Article

Causation in Mixed Methods Research: The Meeting of Philosophy, Science, and Practice

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Abstract

This article provides a systematic and pluralistic theory of causation that fits the kind of reasoning commonly found in mixed methods research. It encompasses a variety of causal concepts, notions, approaches, and methods. Each instantiation of the theory is like a mosaic, where the image appears when the tiles are appropriately displayed. This means that researchers should carefully construct a causal mosaic for each research study, articulating what is causally relevant given their particular research questions, purposes, contexts, methods, methodologies, paradigms, and resources. Our theory includes 11 propositions that can guide researchers addressing causation.

Keywords

causal pluralism, mixed methods, theory of causation

The purpose of this article is to articulate how mixed methods research (MMR) can address the timeless issue of cause-and-effect or causation. Although the issue of causation has been extensively articulated for quantitative research (e.g., Shadish, Cook, & Campbell, 2002) and qualitative research (Maxwell, 2004a, 2004b, 2012a), causation has not been systematically articulated yet for MMR, except with regard to the relevance of critical realism for causation (see Maxwell & Mittapalli, 2010). We believe that a pluralistic theory of causation is needed in MMR for at least two reasons: (a) causation, in its multiple forms (e.g., local/event-level in qualitative research and general/variable-level in quantitative research), is ubiquitous in the social/natural world, and mixed methods researchers are well situated to advance causal understanding and explanation of the complex, multilayered world in which we exist; and (b) our pluralistic, philosophical theory of causation fits very well with the pluralistic reasoning that is at the heart of much MMR practice. In this article, we attempt to build an integrated theoretical account of causation for MMR that builds on mixed methods reasoning and the quantitative research, qualitative research, and philosophy of social science literatures on causation. Our theory uses the ''both-and'' logic and the ''logic of synthesis/integration'' that are commonly used in MMR

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(see Johnson, 2016; Johnson & Schoonenboom, 2016). These logics are at the heart of the philosophical theory of *dialectical pluralism* (Johnson, 2016), which we recommend for systematically dialoguing with the many, very important, theories, paradigms, and poles on various dichotomies discussed in this article.

We specifically articulate the following: What are the key dimensions of causation? What are the major theories and concepts of causation? How is causation used in quantitative research? How is causation used in qualitative research? and How can we move toward a new theory of causation inspired by MMR? Causation *is* relevant in many, if not most, MMR research studies, and we therefore need a more systematic way of thinking about causation.

A Causal Mosaic for Social Research

In the past decades, the philosophy of causality developed a rich battery of concepts, notions, and accounts of causation (e.g., Illari and Russo [2014] discuss more than 20 of these accounts, including the most classic references such as Wesley Salmon, Nancy Cartwright, and James Woodward). Almost invariably, all of the proposed causal theories, when used in isolation, have been criticized. A notable example is the counterfactual analysis of David Lewis, against which many counterexamples have been designed. Although many of these counterexamples are correct, a qualified pluralist approach should also explain why counterfactuals remain a useful tool for hypothesis generation and testing (for details, see Illari and Russo, 2014, Chapter 9). Similarly, the Salmon–Dowe approach, based on causal processes, is arguably tailored to physics and not very illuminating about biology, or other fields. This prompted the development of other accounts, for instance a mechanistic one (see Illari and Russo, 2014, Chapters 11, 12). Faced with the variety of concepts offered, the philosopher of science is left with two options: either continue searching for *The-One* concept of causality or attempt to understand what links these concepts together in a coherent account. This is the idea of the causal *mosaic* (Illari and Russo, 2014): Causal concepts are like tiles that, put next to one another, and in the right way, will let an image emerge. And the image will be a sophisticated causal theory. So, the question is how to arrange the tiles, in order to create a recognizable and useful image for each research study.

On the one hand, one should distinguish different *philosophical* questions/assumptions about causation: The *metaphysics* of causality is interested in what the nature of the relation is, or what causal relata are; *epistemology* and *methodology* are concerned with how we reason causally, what notions guide model building and model testing, what models should be constructed and empirically tested, depending on the research question at hand; the *semantics* of causal claims has to do with the meaning of cause/causality, which may be context sensitive; finally, questions about *use* of causal knowledge are essential to those domains where actions and interventions are important. On the other hand, one should distinguish different *scientific* questions/assumptions about causation: *Inference* is about whether, and with what degree, C causes E (C=cause and E=effect); *explanation* is about *how* C causes E; *control* has to do with methods to limit the action of confounders; and *reasoning* pertains to the logic behind the causal methods.

Once the philosophical and scientific questions are specified, one can then ask more domain-specific questions for the particular research study. There is no one single causal mosaic, but there are as many as we need to build, on a study-by-study basis, according to the specific research domain, questions, goals, assumptions, context, and methods and methodological approaches. It is also on a case-by-case basis that one can decide which aspects or elements of the causal mosaic are more relevant or carry more weight. The merits of any of these decisions are to be evaluated on *pragmatic* grounds and keeping in mind two important objectives of causal analysis: We want to obtain an understanding of what the world is like, in order to do

something about it. Taken singularly, none of the existing causal accounts, even the more prominent ones, can achieve what a mosaic view can do.

Some Dimensions of Causation in the Philosophy of Social Science

One makes multiple philosophical assumptions when articulating each causal claim. We therefore start by explaining a few causal dimensions to be considered in each research study. We illustrate the dimensions using a classic example from Miles and Huberman (1994; Example 1).

The first dimension is *singular* versus *general* causation. This distinction is seen in the approximate synonyms of token/single-case/local/idiographic/individual-level causation versus type/generic/nomothetic/group-level causation. Two examples of singular causation are ''That particular iceberg caused the Titanic to sink''; and ''The poor treatment of employees over the past 6 months by President Steve Johnson is one of the causes of the employees' recent observed and reported dissatisfaction with management at ZIPP Corporation headquarters in Rochester, Michigan.'' The claim is that a singular instance is real and, perhaps, is a unique causal event. Singular/local causation is of special interest to qualitative researchers because qualitative research is focused on understanding *particular* groups, places, events, phenomena, and so on.[1]

In contrast to singular causation, general causation addresses how phenomena operate generally, apart from particulars, especially at the abstract level of variables. Some examples of *general* causation are ''Smoking causes lung cancer'' and ''Proximity leads to decreased prejudice and discrimination.'' General causation is popular in quantitative research where researchers routinely examine statistical relationships with a search for regularity, invariance, and replicability at the general/abstract level of analysis. In sum, singular causation operates at a local level and general causation operates at the abstract level of variables. In Example 1, the case-oriented analysis focuses on singular causation: Its purpose is to understand how one individual, Nynke van der Molen, came to decide to enroll in veterinary studies. The variable-oriented analysis focuses on general causation: It describes the factors that in general affect people's decisions to attend a university.

General causation can be determined using traditional statistical modeling approaches showing average effects or from the results of multiple singular (within case) qualitative models by creating cross-case models using MMR techniques such as diagrams, matrices, and models (Miles & Huberman, 1994.)[2] *According to our pluralistic theory of causation for MMR presented here, having general and local causal understanding is important for truly understanding the phenomena under scrutiny.* This theoretical proposition fits the mixed methods perspective of obtaining a maximal understanding of one's research object (Creswell & Plano Clark, 2011; Johnson, 2016).

A second philosophical dimension underlying causation is determinism on one pole versus indeterminism on the other pole. Strong determinism is the view that every event in the past, present, and future was/is/will be *fully* caused, and, therefore, there is only one possible instantiation of the world. Strong *in*determinism is the view that the world is mostly nonregular and complex and not at all predictable. Moderate indeterminism or soft determinism is perhaps more in line with mixed methods thinking—this is the view that there is some regularity and some ''free play'' or probability operating in human and natural worlds. In the variable-oriented analysis of Example 1, emphasis is placed on what is constant among cases. It is deterministic to the extent that it is assumed that the factors that influence the decision to attend a university and their interrelations will also be effective in future cases (although they might change as society changes). The case-oriented analysis, on the other hand, emphasizes the fact that Nynke has a choice, and thus that different outcomes may be possible. In addition, it places an emphasis on unique events that had an influence on Nynke's decision, again suggesting the possibility of a

Example 1. Nynke van der Molen's Decision to Attend University.

In a *variable-oriented* analysis . . . [w]e'd see that deciding to attend university is mainly influenced by school performance, which is boosted by parent expectations (pushed up by SES); they also have a direct effect on the university decision. Peer support has a small effect as well. In a *case-oriented* analysis . . . we need to look at a full story of case #005: Nynke van der Molen, whose mother trained as a social worker but is bitter about the fact that she never worked outside the home, and whose father wants Nynke to work in the family florist shop. And we need to invoke chronology: Nynke's closest friend, Leonie, decided in the first semester of 1989-1990 to go to university, before Nynke started work in a stable. That occurred about when her mother showed her a scrapbook from social work school—a moving eye-opener for Nynke—and preceded Nynke's decision in the second term to enroll in veterinary studies. These and other data in the story . . . would let us begin to trace the flow and configuration of events to see how Nynke came to make her decision. (Miles & Huberman, 1994, p. 173)

different choice, if these events had not occurred. Finally, it shows that different persons, Nynke and Leonie, may make different decisions.

According to our pluralistic theory of causation for MMR, it is helpful to be open to the ideas in both determinism and indeterminism as one studies the world. This theoretical proposition fits the mixed methods concept of listening to both sides of dichotomies and obtaining useful balances and third perspectives (Johnson, 2016; Johnson & Gray, 2010).

A third philosophical causal dimension of interest is nominalism versus universalism or alternatively labeled individualism versus holism (see e.g., Zahle & Collin, 2014). Nominalism claims that only *particulars* are real (e.g., Mary, a particular triangle, a particular dog named Spot). Analogously, individualism in social science holds that social phenomena are to be explained in terms of the action and behavior of single individuals. Universalism claims that *universals* such as triangles and classrooms, and larger and abstract entities such as variables (gender, social class, nations) exist in addition to particulars. Analogously, holism holds that social phenomena need to be explained by appealing to factors and forces at the social level, and that constrain or determine the action and behavior of individuals. Qualitative research writing shows a penchant for nominalism (in reporting findings for particular people, groups, places) and quantitative research writing shows a penchant for universalism (in the form of variables). In the case-oriented analysis of Example 1, the focus is on particulars, on one particular individual, Nynke van der Molen. In the variable-oriented analysis in Example 1, the focus is on universals, on ''deciding to attend university,''''school performance,''''parent expectations,''''socioeconomic status (SES),'' and ''peer support.''

According to our pluralistic theory of causation for MMR, it is helpful to be open to understanding both realism/universals and nominalism/particulars. This theoretical proposition fits the mixed methods concept of listening to both sides of dichotomies and obtaining useful balances and third, integrated perspectives (Johnson, 2016; Johnson & Gray, 2010).

A fourth philosophical dimension of causation is seen in what Shadish et al. (2002) call molar causation versus molecular causation. *Molar* causation is causation produced by a whole or a package such as an intervention program. *Molecular* causation is direct causation(s) produced by specific parts of the whole. For molecular causation, one would need to ''unpack'' the whole and determine what component parts are causally active. In Example 1, it was stated that SES influences student performance. That's a molar causal claim. A molecular analysis would attempt to unpack SES into its component parts, determining their separate possible/relative influences (i.e., social status, education, and income). *According to our pluralistic theory*

of causation, it is important to understand both molar and molecular causation. This theoretical proposition fits the MMR goal of obtaining fuller and better understanding of causation.

A fifth philosophical dimension of causation is seen in what Shadish et al. (2002) call causal description versus causal explanation. *Causal description* is what is readily obtained using randomized controlled trials (RCTs), that is, a description of changes in ''program-package X'' causing changes in the dependent, Y, variable(s) of interest, without evidence of underlying mechanisms. *Causal explanation* is present when the black box is transformed into a ''clear box,'' where we can see the intervening processes that produce the outcome (Scriven, 1994). This process involves understanding intervening/mediating variables (e.g., C → M → E), and moderator/interaction variables, where the C → E relationship ''depends on'' or varies across the levels of the moderator variable

More generally, causal *explanation* addresses the questions of how and why a cause C produces an effect E. Qualitative research can help in understanding causal explanation through observations, interviews, and case studies (Blatter & Haverland, 2012; Johnson & Schoonenboom, 2016; Langley, 1999; Maxwell, 2012a, 2012b). Example 1 starts from the well-known causal description that SES has an influence on university attendance. Both variable-oriented and case-oriented analysis can provide a causal explanation. The variable-oriented analysis does so by showing that the influence of SES on university attendance is mediated by parent expectations and school performance. The case-oriented analysis explains the time-ordered circumstances in which an individual in an environment characterized by a specific SES comes to make her decision to attend a university.

According to the pluralistic theory of causation for MMR, it is important to understand both causal description and causal explanation. This theoretical proposition fits the MMR goal of obtaining a fuller and better understanding of causation on a study-by-study basis.

Major Theories of Causation

In this section, we attempt to (a) show the plurality of causal theories available to mixed methods researchers and (b) provide *Journal of Mixed Methods Research* readers with some powerful options for thinking about causation. The different causal theories are divided into two broad groups: accounts of difference making versus accounts of production. We also provide a description of agency causation. All of these provide conceptualizations of what we seek *evidence of*. Accounts of *difference making* emphasize the importance of determining *that* a cause C makes a difference to (occurrence of) the effect E. Accounts of causal *production* emphasize the importance of identifying processes/mechanisms that connect the cause (C) and the effect (E). The next proposition of our theory of causation is that *in many MMR studies, it is helpful to have evidence of both difference making and causal production*. This fits with the MMR both-and logic and the logic of obtaining multiple sources of evidence for producing stronger, more warranted assertions. There are several varieties of difference-making and production causation. We will illustrate these varieties, using a real-life example (Example 2), which has been simplified for demonstration purposes.

Difference-Making: Probabilistic Causation

Probabilistic causation is very popular in current quantitative social/behavioral science. The key idea is as follows: Ceteris paribus, C is a cause of E if and only if C changes (i.e., raises or lowers) the probability of occurrence of E. In notational form, it looks like this: C_t causes $E_{t'}$ in P_i iff $Pr(E_{t'}/C_t \text{ in } P_i) \neq Pr(E_{t'}/\text{not-}C_t \text{ in } P_i$; where, Pr = probability; / = given; C = cause; E = effect; P = population; iff = if and only if). Practically speaking, probabilistic causation reflects

the belief of many in quantitative social/behavioral science that the relationships we find are probabilistic rather than universal laws. This is easily seen because we never explain all of the variance in our outcome variables and the average effects given in our equations do not apply to every single individual or group. The first sentence of Example 2 states that providing textbooks can increase the average student test score. This is a form of probabilistic causation: It is not the case that the score of every student who receives a textbook increases, only that, generally speaking and aggregated over all students, the probability of a higher test score increases when textbooks are provided.

Difference-Making: Counterfactual Causation

David Hume defined causation in two ways, and counterfactual causation was one of them. Here is how Hume (1777/1993) put the idea: ''We may define a cause to be an object followed by another [. . .] where, if the first object had not been, the second never had existed'' (p. 51). This idea is further articulated by many others (cf. Kutach, 2014; Lewis, 1973; Rubin, 1974), and is defined as follows: An event E causally depends on an event C just in case (a) if C had occurred, then E would have occurred and (b) if C had not occurred, then E would not have occurred. This formulation is particularly suited to detect singular causation. Yet this intuition—had C not occurred, E would not have occurred either—has been picked up to address general causation, for instance, in the framework of the potential outcome model (Holland, 1986; Rubin, 1986). This made the counterfactual approach quite popular among experimental researchers who emphasize random assignment to treatment and control groups and the use of a no-treatment control group to estimate the counterfactual (i.e., what the participants would have been like, had they not received the treatment condition). The difference between the outcomes for the treatment and control groups is the estimate of the ''net effect'' of the treatment. In Example 2, the finding from the general literature of the presence of a causal effect came from the studies showing condition (a) that providing books to students, C, produced higher student test scores, E (C was followed by E in experimental and correlational studies), and evidence of condition (b) came from the use of control groups in some studies in the literature, specifically, that in the absence of the provision of books, higher test scores were not present.

Difference-Making: Regularity or Regularism

Many current philosophers of science view Hume's regularity theory as a type of difference-making causation (Illari & Russo, 2014; Psillos, 2004). However, what became the ''regularity view'' was also clearly stated by David Hume (1777/1993) when he said,

> We may define a cause to be an object, followed by another, and where all the objects similar to the first, are followed by objects similar to the second . . . [a cause is] an object followed by another, and whose appearance always conveys the thought to that other. (p. 51)

This sort of causal reasoning (i.e., causation as invariable regularity) is seen in the search for *lawful* bivariate and multivariate relationships by some scientists. The belief in laws is commonly found in the natural sciences. In the social sciences, postdating Hume, probabilistic relationships are more commonly encountered. In Example 2, regularity was shown in the empirical literature that showed, more than once, a relationship between provision of text books (C) and increased test scores (E).

Example 2. Providing Textbooks in Primary Schools in Rural Kenya—Glewwe, Kremer, and Moulin (2009), Discussed in Johnson and Schoonenboom (2016).

An intervention study by Glewwe et al. (2009) started from a well-established belief that was grounded in research in various contexts: Providing textbooks to schools where they are scarce can substantially increase the average student test scores. However, in an RCT in rural Kenya, in which primary schools were randomized to treatment condition, this effect was not found. Providing textbooks can only have an effect if children are able to read these books. A qualitative inquiry in the schools, in which children were asked to read the textbooks, revealed that the majority of the children were unable to read these difficult textbooks, written in English, which was not their first language. Further quantitative subgroup analysis showed that there was an effect for high-achieving students, who were able to read their textbooks.

Difference-Making: Necessary and Sufficient Causation

The idea of necessary and sufficient causation usually assumes strong determinism. According to *necessary* causation, the cause must be present for the effect to happen, ceteris paribus. According to *sufficient* causation, a cause is enough, all by itself, for the effect to happen, ceteris paribus. Necessary and sufficient language does not fit probabilistic causation because the ideas follow deductive logic. This approach is nonetheless used, often successfully, in social/behavioral science practice, where practitioners generally do not worry about the problematic notion of strong determinism. The most systematic methodological/analytical approach is found in Charles Ragin's (2014) qualitative comparative analysis (QCA). The idea is to act *as if* determinism plays out in the world and then reason from truth tables and determine approximate necessary and sufficient causes.

A classic idea about necessary and sufficient causation is the so-called INUS condition, the ''insufficient but non-redundant part of an unnecessary but sufficient condition'' (Mackie, 1974, p. 62). In the statement ''the short circuit caused the fire in the living room this morning,'' the short circuit is, by itself, insufficient because additional conditions had to be met for a fire (e.g., presence of oxygen). The short circuit is a nonredundant component of the whole set of conditions, which includes the presence of oxygen. According to Mackie (1974), causes are *at a minimum* INUS conditions, in the sense that they do not 'act' on their own, but always in conjunction with some other conditions.

The INUS condition applies to Example 2 as well. There are many ways in which students' test scores can be raised, such as more time spent on tasks that prepare for the test, more individualized support, or better explanations of the subject matter at hand. Providing text books that children can read is only one of them, and is therefore an unnecessary condition for the effect to occur. Providing text books that children can read is a sufficient condition, which, when applicable, will raise the average student test scores. The sufficient cause ''providing textbooks that children can read'' contains two nonredundant parts: ''providing textbooks'' and ''textbooks that children can read.'' If one of these parts is missing, the effect will not occur: If children are provided with textbooks that they cannot read, or if children are able to read specific textbooks, but these are not provided, then the average student text score will not raise (ceteris paribus).

Difference-Making: Manipulation and Invariance

The manipulationist account of causation can be defined as follows: ''C causes E if and only if, were we to manipulate C, E would also change'' (Illari & Russo, 2014). Strong causal evidence

is seen when, under manipulation, the same result obtains across multiple people and multiple settings; this idea is called ''invariance'' in the philosophy of science and it ultimately means the causal relation can be generalized (for a discussion, see Russo, 2014). This is the kind of causation assumed and promoted by some experimental researchers—they claim that manipulation of the causal variable must, in principle, be possible; otherwise, one must remain silent regarding causation. A sort of either/or logic is being used here: Manipulation equals full causation, no manipulation equals zero causation. Changes in the manipulated causal variable are said to ''explain'' the changes in the effect variable, although what is most often present is causal description (Shadish et al., 2002). A difficulty with the binary viewpoint is that much natural and social science must be conducted on entities/variables that cannot be manipulated for various reasons, making a *universal* requirement of manipulation unreasonable. Despite claims otherwise, scientists can obtain valuable and useful evidence of causality outside of experimental research (Hill, 1965; Johnson, 2001; Russo, 2009, 2011, 2014).

Example 2 can be seen as a weak form of manipulation. The cause (providing textbooks) is manipulated at the individual level: Before the experiment, individual children do not make use of textbooks, and during the experiment, individual children do make use of textbooks. Manipulation is also seen at the group level. The average effect is obtained by comparing what happens with children who make use of the textbooks (the experimental group) with the children who do not make use of textbooks (the control group). Such cases have been called counterfactual causation, but they contain manipulation as well because the independent variable (presence of textbooks vs. no presence of textbooks) is said to be manipulated.

Example 2 also shows a form of invariance: The effect of providing textbooks had been established in various contexts: The same result had been obtained across multiple people and multiple settings. This supports the making of generalizations. But the pluralistic theory of causation reminds us to note that general (average) causation does not mean causation was present for every individual.

Causal Production: Mechanistic Causation

Causal production establishes a connection between events or variables C and E by providing an explanation of *how* C produces E. Such explanations can operate at different levels of abstraction. For an example of lower level mechanisms, individual-level factors might be used to ''explain'' the relationship between social variables—that type of religion affects economic behavior is explained by the individuals and activities making up religion and economic behavior. Mechanisms can also be theorized to operate at the same level of abstraction—type of religion affects collective attitudes, which affect macro-economic behavior.

The mechanisms in Examples 1 and 2 operate at the individual and group levels. Mechanisms at the individual level explain how the effect of an individual's SES on that individual's university attendance is mediated by that individual's parental expectations and the individual's school performance (Example 1). The effect of providing a textbook on an individual's test score (Example 2) is mediated by that's individual's ability or inability to read the textbook. If this individual relationship holds for many individuals, it will also be seen in the group or aggregate level of causation.

In Example 3, a hypothesized mechanism proved to be wrong, and qualitative data uncovered the correct mechanism.

Methodological individualists contend that social factors do not exist apart from their lower level individual constituents (Little, 2011), but holists or methodological collectivists believe that higher level concepts can causally influence one another (Elder-Vass, 2010; Kincaid, 1996, 2014). For the latter, the social concept is said to have an emergent property or causal power;

Example 3. The U.S. Scared Straight Program.

A systematic review has shown that the U.S. Scared Straight program does not work (Petrosino, Turpin-Petrosino, Hollis-Peel, & Lavenberg, 2013). In this program, youth at risk were exposed to a short spell of prison life, on the basis of the theory that this experience would be enough to keep them on a straight path. That is, prison life leads to being scared, which leads to improved behavior once in the real world. Qualitative data, however, showed no relationship between the program and improved behavior. A different mechanism was operating: rather than being scared, some youth enjoyed prison life, saw prisoners as role models, and prisoners they met provided them with criminal contacts outside (adapted from Johnson & Schoonenboom, 2016, p. 598-599).

that causal power is merely implemented through individuals. The emergent properties create dispositions of the whole, and we can interrelate and discuss these macro holistic variables in our theories without listing all of the individual parts and details. This is similar to the idea that water exists as whole, beyond its individual components of hydrogen and oxygen. You might say macro-variables are constituted by macro-actors and result in macro-consequences. Given the both-and logic of mixed methods (Johnson, 2016), it might be wise to respect both methodological individualism and holism because both viewpoints can help the explanatory process.

To help the reader further understand mechanistic causation, we adopt here the Illari and Williamson (2012) definition of mechanism that aims to capture the essential elements of the leading accounts (Bechtel & Abrahamsen, 2005; Glennan, 2002; Machamer, Darden, & Carver, 2000). Illari and Williamson (2012, p. 120) say: ''A mechanism for a phenomenon consists of entities and activities organized in such a way that they are responsible for the phenomenon.'' This definition is sufficiently general to be applied to different contexts, from biology and neuroscience to social science, which we take to be an important virtue.

Another Type of Causation: Agency Causation

The seventh theory of causation addresses the issue of the causal influence of people on their actions. This is particularly important in social science contexts, and even more when qualitative methods are used for causal analysis. Agency causation has a lengthy pedigree (Collingwood 1938; Gasking, 1955; von Wright, 1975). Illari & Russo (2014) discuss agency causation in the context of the contemporary philosophical debate, and Bandura (1991) discusses it in the context of self-efficacy and in his philosophy of social science. In general, agency theories attempt to link causation to the *actions* of agents. One way in which this can be done is found in Bandura's social cognitive theory, where people evaluate their alternatives and the likely outcomes; next, they make causal decisions; finally, they act, producing causal outcomes. Alternatively stated, consideration and selection of an action by an agent is said to cause agency-controlled behavior. It is often important to understand (and measure) individual and collective thinking and its consequences. The case-oriented analysis of Example 1 shows agency causation. It explains how, acting on events that occurred to her, a particular person, named Nynke van der Molen, decided to enroll in veterinary studies.

The causal theories just discussed are summarized in Table 1. We conclude this section with an additional theoretical proposition: *The more philosophical theories of causation satisfied, the stronger the evidence for causation*. We are using a nuanced cumulative logic because it is essential that thoughtful and appropriate combinations of causal theories be used in practice

Table 1. Core Ideas of the Traditional Causal Theories.

Account	Core idea
Probabilistic causation	The occurrence of C alters the chances of occurrence of E.
Counterfactual causation	C causes E means: If C had occurred, E would have occurred, and if C had not occurred E would not have occurred either.
Regularity or regularism	C causes E means: Instantiations of E regularly follow instantiations of C.
Necessary and sufficient causation	Necessary and sufficient causation (INUS): Causes are insufficient, but a nonredundant part of an unnecessary but sufficient condition.
Manipulation causation and invariance	C causes E if manipulating C makes E occur and the relation between C and E is stable enough
Mechanistic causation	C causes E if there is a mechanism linking C to E.
Agency causation	Consideration and decision (C) to act by an agent causes agency-controlled behavior (E).

(e.g., counterfactual and mechanistic causation complement each other quite well in many instances). Although our theory is prescriptive about taking a pluralistic stance, it remains rather liberal about the choice of philosophical theories and how they have to be combined. Specific causal mosaics for empirical studies are thoughtfully set up and judged on pragmatic grounds rather than a priori principles.

Causation in Quantitative Empirical Research

Quantitative research studies the relations among variables with the goal of producing a set of generic or general relationships that stand by themselves metaphysically and allow, epistemologically speaking, description, prediction, and explanation. Typically, quantitative research also follows the assumptions of probabilistic causation and soft determinism, which assumes there is predictability in the human world but also some chance and flexibility. Most quantitative researchers probably recognize that some causes are not directly manipulable (for physical and ethical reasons) but still must be studied causally. Some quantitative researchers also rely on the concept of multiple causation of effects, although this viewpoint is perhaps even more common in qualitative research. For example, thinking qualitatively, virtually any *event* in history (and in everyday life) has multiple identifiable factors leading to its occurrence (e.g., the ''causes'' of World War I).

Quantitative researchers in the social sciences often attempt to draw causal conclusions from experimental and nonexperimental quantitative data (Johnson & Christensen, 2017). For example, significant parts of psychology and education emphasize experimental data as required for claims of cause and effect. Likewise, sociology, economics, epidemiology, and political science have frequently relied on nonexperimental quantitative data for cause-and-effect conclusions. Nonexperimental quantitative data can provide *some* evidence for causation when the researcher moves beyond simple bivariate correlations, tests prior hypotheses, uses one or more methods of control (e.g., matching, statistical control, holding variables constant), establishes evidence of time order (via theory or longitudinal data), identifies and tests for rival hypotheses and alternative theoretical models, and so on (e.g., Glasziou, Chalmers, Rawlins, & McCulloch, 2007; Johnson, 2001; Rychetnik, Hawe, Walters, Barratt, & Frommer, 2004). All of these researchers across disciplines, however, require substantial and rigorous evidence of causation before such a claim can be made. The causal claim approximately boils down to, at a minimum, the meeting of the following three criteria:

1. C and E must be related (in the sense of statistically correlated)
2. C must occur before E.
3. The relation between C and E must not be subject to an alternative explanation; that is, one must rule out all plausible alternative explanations.

The three conditions are named, respectively, the relationship condition, the temporal-order condition, and the lack of alternative explanations condition.

Methodologically speaking, some quantitative research is based on experiments (e.g., RCTs and quasi-experiments) and some is based on nonexperimental quantitative approaches (e.g., nonexperimental longitudinal and structural equation modeling) to probe causation. The variable-oriented approach in Example 1 is nonexperimental, as the researcher did not attempt to influence the decision to attend a university through specific interventions or experiments. Conversely, Example 2 is an experiment, in which textbooks were provided as part of the inquiry and its effects were observed.

The more approaches used (with the RCT as the current regulative gold standard) and the greater the replication, the stronger the evidence of causation obtained, according to mainstream quantitative research. In addition to general, variable-oriented designs (e.g., RCTs, quasi-experiments, nonexperimental quantitative research designs), idiographic designs (e.g., A-B-A, A-B-A-B, multiple baseline, changing criterion designs) are used in quantitative research. The former top-down designs produce nomothetic knowledge that is difficult to apply to individuals, and the latter bottom-up designs produce idiographic knowledge that can be generalized with some risk. *According to the pluralistic theory of causation for MMR, it is essential to understand that both nomothetic and idiographic methodological approaches can be used to produce evidence of causation, the approaches produce complementary wholes, and produce superior and more defensible scientific knowledge* (Johnson & Stefurak, 2013; Robinson, 2011).

In short, current quantitative research uses a *sophisticated* approach to making claims about cause and effect among two or more variables. It does not rely on a simplistic regularity theory. Although quantitative researchers believe they are getting at causal truth, they also realize that empirical truths are often context and model dependent, and warrant is directly based on the *degree* of empirical evidence for the causal claims, rather than making strong claims of definite or ultimate *proof* of cause and effect. In empirical research, proof is something envisioned in the long term, in the distant future. Until then, we must rely on degrees of evidence.

Causation in Qualitative Empirical Research

Generally speaking, causation is not high on the list of concerns of qualitative researchers. The five most common approaches in qualitative research are phenomenology, ethnography, case study, narrative inquiry, and grounded theory (Johnson & Christensen, 2017). Only the last seems focused on causation (although they use other language such as ‘‘actions/interactions and consequences’’; Corbin & Strauss, 2014). Case study researcher Robert Stake (2010) puts the qualitative position thus:

> The qualitative researcher uses some of the words of causal connection [. . .] but (if done properly) makes reference to the limited, local, and particular place and time of the activity. Even then [. . .] the purpose has not been to attain generalization but to add situational examples to the readers' experience. (p. 23)

Interestingly, the classic version of grounded theory that is most connected to causation (and focused on producing substantive and middle-range theories) has been criticized as too abstract,

too general, and too quantitative by some qualitative researchers. In response, constructivist, local grounded theory approaches have been developed (e.g., Charmaz, 2014). In short, mainstream qualitative research is focused on the local and even its most ''quantitative'' variant often avoids ''causal talk'' (Denzin & Lincoln, 2018).

Much of qualitative research has allowed quantitative research to control and win the battle over the use of causal language. In our view, however, qualitative research should not relent because causation *is* relevant for much qualitative research; it is just a different sort of causation, a singular and local causation. Causation certainly occurs at local levels. Because of the quantitative research control of the term *cause* and the potential backlash, however, we suspect that many qualitative researchers have resorted to using other words to disguise their causal talk.

One well-known and fruitful approach to causation in qualitative research has been developed by Joseph Maxwell (2004a, 2004b, 2012a, 2012b; see also Bazeley, 2013). Maxwell's critical realist approach spends quite a bit of time explicating the contention that local, mechanistic causation is appropriate for qualitative research. Qualitative research operates at the local level and observes *complex* causal processes that are often missed in quantitative research because of its relatively blunt instruments and analytical procedures for uncovering causal complexity.

Maxwell's approach focuses on *events* and processes that connect them. Central are human agents, their experiences, thoughts, meanings, and actions. Not surprisingly, both mechanistic causation and agency causation play an important role. Here, mechanistic causation should be read as ''C is an account of E.'' In an account of how E came about, some events may play a more prominent role than others. Causation takes place in the context as a whole, and the context cannot be separated from the cause. Therefore, rather than stating that there is a mechanism relating C to E, the view here is that C, taken as the account as a whole, *is* the mechanism. Note also that explanations of human actions involved may refer to considerations and decisions of the agents, that is, to agency causation. Most likely, an explanation will also involve many elements that are beyond consideration of the human agents.

In qualitative observation, we are able to see specific local causation in action, and in qualitative interviews, we are able to learn about specific local causation in action, including agency causation. There is no need for variables or counterfactuals (Mohr, 1995) in this direct approach to causation. This ''local causation view'' is also inspired or in line with the viewpoint of methodological individualists or localists, who claim that social causation operates at the level of acting and interacting individuals (e.g., Little, 2011).

The case-oriented analysis in Example 1 exemplifies a qualitative approach to causation. The experiences and actions of Nynke are central, and events, such as the decision of Leonie to go to the university and the scrapbook that her mother shows to Nynke, played a crucial role. Leonie's decision and her mother's scrapbook and any other events present are viewed as account of how and why Nynke decided to go to university. In this situation, one can choose to use or not to use the word ''cause'' depending on one's perspective. One can argue that these are multiple local causes, although many qualitative researchers choose not to use causal language. Notice that we do not learn about counterfactuals in this example (What would have happened if Leonie had not gone to university?); the focus is on what does happen, and on acting and interacting individuals (Nynke, her mother, Leonie).

One promising approach to studying causation via qualitative research is *causal process tracing* (CPT; cf. Beach & Pedersen, 2013; Bennett & Checkel, 2014; Blatter & Haverland, 2012; Collier, 2011; Mahoney, 2012). CPT is based mostly on qualitative interview and observation data, especially case study data. Here are the assumptions of CPT according to Blatter and Haverland (2012): (a) Most social outcomes result from a combination of causal factors (multiple causation); (b) there are multiple paths to the same outcome (equifinality); (c) factors can operate differently in different contexts (context dependency); and (d) causality plays out in

time and space, which can be studied empirically. Observations over time (longitudinal qualitative data) are used to study how a causal process unfolds and it can provide comprehensive storylines. These show, among other things, temporal order of events. Observations also show pathways and causal chains leading from C to E, providing what is called ''smoking gun'' evidence of cause and effect (Collier, 2011). Interviews, called ''confessions,'' provide evidence of action potential (i.e., how individual agential thinking helped produce events of interest). All of this information is used to make causal inferences and show ''recipes'' for producing outcomes (Blatter & Haverland, 2012). CPT (a) provides understanding of complexity (mediation and moderation, equifinality, multiple causes); (b) can be used to generate and test theories; (c) can increase internal/causation validity (understanding a mechanism increases our confidence in a causal relationship); and (d) provides ''possibilistic generalizations'' (i.e., generalizations about what processes can occur in the world rather than statistical generalizations).

According to our pluralistic theory of causation, mixed methods researchers should examine and combine evidence of local causation with evidence of general causation (they are complementary), and CPT offers one important methodological approach for researchers to develop further and use in their research practice.

Continuing Our Journey Toward a Pluralistic Theory of Causation

We now return to the statement at the beginning of this article that there are various notions of causation, and see how we can apply this idea in MMR practice. Put differently, we contend MMR should endorse a pluralistic theory of causation because it fits its philosophy and methodology. The philosophical literature offers a number of accounts of pluralism, discussed in detail in Illari & Russo (2014, Chapters 23, 24). However, none of these captures the *several* dimensions we are interested in. According to our theory, multiple arrangements of different theories of causation can fit a mixed methods approach to causation because MMR seeks to thoughtfully and creatively combine ideas from both qualitative and quantitative research and philosophy. This means that our theory is pluralist also in the following respects: methodology, evidence, concepts, and ontology.

The first type of pluralism we advocate is *methodological*: Many methods and methodologies are different and useful in finding, demonstrating, and unpacking causation. From this, we derive evidential pluralism: Many *sources* of evidence are needed and used in our making of causal claims. From evidential pluralism, a form of *conceptual* pluralism follows: There are different kinds of causes at the conceptual level. This is seen in the multiple causal theories outlined earlier in this article; each provides a different concept of causality. It is then legitimate to conjecture that adopting these forms of pluralism would also lead to a most fundamental type of pluralism, namely *ontological* according to which there are different kinds of causes at the level of reality. In turn, this would mean that qualitative and quantitative research focus on causation at different levels of abstraction: Qualitative research addresses singular and complex causation that *exists* at the local level and quantitative research focuses on developing knowledge of general causation and knowledge that *exists* at a higher level of abstraction.

The point here is that qualitative research and quantitative research need different ontologies and different ontological foci and interests, *and both are important for MMR*; both have something important to add to scientific knowledge. However, one need not agree with all of the pluralisms to *use* our theory because the theoretical assumptions can be tailored slightly in practice. For example, one can collect and interpret data addressing both local and general causation without taking the stance that these are ontologically different. In short, a *pragmatic* version of our theory might be fine in practice. That is, one might use multiple perspectives of causation while keeping the focus on the purpose of the research and useful outcomes that might be

produced, rather than focusing on the philosophical/metaphysical assumptions of the multiple perspectives.

When applied to MMR practice, which is the main aim of this section, the idea of causal pluralism has the following important implication: *MMR researchers should rely on multiple theoretical and methodological criteria for establishing evidence causation*. One set of criteria or guidelines comes from the work of Bradford Hill and his colleagues (Doll, 1992; Hill, 1965; Susser, 1977) in epidemiology and medicine. Epidemiologists attempt to show causal relations among variables (popular in quantitative research), but they also attempt to show the causes of particular events (e.g., an outbreak of a disease in a particular area). The Bradford Hill guidelines favor evidence from causal repeatability, but one can also use the reasoning for particular cases/events. Also, evidence of some causal repeatability can be important even in qualitative research when these researchers attempt to move beyond singular or particular causation (e.g., using comparative case studies and meta-syntheses) to support policy making beyond a particular place/person/group/setting. Our general theoretical proposition is that *mixed methods researchers can and should consider multiple guidelines and viewpoints for causation and oftentimes, the more viewpoints are appropriately combined and cumulatively met, the greater the evidence of causation*. Here is one set of guidelines, the Bradford Hill guidelines, for establishing multiple kinds of evidence of causation to be thoughtfully combined:

1. *Strength of association*. The stronger the relationship between the independent variable and the dependent variable, the less likely it is that the relationship is due to an extraneous variable.
2. *Temporality.* It is logically necessary for a cause to precede an effect in time.
3. *Consistency.* Multiple observations, of an association, with different people under different circumstances and with different measurement instruments increase the credibility of a finding.
4. *Theoretical plausibility.* It is easier to accept an association as causal when there is a rational and theoretical basis for such a conclusion.
5. *Coherence.* A cause-and-effect interpretation for an association is clearest when it does not conflict with what is known about the variables under study and when there are no plausible competing theories or rival hypotheses. In other words, the association must be coherent with other knowledge.
6. *Specificity in the causes*. In the ideal situation, the effect has only one cause. In other words, showing that an outcome is best predicted by one primary factor adds credibility to a causal claim.
7. *Dose–response relationship*. There should be a direct relationship between the risk factor (i.e., the independent variable) and people's status on the disease variable (i.e., the dependent variable).
8. *Experimental evidence.* Any related research that is based on experiments will make a causal inference more plausible.
9. *Analogy.* Sometimes, a commonly accepted phenomenon in one area can be applied to another area.

For an example, here is an adaptation of Johnson & Christensen (2017) application of the Hill guidelines to the classic causation case of ''smoking and lung cancer.''

1. *Strength of association*. The lung cancer rate for smokers was quite a bit higher than for nonsmokers (e.g., one study estimated that smokers are about 35% more likely than nonsmokers to get lung cancer).
2. *Temporality.* Smoking in the vast majority of cases preceded the onset of lung cancer.
3. *Consistency.* Different methods (e.g., prospective and retrospective studies) produced the same result. The relationship also appeared for different kinds of people (e.g., males and females).
4. *Theoretical plausibility.* The biological theory that smoking causes tissue damage that over time results in cancer in the cells was a highly plausible explanation.
5. *Coherence.* The conclusion (smoking causes lung cancer) ''made sense'' given the current knowledge about the biology and history of the disease.
6. *Specificity in the causes*. Lung cancer is best predicted from the incidence of smoking.

7. *Dose–response relationship*. Data showed a positive, linear relationship between the amount smoked and the incidence of lung cancer.
8. *Experimental evidence.* Tar painted on laboratory rabbits' ears was shown to produce cancer in the ear tissue over time. Hence, it was clear that carcinogens were present in tobacco tar.
9. *Analogy.* Induced smoking with laboratory rats showed a causal relationship. It, therefore, was not a great jump for scientists to apply this to humans.

The Bradford Hill guidelines produce complementary evidence for causation. *Generally, the more Bradford Hill ''bases are covered'' the better one's evidence for a causal claim*—this is a fundamental proposition in our pluralistic theory of causation. But, of course, the aggregation of the viewpoints is not merely a matter of quantity. It is worth noting that, if one follows Hill's guidelines, strictly speaking, one can come up with different causal claims. Consider an example from social science about the relation between ''education'' and ''future earnings.'' Following strength of association, one might conclude that ''Education is positively associated with future earnings''; following theoretical plausibility, one might conclude that ''Education seems to cause higher future earnings via socioeconomic mechanisms''; following quasi-experimental evidence, one might conclude that ''The study indicated that the group with higher level of education showed consistently higher earnings than the matched group with a lower level of education''; or, following a qualitative approach, one might understand how ''people with much education transform that education into membership in occupations of high social standing'' (Abbott, 2001, p. 67).

The point is that these are all *different* causal claims that are true in virtue of different objects, relations, or states of affairs being (or not being) present. This, in philosophical jargon, is the question about *truthmakers*, namely what makes a claim *true*. Some of Hill's guidelines require variables and correlations between variables to hold; others require identifying mechanisms of action at different levels; yet others may require counterfactual relations to cash out causation. Analogously to what happened to causal theories, there have been attempts to identify *The-One* truthmaker that would fit all sorts of causal claims, and *The-One* type of causal relata in the world. But it is clear from our pluralistic theory of causation that this is not possible, and we must instead admit of a *plurality* of truthmakers and of causal relata. Legitimate relata candidates, depending on the context, are variables, probabilistic relations, capacities, objects, actions, and so on. The key question is about how *together* they can contribute to understanding a given phenomenon in a particular research study or collection of related studies.

The philosophy of causality discusses causal *truthmakers*, that is, types of outcomes that provide evidence of causation (Illari & Russo, 2014). Our pluralistic theory of causation treats truthmakers analogously to the treatment of causation using the Bradford Hill criteria, propositionally stated as *the more philosophical truthmakers are thoughtfully, logically, and systematically combined (vis-à-vis the particular research question and context) and are satisfied, the stronger the evidence of causation*. Keep in mind that not all combinations are equal. Some are more important than others; for example, one always needs to rule out alternative explanations when making a claim. Researchers must make the case for their particular combinations of criteria and evidence in relation to their research questions, their research purposes, and their research contexts. The forming and use of evidence of causation will be based on the causal mosaic constructed for each research study for guidance and justification of the particular causal claims. Here is a (nonexhaustive) list of various sources of causal evidence or truthmakers for your use:

(a) Evidence of capacities (i.e., Does the causal variable have the capacity or ''causal oomph'' to produce the effect?)
(b) Evidence of probabilities (i.e., Do we have probabilistic evidence of causation?)
(c) Evidence of regularity (i.e., Is there a regular relationship between C and E?)
(d) Evidence of possible worlds in counterfactual thinking (i.e., Does the causal relationship hold in this world but not hold in an alternative similar world without the treatment?)
(e) Evidence of natural laws (i.e., Is there a lawful relation from which the case of causation can be deduced?)
(f) Evidence of information causation (i.e., Is there a link or some sort of ''causal line'' or movement/transmission of bits of ''information'' from C to E?)
(g) Evidence of difference making (i.e., Did the treatment condition ''make a difference'' compared to a control group?)
(h) Causal evidence at multiple levels (e.g., social, psychological, biological, and physical levels of reality)
(i) Evidence of manipulation impact (i.e., Does manipulating the world in some way end up with the presumed effect?)
(j) Evidence of reasonableness (i.e., Does a causal relation between C and E make sense?)
(k) Evidence of temporal order of cause and effect (i.e., When C occurs, does E follow?)
(l) Evidence that all plausible rival explanations have been ruled out
(m) Philosophical/scientific evidence (Do the following kinds of evidence converge into a meaningful whole: ontological, epistemological, axiological, and methodological?).

In summary, when using the pluralistic theory of causation developed here, researchers are directed to use multiple kinds of causation and multiple sources of evidence when establishing degrees of causal evidence. This means, for example, satisfying more Bradford Hill criteria will, ceteris paribus, lead to a stronger evidence base for causal claims. Likewise, generally speaking, the more truthmakers realized, the stronger the evidence for causation. Furthermore, both singular/local/token and general/generic/type causation can provide complementary evidence that together provide a better understanding of causation. An interesting corollary of our pluralistic theory of causation is that *claims based on correlational and experimental evidence are strengthened when one also has evidence of mechanism*. In the philosophy of science literature, this idea is formally known as the Russo–Williamson thesis (Russo & Williamson, 2007; Clarke et al., 2014). This thesis states that evidence of difference making and mechanism are typically required for high-quality causal inferences. Finally, regarding the causal theories outlined earlier, *according to our pluralistic theory of causation, the more theories of causation that are logically satisfied, the stronger one's evidence of causation*—that's because the causal claim will have survived *multiple tests* that are emphasized by multiple communities addressing causation. Put differently, the causal claim will have survived several and different subsequent ''mangles of practice'' (Pickering, 1995).

Summary: The Formal Structure of the Pluralistic Theory of Causation for MMR

Key Theoretical Idea

According to the mosaic view of our causal theory, there is not *one* theory of causation that satisfies all scientific domains or all specific studies. Accordingly, one should construct an appropriate causal mosaic for *each* research study, in order to know what is causally relevant and to articulate one's assumptions and approaches for warranting one's causal claim(s).

Theoretical Assumptions

Causal pluralism underlies the theory, specifically, methodological, evidential, conceptual, ontological, and multiple-level pluralism. At the same time, one can use the theory pragmatically to determine its value in solving causal problems in the social–behavioral sciences.

Theoretical Propositions

- **Proposition 1:** Both quantitative and qualitative philosophical and methodological approaches to causation are important for a fully defensible social science.
- **Proposition 2:** Having general/nomothetic and local/idiographic causal understanding is important for truly understanding research phenomena.
- **Proposition 3:** It is helpful to be open to the ideas in both determinism and indeterminism as one studies the world.
- **Proposition 4:** It is helpful to be open to understanding both universals (holism) and particulars (individualism).
- **Proposition 5:** It is important to understand both molar and molecular causation.
- **Proposition 6:** It is important to understand both causal description and causal explanation.
- **Proposition 7:** It is important to have evidence of both difference making and causal production.
- **Proposition 8:** The more philosophical theories of causation are logically satisfied, the stronger the evidence for causation.
- **Proposition 9:** The more Bradford Hill causal bases are covered, the better one's evidence for a causal claim.
- **Proposition 10:** The more philosophical truthmakers are thoughtfully, logically, and systematically combined (vis-à-vis the particular research question and context) and are satisfied, the stronger the evidence of causation.
- **Proposition 11:** Researchers can and should consider multiple guidelines and viewpoints for causation, and, oftentimes, the more viewpoints appropriately combined and cumulatively met, the greater the evidence of causation.

Based on the 11 propositions, hypotheses can be operationalized and empirically tested to determine the accuracy and value of the pluralistic theory of causation provided in this article.

Conclusion

This concludes our journey, as we now hand the 11 propositions over to the research community. We hope practitioners of MMR will not be deterred by the lack of attention to causation in current mixed methods books. Instead, we hope researchers will move forward with renewed confidence and effort by carefully writing causal research questions, designing strong studies to answer those questions, using causal language throughout the research study, and producing increased understanding of causation as it operates in the world in which we live. We hope this article will provide an important reference for researchers *and* will inform the social-and-related-sciences literatures about the *usefulness* of our pluralistic theory of causation, and help us all better to address and solve the scientific problems we face.

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Notes

1. Admittedly, this alleged simple dichotomy obscures several layers of complexity, embedded in the practice of different scientific disciplines and in the ways the philosophical literature on causality approached the issue (for a detailed discussion, see Illari & Russo, 2014). For instance, it is not obvious to hold the view that singular causation is primary and generic causation is a mere aggregation. For one thing, this characterization does not suit a mixed methods approach. For another, we are claiming there is an important ontological distinction to be made that both are real and are distinguishable.
2. The respective terms are relabeled metadiagrams, metamatrices, and metamodels in the newest edition (Miles, Huberman, & Saldana, 2014).

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