either too strict or too loose criteria for the cause.

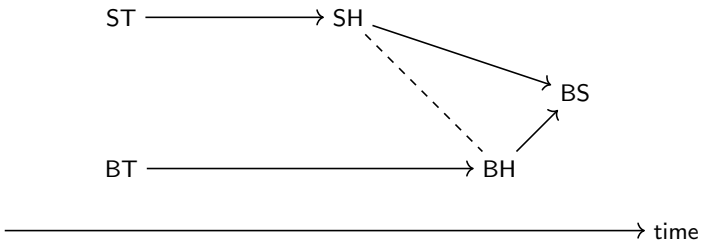

Fig. 3. Preemption

2.2 Theories of Causation

Causation is a widely researched field [2], however only some discussions are relevant to legal analysis. Here, we discuss three approaches to causal analysis, starting with the simple counterfactual *but-for* test. We then introduce two more advanced causal theories: Hart and Honoré's [11] theory of necessary and sufficient conditions and Halpern and Pearl's [10] structural approach to *'actual causation'*. The differences between these approaches are illustrated through their ability to analyse the causal examples.

But-for test. A common way of thinking about causation in everyday and legal reasoning is the but-for causation or *sine qua non* test. It considers what could have been the outcome if only one aspect of an event in question was different from the original scenario. In other words, one engages in counterfactual reasoning of the kind: had a not happened, b would not have happened. If one accepts the reasoning, one has shown that a is an essential element in the occurrence of b. This approach is very effective in showing the impact of a single change in the chain of reasoning. However, it has been repeatedly shown that the

necessity conditions are insufficient to capture more detailed causal relations [11, 17].

For instance, modelling the firing squad example in the but-for test would lead to conclusion that none of the shooters was the cause. Recall that both S1 and S2 independently caused the prisoner's death (PD). To qualify as a but-for cause, each of the shooters would have to be necessary for killing the target. Essentially, to successfully pass the but-for test one should be able to claim that had shooter one not shot the prisoner, the prisoner would not have died. However, such a conclusion is false in this scenario, because the prisoner would still have died due to the second shooter hitting the target. Similarly in the preemption example, the but-for test is too broad to pick out the cause: the bottle would still have been shattered, even if Suzy missed the target, because Billy's rock followed right after and would have shattered the bottle instead. Scenarios like these require a more restrictive test to pick out the cause.

Hart and Honoré's NESS. A more elaborate approach to causal analysis was proposed by the legal philosophers H.L.A. Hart and Tony Honoré in *Causation in the Law* [11]. Their approach, NESS, focuses on sets of sufficient conditions in determining the cause. The supporters [28] of the NESS view argue that in *"a specific situation a causally relevant condition is a necessary element of a set of conditions jointly sufficient for the harmful outcome"*. In other words, NESS searches for minimal sets of conditions that are sufficient for the outcome. If an element is necessary for the sufficiency of the set, then it is considered to be the actual cause. Similar reasoning has also been used in the legal discourse. For the assessment and comparison of alternative causal models, the NESS approach uses the idea of commonsense generalisations about causal links [12]. In particular, the comparison is conducted between the instantiations of such generalisations and how well these instantiations conform with the general case.

The distinction between the necessary and sufficient conditions allows us to capture more complex causal relations compared to the but-for necessity test. In the firing squad example, NESS models each shot as a necessary element of minimally sufficient sets of conditions for the prisoner's death. In other words, according to NESS both shooters contributed to causing the death of the prisoner. Attributing blame to each of the shooters also follows the intuitions held in law. However, NESS test fails to model the cause in preemption scenarios. In the rock throwing example, Suzy's rock preempts Billy's hitting the bottle by shattering it first. An accurate model should capture that the rock reaching the bottle first is the actual cause. Interestingly, the causal approaches chosen here do not explicitly present the temporal elements in causal relations. Instead, the discussions revolve around the dependencies between the events. Therefore, the fact that Suzy's rock reaches the bottle before Billy's is captured by saying that Billy's rock hits if and only if Suzy's rock does not. These nuances, which capture the structural and temporal order of events, are missing from the NESS sufficiency test, making it difficult to model the intuitions about Billy's throw.

Halpern and Pearl's 'Actual Causation'. Causation holds a central role in the artificial intelligence and law research. I.e. causal explanations and predictions help understanding and modelling legal reasoning and legal responsibility attribution [14]. This matter has been approached from various perspectives, including argumentation [26, 27], logic [13, 15, 25], statistics [6], and new, task specific approaches [8, 18]. In this paper, we investigate the ways Halpern and Pearl's (HP) theory of 'actual causation' can explain the causal links found in the case law. They provide a formal approach to causal analysis; they model causation by combining necessary and sufficient conditions, interventions and counterfactual reasoning. An extended version of HP 'actual causation' allows comparing alternative explanations based on notions of normality, typicality and defaults.

Here we provide a simplified introduction to their theory (for the full definitions see [10]), and illustrate it though an example concerning preemption.

A causal model M, according to HP, consists of a set of variables and a set of structural equations. The equations determine how the values of certain variables depend on the values of other variables. The variables whose values are dependent on other values are called *endogenous*, the independent variables (whose value is determined outside of the model) are called *exogenous*. Only endogenous variables can be causes. For instance, in modelling the lighting of a candle, lighting a match may be modelled as an endogenous variable, while the humidity and temperature of the room may be modelled as exogenous variables.

Atomic events are modelled as assertions on the values of a variable; for instance the proposition that Billy throws a stone is modelled as $BT = 1$, where BT is a boolean variable and 1 means "true". In our model concerning two children, Billy and Suzy throwing rocks at the bottle, the exogenous variables are the set of circumstances that determine whether each child throws a stone or not. For instance we may denote such circumstances as CS (the circumstances in which Suzy throws) and CB (the circumstances in which Billy throws). Let us assume that the endogenous variables are ST (Suzy throws), BT (Billy throws), SH (Suzy hits), BH (Billy hits), BS (bottle shattered).

The structural equations are the following, where *max* is a function that takes two (or more) input values and outputs the maximum value, and *min* is a function that takes two (or more) input values and outputs the minimum value:

- $ST = CS$ ($ST = 1$ iff $CS = 1$)
- $BT = CB$
- $BS = \max(SH, BH)$ (the bottle is shattered if one of the two hits it);
- $SH = ST$ (whenever Suzy throws she hits); and
- $BH = \min(BT, 1 - SH)$ (Bob hits the bottle if he throws and Suzy has not already hit it; we assume that Suzy anticipates him).

We write $X = x$ to express the fact that variable X has value x, in the given model. We also write $\vec{X} = \vec{x}$, where $\vec{X}$ is a list $X_1, \ldots X_n$ of variables and $\vec{x}$ is a list of values $x_1, \ldots x_n$ for such variable, for the conjunction $X_1 = x_1 \wedge \ldots \wedge X_n = x_n$.

A key notion in this approach is the idea of an intervention, which consists of assignments of values to variables. We write $X \leftarrow x$ to denote the assignment of value x to a variable X, and write $\vec{X} \leftarrow \vec{x}$ to denote that variables $\vec{X}$ are assigned values $\vec{x}$. Interventions produce counterfactual situations, namely the intervention $X \leftarrow x$ produces the situation that would result by changing what was the value of X to its value as x. For instance, where we start with a situation in which Suzy had thrown the ball, the intervention $ST \leftarrow 0$ produces the counterfactual situation in which Suzy had not thrown the ball. Thus $\vec{X} \leftarrow \vec{x}$ is an abbreviations for $\{X_1 \leftarrow x_1, \ldots X_n \leftarrow x_n\}$.

The expression $(M, \vec{u})$ refers to the fact that the model M is initialised by assigning values $\vec{u}$ to the exogenous variables $\vec{U}$.

The notion of a cause is reduced to this idea of intervention: to determine whether a certain event (the fact that certain variables had certain values) causes a proposition to be true, we consider whether assigning such values to the variables would make that proposition true. For instance, to determine whether the fact that Billy threw a stone, i.e., the fact that BT = 1, caused a bottle to be shattered, we consider whether the proposition BS = 1 would still be true by the intervention $BT \leftarrow 0$, i.e. were Billy not to have thrown the stone.

Let us now consider in detail the definition of an 'actual cause' by HP. To qualify as an 'actual cause', an event has to satisfy the three conditions below, denoted as AC1, AC2, AC3.

Condition $AC1$ states that the given model entails that both the cause $\vec{X} = \vec{x}$ and the effect φ have happened; that is, they hold in the same model and assignment.

AC1. $(M, \vec{u}) \models (\vec{X} = \vec{x})$ and $(M, \vec{u}) \models \varphi$.

In the example of Suzy and Billy, if Suzy causes the bottle to be shattered, it must be true that both she threw the stone ($ST = 1$) and the bottle was shattered ($BS = 1$).

Condition $AC2$ includes two prongs, named $AC2(a)$ and $AC2(b)$.

For $\vec{X} = \vec{x}$ to be a cause, there must be set of variables W and Z having the following features (we can think of Z as including the variables whose values are affected by the causal process generated by $\vec{X} = \vec{x}$, and W as the variables involved in other causal processes).

AC2(a) provides a necessity condition under counterfactual circumstances. In other words, had the cause not happened then the effect would not have happened. More formally, had the variable $\vec{X}$ had value $\vec{x}'$ rather than $\vec{x}$, and had certain other circumstances taken place wherein variables $\vec{W}$ had values $\vec{w}$, the effect ϕ would not have taken place.

AC2(a). $(M, \vec{u}) \models [\vec{X} \leftarrow \vec{x}', \vec{W} \leftarrow \vec{w}]\neg\varphi$.

In our example, assume that $\vec{W}$ includes variable ST, and that we want to establish whether Billy's throw causes the bottle to shatter. To take into account the possibility of multiple causes, we should consider the circumstance in which

Suzy had not thrown the stone ($ST \leftarrow 0$). The putative cause $BT \leftarrow 1$ meets this condition AC2(a), since under if the cause had not happened ($BT \leftarrow 0$) and Suzy had not thrown the stone ($ST \leftarrow 0$), then the bottle would not have been shattered ($BS \leftarrow 0$).

Condition AC2(b) is the most complex one: it states that the cause would have produced its effect even by assuming that no alternative causes are present, but that perturbations exist such that causes may be determined in the causal process being considered.

> AC2(b). $(M, \vec{u}) \models [\vec{X} \leftarrow \vec{x}, \vec{W'} \leftarrow \vec{w}, \vec{Z'} \leftarrow \vec{z}^*]\varphi$ for all subsets $\vec{W'}$ of $\vec{W}$, and all subsets $\vec{Z'}$ of $\vec{Z}$,
> where we abuse the notation and write $\vec{W'} \leftarrow \vec{w}$ to denote the assignment where the variables in $\vec{W'}$ get the same values as they would in the assignment $\vec{W} \leftarrow \vec{w}$ (and similar for $\vec{Z}$), while $\vec{z}^*$ denotes the values that the variable in $\vec{Z'}$ had in reality (in the original model).

In our model, $BT = 1$ fails to meet the latter requirement, since the effect $BS = 1$ does not exist when combining (a) the inclusion in $\vec{W'} \leftarrow \vec{w}$ of the counterfactual assumption Suzy does not throw ($ST \leftarrow 0$), with (b) the inclusion in $\vec{Z} \leftarrow \vec{z}*$ of the fact that Bill does not hit the bottle ($BH \leftarrow 0$), i.e. the fact that his action was pre-empted. If the model is modified in this way, it no longer entails that the bottle is shattered. Finally, the last condition requires that the cause of an effect must be non-redundant, i.e. no subset of it would meet the first two conditions.

> AC3. $\vec{X}$ is minimal; no subset of $\vec{X}$ satisfies conditions AC1 and AC2.

ly could have been the real cause, but due to Suzy's actions was preempted. This is the same as in Suzy's throw analysis, where AC1 and AC3 hold automatically. For AC2, the variables are as follows $\vec{Z} = \{BT, BH, SH, BS\}$, $\vec{W}\{ST\}$, and $w = 0$. While AC2(a) remains the same as in Suzy's analysis, Billy's hit fails to satisfy the sufficiency condition AC2(b). It is not the case that $M(1,1) \models (BT \leftarrow 1, BH \leftarrow 1, ST \leftarrow 1, SH \leftarrow 0)BS = 1$ because we already set the causal model to include the structural dependencies between SH and BH; in other words, it is impossible for Billy to hit the bottle once it has been hit by Suzy.

Despite the scepticism around the hypothetical nature of these manipulations, lawyers are often interested in what would have been the case if one or more elements in the case had been different. For instance, if a nurse had mixed up the doses of anaesthetic before the doctor used the wrong instrument in the operating theatre. Both actions would have caused the patient's death, but one preempted the other. These and similar questions are at the core of many cases of medical negligence, which are becoming increasingly difficult to analyse without a causal framework, especially with questions relating to duty of care.

While HP's 'actual causality' is an advanced approach and is able to model the examples of overdetermination and preemption, it is highly technical and has

not yet been translated into a workable version for legal reasoners. Our aim, as part of the development of the semi-formal argument framework, is to translate these causal theories into the language of argument schemes proposing a best-fit approach in the analysis of causal arguments. Without the incorporation of the formal analysis into the language and concepts of legal reasoning, the use of such models remains highly theoretical. We believe that with the increasing complexity of cases brought to the medical courts, a solid framework for causal argument assessment is necessary.

2.3 Causal Arguments: Towards a Semi-formal Approach

So far we have looked at three major theories that establish cause-in-fact through various forms of logic. These theories differ from each other in their levels of restriction in defining causes and the complexity of the tests themselves, i.e. their effectiveness in each of the causal situations. While all three approaches are able to capture the cause-in-fact in direct causal scenarios, the hard cases of causality require broadening the causal criteria from the notions of pure necessity to sufficiency and contingency-based analysis. As illustrated by the firing squad and rock-throwing examples, NESS and 'actual causality' further distinguish causal subsets that separate hypothetical from the real. Moreover, only HP 'actual causality' is able to capture the subtleties and order of events characteristic to preemptive causes. In these problem examples we observed that there is a cost in choosing one theory over the others. In particular, the 'but-for' test is easy to apply, but it fails to capture the complexities of anything beyond non-direct causal links. By contrast, the more complex theories are well-formalised and provide more accuracy in capturing the cause, but remain counterintuive and cumbersome to apply. Furthermore, it remains controversial whether the increased accuracy of these approaches is a reasonable trade-off for the efforts in applying them in law. Currently, the but-for test remains the dominant test for causality in law, while causal sufficiency is discussed only in a limited set of legal cases. None of the advanced theories are in themselves optimal for analysis of causation in law at their current form. In particular, we believe that these theories could be integrated in a richer framework that addresses the specificity of legal reasoning and reasoning from expert witness testimonies.

We have observed that the analysis of causal arguments in law is a multilayered process and involves establishing and then assessing the cause-in-fact based on a number of criteria. One of the issues flagged by the judges and legal scholars [17] is the blurred division between causal analysis based on evidence itself and assessment based on legal norms and policies. Such a distinction is crucial in events of complex causal phenomena. For instance, in the case of preemption the results based on pure formal analysis would show that only the first actor is the cause of the effect, which is counterintuitive to legal thinking where an actor who has completed the action with intention to do harm should also be held responsible. Care should be taken in translating a counterintuitive theory in legal terms, as there is a growing scepticism of the formal theories in law. Yet, law already contains loaded terms such as 'legal cause', 'proximate cause', and

'material contribution' that go beyond the cause-in-fact analysis and represent policy-based considerations. All three causal theories make direct references to their potential use in law, but the mixed nature of the causal reasoning in law is either missed or underrated. When proposing our framework, we acknowledge that the modelling and evaluation of cause-in-fact is the first step in the causal analysis in law. It is followed by a long list of criteria, including legal rules, policies, and precedent cases to determine the liability attribution. In addition, it is important to appreciate the limits of human reasoning, such as explicit and implicit biases [20], and over-reliance on commonsense reasoning. Law necessarily imposes constraints on the final outcome of the causal analysis for the purposes of decision making. However, the uncertainty in legal reasoning should not (entirely) undermine evidence based reasoning and formal analysis.

We propose the use of a semi-formal causal argumentation framework to narrow the gap between the formal causal theories and legal analysis. Such a framework has two main functions. Firstly, it models the causal arguments based on the language used by the legal reasoners in the case [21]. We abstract from the case record and model the arguments using a notion of causal rules along with strict and defeasible inferences. We use the causal rules and strengths of beliefs about the causal links based on the arguments by the parties in the case. Secondly, with an eye towards future developments of the framework, we propose an extension that allows to assess the causal arguments based on a unique selection of criteria. These criteria include a list of causation specific critical questions, evidential reasoning theories to assess the explanatory value of the arguments, which weigh the arguments based on the notion of normality, and new task specific argumentation schemes designed on the basis of the three causal theories analysed in this paper. The novelty of this proposal is in the way it assesses causal arguments. In addition to the existing approaches to argument analysis, we translate the formal causal theories into argument schemes to assess the underlying causal structures of the arguments presented in the courts. A correct application of the framework will result in further justification and increased accuracy of the judge's reasoning when assessing the cause-in-fact in the cases.

In the next section, we introduce the legal criteria, facts, and expert witness testimonies of the *Althen* case. We then provide our framework for modelling causal arguments, which is then applied to the *Althen* case.

2.4 The Althen Case

We use the *Althen* decision [1] for analysing the evidentiary and legal issues regarding causation. In particular, we examine the expert witness testimonies about the causal link between the vaccines and petitioner's symptoms. Furthermore, we analyse how the special master compares and weights these arguments to attribute legal responsibility. Our aim is to show the complexity of establishing direct causation in a real case. We are particularly interested in unpacking the legal criterion that requires evidence of a 'logical sequence of cause and effect'. We claim that our semi-formal model is able to support the analysis by the expert witnesses in satisfying this criterion.

Legal Criteria. In the US Vaccine Courts, a causal link is successfully proven if the vaccine is either listed on the approved 'Vaccine Injury Table' or the petitioner satisfies the cause-in-fact conditions set out by the court (also know as, the off-table vaccine injury). This case involves an off-table vaccine injury. The claimant's burden (also known as the Stevens test) can be summarised as follows: "the claimant has to show by preponderant evidence that the vaccination brought about her injury by providing a medical theory causally connecting the vaccination and the injury, a logical sequence of cause and effect showing that the vaccination was the reason for the injury, and a showing of a proximate temporal relationship between vaccination and injury; if the claimant satisfies this burden, they are entitled to recover unless the government shows, also by a preponderance of evidence, that the injury was in fact caused by factors unrelated to the vaccine".

Facts of the Case. The case concerns Mrs. Althen (petitioner) and her worsening health conditions after receiving a tetanus toxoid vaccine (TTV). Prior to the vaccine, petitioner was reasonably healthy. After roughly two weeks from the vaccination date, petitioner started reporting various symptoms ranging from blurred vision, to steady headache and temporal loss of vision. Patient's health complaints and hospital visits continued over the following three years. Petitioner was exposed to various types of treatments and extensive medical testing (MRI, EEG, blood tests) that showed inconclusive results for acute-disseminated encephalomyelitis (ADEM), multiple sclerosis, and vasculitis. Subsequently petitioner applied for compensation at the Vaccine Court, which requires that petitioner establishes a causal link between the TTV and ADEM.

Expert Witness Testimonies. There were three medical expert witnesses assigned to this case, of which we discuss two competing testimonies. Dr. Smith for petitioner arguing for the positive causal link between TTV and ADEM, and Dr. Safran for respondent rejecting the causal link. While both experts agreed that it is theoretically possible that the TTV could cause ADEM, they did not agree on the general model being satisfied in this particular case. The main disagreement between the experts was about the role of the TTV in demyelinating the healthy cells in the patient (potentially leading to the ADEM). Dr. Smith supported his opinion by referring to the medical theory of cell degeneracy, which shows cell modification through evolution and can explain T cell reaction after a vaccine. The theory of cell degeneracy is based on the premise that TTV can attack and destroy both bad (antigen) and good (myelin) cells, as the antigen and myelin are *sufficiently similar*. Dr. Safran denies the link in this particular case on two grounds. Firstly, he argues that antigen and myelin cells are not proven to be sufficiently similar to be mistaken by the active components of the vaccine. Secondly, the symptoms observed on the patient can be better explained by multiple sclerosis. Based on this information, which has been abstracted from the case, we will model the causal explanations given by the experts. We chose the focus of the expert arguments due to their significance in decision making at the vaccine courts. Furthermore, these testimonies provide detailed information on the causal claims made in the case, which are then carefully examined and

weighted by the special master to decide determine the responsibility attribution in the case.

3 Semi-formal Framework and Case Analysis

In this section, we present a semi-formal framework for modelling and assessing arguments about causal links. We then apply this framework to the *Althen* case to capture the views expressed by Dr. Smith and Dr. Safran on the causal relationship between TTV and ADEM. Our analysis is meant to accommodate the arguments about the causal relations that are presented by the expert witnesses, in particular we focus on the disagreements between the parties and the underlying structure of the arguments. Other matters are out of scope at this moment, e.g. the dynamics of dialogue, belief change, and time. It is a static model that represents all the information available. It makes explicit some of the otherwise implicit assumptions that are highlighted in the course of the presentation of the expert testimony and by way of attack.

3.1 Semi-formal Framework

Our semi-formal framework is a propositional language to represent and reason with basic facts/events and causal relations between them. Our model consists of basic knowledge structures and rule schemes using these structures.

Basic Knowledge Structures. There are three basic knowledge structures: factual propositions, predicates for similarity and evidentiality, causal links, and inference rules.

Factual Propositions. A factual proposition has the form

$$\pm H(X)$$

where X is a positive or negative literal. $H(X)$ states that the literal X *holds* and $\neg H(X)$ that this was not the case. For instance $H(TTV)$ and $H(\neg TTV)$ are such literals, affirming respectively, that it holds that tetanus toxoid vaccination was performed, and that it holds that this was not the case. We assume that $H(X)$ and $H(\neg X)$ are incompatible, and that $H(\neg X)$ is indeed equivalent to (it strictly implies and is implied by) $\neg H(X)$. For instance $H(\neg TTV)$ is equivalent to $\neg H(TTV)$

Similarity and Evidentiality. In addition to the factual propositions, the experts in the case also reason with similarity and evidentiality, which we introduce as a relation and a predicate, respectively. We represent similarity as

$$Sim(X, W)$$

which is understood as the propositional content of X is similar to the propositional content of W (and vice versa). We represent evidentiality as

$$EV(X)$$

which is understood to represent the existence of evidence for the propositional content of X.

Causal Links. Causal links are represented as

$$C(L, X, Y)$$

where L takes values 1 or 2, X and Y are factual propositions. $L = 1$ indicates that cause X always (necessarily) produces effect Y, and $L = 2$ indicates that cause X usually (normally) produces effect Y. The causal links and levels of causal links loosely correspond to the language used by the reasoners.[5] For instance, $C(1, TTV, TCellAct)$ is an expression that TTV and $TCellActivation$ have the *always causal* relation, as the witnesses present it.

Inference Rules. We use strict and defeasible inference rules represented as

$$X_1 \dots X_n \rightarrow Y$$

for strict rules, and

$$X_1 \dots X_n \Rightarrow Y$$

for defeasible rules, where $X_1, \dots X_n, Y$.

We assume that strict rules allow for contrapositive reasoning. For our purposes it is sufficient the contrapositive inference where, given all antecedents of a defeasible rule except X, and the negation of the rule's consequent, we can infer X's negation. Thus we assume that a strict rule stands for the set of all of its contrapositions. A contrapositive of a strict rule

$$X_1 \dots X_n \rightarrow Y$$

is a rule

$$X_1 \dots X_{i-1}, \neg Y, X_{i+1}, \dots X_n \rightarrow \neg X_i$$

For instance, if a knowledge base contains the rule

$$H(X) \wedge C(1, X, Y) \rightarrow H(Y)$$

[5] At the initial stages of the case study, we annotated the decision identifying causal and accompanying hedging expressions. After identifying the main causal links in the case, we ranked the various expressions in two levels of strength. For instance, *'a probable causal relation between tetanus toxoid and two injuries', 'it is more probably than not the case that tetanus toxoid can cause the injuries suffered here'* are examples of level 2 (usually causal) support.

it also contains a rule

$$\neg H(Y) \wedge C(1, X, Y) \rightarrow \neg H(X)$$

Accordingly, if Y does not hold and X strongly causes Y, we can infer that X does not hold, for otherwise Y should hold.

To capture the reasoning of the experts in our cases, we need to supplement the general framework above with some schemes for rules.

General Rule Schemes. There are general rule schemes for causal rules, similarity, and evidence (including abduction).

Causal Rules. Rule schemes R1 and R2 capture the inferential relevance of causal relations

$$[R1.]H(X) \wedge C(1, X, Y) \rightarrow H(Y)$$

captures the inferential meaning of necessary causal links. If the precondition holds and the causal link is level 1, we strictly conclude that the effect holds.

$$[R2.]H(X) \wedge C(2, X, Y) \Rightarrow H(Y)$$

captures the inferential meaning of defeasible causal links. If the precondition holds and the causal link is level 2, we defeasibly conclude that the effect holds, but can have exceptions.

Similarity. The case scenario also includes reasoning with similarity, in particular, it is assumed that similar facts cause the same effect. This is captured by the following scheme for defeasible conditionals:

$$[R3.]Sim(X, W) \wedge C(L, X, Y) \Rightarrow C(L, W, Y)$$

Evidence. Finally, our experts reason with evidence. First, it is assumed that having evidence for a proposition defeasibly implies that this proposition holds:

$$[R4.]EV(X) \Rightarrow H(X)$$

Evidence plays two roles in our domain. First, it may *establish facts that contradict the conclusion of a causal inference*. This will defeat the causal argument leading to the conclusion contradicted by the evidence. If that argument culminates with the effect of a level 1 causal relation, defeat may extend to the precondition of that causal relation via contraposition.

Second, evidence may provide *abductive support for establishing the antecedent of a level 1 causal relation*. In fact, it seems that our experts assume that given a necessary causal relation and its effect, we can abductively infer the precondition. We capture this aspect of their reasoning through the following pattern defeasible conditionals:

$$[R5.]H(Y) \wedge C(1, X, Y) \Rightarrow H(X)$$

meaning, that we can defeasibly infer the precondition X when we have established its effect. For instance, given evidence of certain symptoms, we can infer that these symptoms hold by R4; moreover, if such symptoms are strictly caused by an illness, we can defeasibly infer that the illness holds by R5.

3.2 Models in the Language

In this section we apply the semi-formal framework to represent the relations as described by the expert witnesses in the case. We shall use the following atoms, representing the circumstances that are relevant to our case:

Factual Atoms

TTV - tetanus toxoid vaccination being injected in the patient
$ADEM$ - acute-disseminated encephalomyelitis illness
MS - multiple sclerosis
$TCellAct$ - the chemical process of tetanus toxoid vaccination activating T cells
$AntigDestr$ - antigen cells destroyed (T cells should target this, wanted effect)
$MlnDest$ - myelin cells destroyed (T cells should not target this, unwanted effect)
$Symp(Mono)$ - monophasic symptoms occur. This means that the symptoms that occurred just once. $-Symp(Mono)$ means here that the symptoms occurred on multiple occasions.

We use the following causal links to represent the causal claims of the expert witnesses:

C1. Tetanus toxoid vaccination always causes T-cell activation: $C(1, TTV, TCellAct)$
C2. T-cell activation always causes antigen destruction: $C(1, TCellAct, AntigDestr)$
C3. T-cell activation usually causes myelin destruction: $C(2, TCellAct, MlnDestr)$
C4. Myelin destruction usually causes ADEM: $C(2, MlnDestr, ADEM)$
C5. ADEM usually causes monophasic symptoms: $C(2, ADEM, Symp(Mono))$
C6. Multiple sclerosis usually causes recurrent symptoms: $C(2, MS, -Symp(Mono))$

3.3 The Arguments of the Expert Witnesses in *Althen*

We now model the arguments of Dr. Smith and Dr. Safran in *Althen*.

Dr. Smith Given the language and rules above, we model the reasoning by Dr. Smith. He argues in favour of a causal link between the TT vaccine and ADEM. His argument starts from the explicit assumption of TTV (given as a fact). Dr. Smith's argument is based on the explicit assumption $Sim(AntigDestr, MlnDestr)$ and implicit assumption $C(2, ADEM, Symp(Mono))$ (an assumption exposed

by another witness's counter-argument). We take these assumptions as assertions in Dr. Smith's model. We give the assumptions, rules, and inferences in sequence, labelling the reasoning steps by agent. The relations have been annotated leading back to the general rule schemes (R1-R5) and expert witness claims (C1-C6).

Dr. Smith's Model: The rules for Dr. Smith's argument, based on the rules proposed above

1. $H(TTV)$ [explicit assumption]
2. C(1, TTV, TCellAct) [from C1]
3. $H(TTV) \wedge C(1, TTV, TCellAct) \rightarrow H(TCellAct)$ [strict implication following the causal rule R1]
4. $H(TCellAct)$ [from 1, 2, and 3]
5. $C(1, TCellAct, AntigDestr)$ [from C2]
6. $Sim(AntigDest, MlnDest)$ [implicit (similarity) assumption by Dr.Smith]
7. $Sim(AntigDest, MlnDest) \wedge C(1, TCellAct, AntigDestr) \Rightarrow$ $C(2, TCellAct, MlnDestr)$ [an instance of the rule scheme R3 showing defeasible inference]
8. $C(2, TCellAct, MlnDestr)$ [from 5, 6, 7]
9. $H(MlnDestr)$ [explicit assumption]
10. $C(2, MlnDestr, ADEM)$ [from C4]
11. $H(MlnDestr) \wedge C(2, MlnDestr, ADEM) \Rightarrow H(ADEM)$ [defeasible inference following the causal rule R2]
12. $H(ADEM)$ [explicit assumption by Dr. Smith]
13. C(2, ADEM, Symp(Mono)) [from C5, causal claim by Dr. Smith]
14. $H(ADEM) \wedge C(2, ADEM, Symp(Mono)) \Rightarrow H(Symp(Mono))$ [defeasible inference following the causal rule R2]
15. $H(Symp(Mono))$ [from 12, 13, 14]

To support the structural claims, Dr. Smith can also build an argument for ADEM, this time based on the evidence of the symptoms.

1. $EV(Symp(Mono))$ [explicit assumption]
2. $EV(Symp(Mono)) \Rightarrow H(Symp(Mono))$ [following R4]
3. $H(Symp(Mono))$ [defeasible conclusion from 1, 2]
4. $C(2, ADEM, Symp(Mono))$ [from C5]
5. $H(Symp(Mono)) \wedge C(2, ADEM, Symp(Mono)) \Rightarrow H(ADEM)$ [following the abduction rule R5]
6. $H(ADEM)$ [defeasible conclusion from 4,5]

Dr. Safran Dr. Safran's objections against the causal link between the TTV and ADEM can be summarised into two main arguments. Firstly, he argues against Dr. Smith's assumption (see steps 6-8 in Dr. Smith's model) that myelin and antigen cell destruction are sufficiently similar for the vaccine to make an error in distinguishing between these processes.

This argument consists in the single claim

1. $\neg Sim(AntigDest, MlnDest)$

Secondly, Dr. Safran argues against the conclusions based on the assumption of monophasic symptoms. He claims that the evidence shows that the symptoms observed on the patient are reoccurring. He provides further support from the medical literature showing that $ADEM$ has been described as a monophasic condition, consequently defeating Dr. Smith's argument.

His argument against $ADEM$ is is the following:

1. $EV(C(1, ADEM, Symp(Mono)))$ [explicit assumption by Dr. Safran]
2. $EV(C(1, ADEM, Symp(Mono))) \Rightarrow C(2, ADEM, Symp(Mono))$ [following R4]
3. $C(2, ADEM, Symp(Mono))$ [from 1,2,3]
4. $\neg H(Symp(Mono))$ [explicit assumption]
5. $\neg H(Symp(Mono)) \wedge C(1, ADEM, Symp(Mono)) \rightarrow \neg H(ADEM)$ [by transposition of the rule $H(ADEM) \wedge C(1, ADM, Symp(Mono)) \rightarrow H(Symp(Mono)]$
6. $\neg H(ADEM)$ [defeasible conclusion from 4,5]

Furthermore, Dr. Safran provides an alternative explanation of the recurrent symptoms linking them with multiple sclerosis.

1. $C(2, MS, \neg Symp(Mono))$ [from C6]
2. $H(\neg Symp(Mono))$ [explicit assumption]
3. $H(MS) \wedge C(2, MS, \neg Symp(Mono)) \Rightarrow H(\neg Symp(Mono))$ [defeasible inference from R2]
4. $H(MS)$ [defeasible conclusion from 1, 2, 3]

Multiple sclerosis provides an explanation of the evidence, as he sees it, i.e. as including non-monophasic (recurrent) symptoms $H(\neg Symp(Mono))$.

In summary, following the burden of the proof rules in *Althen*, Dr. Smith had to provide an evidentially supported argument about the causal relationship between the TT vaccine and ADEM. He provided such a model based on the similarity assumptions between the cells and further implicit assumptions about the monophasic symptoms and ADEM. For the causal proof to be shown insufficient, the proponent's expert witness either had to defeat Dr. Smith's model or provide an alternative causal explanation of the facts. Based on the arguments presented, we modelled both instances defeating the causal link between TTV and ADEM. It is important to emphasise that in the real case the cause-in-fact analysis constituted only the first step for the liability attribution and ultimately the case was decided against the claimant due to the lack of supportive evidence for the causal link between the vaccine and ADEM in the medical literature.

3.4 Interim Conclusions

The case analysis so far provides an illustration of a use of our semi-formal framework and its ability to capture and model the core points of the expert

witness arguments in the *Althen* case. In addition to modelling the causal relations and their relative strengths, we have begun the integration of evidential reasoning from the evidence to a supported conclusion. A carefully developed evidential reasoning implementation could be especially helpful in legal reasoning where the parties are expected to justify their arguments by showing how the evidence links to their claims. We also observed that there is a mix of everyday and legal causal expressions with causal language often being accompanied by hedging expressions. We integrated different degrees of belief in causal links to represent the nature of discussions in the court, where the expert witnesses and decision-maker often use the language of uncertainty. This suggests a possible extensions based on statistical approaches to further improve the causal models to reflect causal reasoning present in the courtrooms.

4 Discussion and Future Work

In the final section of this paper, we discuss the need for a multilayered approach to causal analysis in law and introduce an early proposal for building an extension to the current framework. Such an extension would include assessment criteria based not only on the critical question [27] and evidential reasoning [3] approaches, but would also include a novel integration of the but-for, NESS, and 'actual causation' theories to assess the strength of the causal arguments.

In this paper, we presented the initial steps in the development of a comprehensive modelling and assessment framework designed for causal argument analysis in law. The interim results of modelling the vaccine injury case showed that the basic notions introduced are easily transferable from argument to argument. It is also possible to substitute some of the basic relations to suit the case at hand. For instance, in *Althen's*, the disagreement stemmed from the similarity relation (Sim) between the cells. In other vaccine injury cases, the core disagreement might rely on a different notion, e.g. compatibility of certain chemical compounds of different vaccines. In contrast, the framework's causal and evidential rule schemes can be directly reused and generalised for other cases, providing that the new case specific variables are added to the models. The argument modelling in the semi-formal framework successfully uncovered a list of hidden premises and implicit inferences, and it provided additional support for the judge's decision. However, modelling in itself is not enough to assess and compare competing arguments.

As it currently stands, causal analysis in law still poses a lot of unanswered questions and there are no comprehensive guidelines for causal argument analysis in the courts [1]. The absence of a common assessment framework of complex notions like *overdetermination*, *preemption*, and *omission* can result in reduced consistency between judgements, increased level of subjectivity and lack of transparency. We believe that better understanding of the causal links and causal arguments would allow to improve these guidelines. Moreover, causal arguments also play an important role in the liability attribution and burden of proof debates [22].

Therefore, we share some ideas for an extension of our framework to include a list of argument assessment criteria. These would be based on the argument schemes and critical questions [27], evidential reasoning theories [3], norm based analysis, and an integration of causal theories discussed in this paper (*but-for*, NESS, and HP's *'actual causation'*). The latter is a novel proposal in legal analysis as it would support an interdisciplinary approach to causal argument assessment in decision making. As we have shown in the causal theory section (Section 2.2), each of the causal approaches has certain advantages in capturing the causes and effects. Our aim is to translate the intuitions and formalisations of the NESS and 'actual causation' three prong test into argument schemes that use the basic notions of ASPIC$^+$ [16], enabling inferences from the knowledge base. These argument schemes will be used to assess the underlying logical structure [5] of causal links and how well the particular causal arguments are justified. We acknowledge that cause-in-fact is not the sole source of arguments in legal decision-making and strive for a framework that would also support norm-based analysis. One of the aims is to unpack some of the causal logic behind the policy based considerations of 'legal cause' and 'material contribution', which have been claimed to remedy a strict application of logic [17].

The goal of this research is to develop an approach that identifies, formally models, and supports reasoning with causation in a manner that is relevant to the legal domain. This paper presents the modelling steps of the overall research objective as identified in [7]. In this paper, we have mainly focused on representing simple causal relations that are expressed through complex arguments in the court. The proposed inference rules can be reused to model similar cases in the vaccine court, and have the potential to model a large variety of causal issues. We also assessed three dominant causal theories modelling the underlying logical structure of causation. This analysis serves as an initial step for building an extension to the semi-formal framework. As part of the future work, we intend to use the methods and insights developed here to tackle more challenging causal issues. Furthermore, we have begun building an extension to provide a way to compare and assess the causal arguments. We aim to integrate the causal tests of the three theories and the modelling framework discussed in this paper. In its current state, the framework represents the causal links as understood by the expert reasoners and the judge, while the causal theories discuss how the logical structure of such links can be assessed. There is a lot more that needs to be done to understand and automate the arguments and reasoning based on causal models. However, in accord with the other specialists in the field [14] we believe that it is work worth pursuing in order to understand the complex nature of legal reasoning and argumentation while respecting the practical constrains of legal discourse.

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