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A Methodological Outlook on Causal Identification and Empirical Methods for the Analysis of Social Mechanisms*

Abstract: The debate on empirical tests of social mechanisms suffers from a fragmented view on the relative benefit of the empirical method a researcher considers to be superior, compared to the flaws of all other methods. In this outlook, I argue that disciplinary barriers might be surmounted by a common methodological perspective on the analysis of social mechanisms. First, experimental, quantitative, qualitative, and simulation methods (agent-based modeling) are all required, but also capable to deal with the issue of *causal identification*, respectively. Second, having established causal identification (among which I subsume strategies to deal with *causal heterogeneity*), each method disposes of genuine techniques to deal with the most crucial property of mechanism-based explanations: *input-mechanism-output (IMO) relations.*

1. Introduction

Since its beginning in Hedström and Swedberg's (1996) seminal paper, the research program of mechanism-based explanations in the social sciences and the one of analytical sociology yielded several influential editor volumes (Demeulenaere 2011; Hedström/Bearman 2011; Hedström/Swedberg 1998; Manzo 2013) and various other publications beyond that (for an overview cf. Hedström/Ylikoski 2010). While both the plurality and the diversity of the publications resulting from the debate might be regarded as an indicator of its academic success, more critical voices rather conceding a sheer 'mechanism talk' could also be noted (Kalter/Kroneberg 2014; Norkus 2005). In particular, critics blame the mechanism approach for still not having surmounted a notable gap between mere theoretical treatises on the one hand and rather loose and *ad hoc* empirical applications on the other hand (ibid.). The argument of this outlook of the special issue at hand is that the research program is in need of a more obliging *methodological* framework which builds on crucial propositions of mechanism-based explanations and translates them into empirically operationalizable techniques of a variety of research methods.

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By assuming that social mechanisms are productive of events, social scientists commit themselves to the view that social mechanisms exist and can be investigated. Leaving aside his ontological theory, Bhaskar (1975, 69) developed the idea that scientific methods can identify mechanisms by means of a *closure*, i.e. excluding those influences conceded to be irrelevant but supposed to be possibly interfering.[1]

When the idea of mechanism-based explanations in the social sciences emerged in the 1990s, a major criticism was directed against too simplistic quantitative methods mixing up significant correlations or regression coefficients with causality (Freedman 1987; also see Tranow et al. in this volume). As a consequence, the research program of analytical sociology which devotes itself to social mechanisms and how they can be tested primarily focuses on agent-based modeling (ABM). The programmer of ABM can build up a closed system by herself and control the initial distribution of agents, their preferences, beliefs and action rules, etc.

While by no means I wish to disavow the potential of ABM to generate and advance sociological theories and to shed light on transformational mechanisms in particular, I would yet argue that it is neither the only empirical method suitable to test for social mechanisms, nor can it elude the requirements of causal identification that have already been applied especially to quantitative methods of analysis.

In order to isolate the causal effect of interest, I follow propositions by Manski (1993) and Keele (2015) to differentiate between causal identification and empirical methods. However, while Keele (2015) narrows the empirical methods allowing for the analysis of causal effects to statistical methods, I hold the view that qualitative methods and agent-based modeling are also suitable to analyze causal effects, yet they have to solve the issue of causal identification as well.

In the following, I will first outline the potential of each method to identify causal effects. A special focus will be on available strategies to deal with *causal heterogeneity*. Recall that one crucial demand of mechanism-based explanation was the plea for *middle-range theories* (Boudon 1991; Hedström/Udehn 2009; Merton 1957). Being opposed to the deductive-nomological framework, mechanism-based explanations rely on regularities instead of general laws (Hedström/Swedberg 1996; Little 1998; Ylikoski 2013). As a consequence of giving up the idea of general laws, middle-range theories are possible which aim to provide realistic (or: *understanding*) instead of just parsimonious explanations for social phenomena. More precisely, middle-range theories no longer suppress causal heterogeneity just in order to preserve a parsimonious theoretical model. Instead, it is possible (and in many cases also more realistic) that the impact of

[1] The natural sciences are usually assumed to be better prepared for arriving at a suchlike closure by means of the randomized experiment, while the human and social sciences in many instances lack this option (Collier 1989, 130f.). This coincides with the view that within fundamental physics, the assumption of universal laws may still be justified (Woodward 2003, 19, 240), whereas in the social sciences, we can only assume something like *lawish generalizations* (Reutlinger 2013, 18) or *law-like regularities* (Little 1998, 240–243). Yet, the position to be defended in the contribution at hand is that by an appropriate application of empirical methods, social sciences can at least approximate a suchlike closure (cf. Kemp/Holmwood 2003).

a treatment on the outcome may vary for different social groups. Hence, my argument is that unless an interaction between treatment/predictor and mediator is concerned, causal heterogeneity has to established *before* the mechanism(s) accounting for it can be identified.

Second, having discussed causal identification, I will elaborate on the disposable techniques which are suited to address the most crucial property of mechanism-based explanations: *input-mechanism-output (IMO) relations*. While the literature is not consistent on whether the term 'mechanism' should be used for the mid-part of the chain only or for the entire process (Mayntz 2004, 244), it is a commonplace in the philosophy of social sciences that a prerequisite of a causal relation—and this is what mechanism-based explanations strive to establish—is a diachronic relation between cause and effect (Ylikoski 2012, 33). Hence, any method that wants to answer the question why and how a causal effect comes about has to find a technique that is suitable to identify the causal succession between I, M, and O.

Before doing so, let me clarify a more conceptual issue: the notion of a 'causal effect' utilized in this paper is not an ontological, but rather a methodological one. From an ontological view, it could legitimately be objected that any attempt to separate the identification of mechanisms from causality violates the most common definition of causality in mechanism-based research stating that a causal effect is always brought about by some kind of mechanism (Bhaskar 1975; Mingers 2004). However, given that we can never observe causation *per se*, but only the empirical data we suppose to be instantiated by a cause, we first have to rely on our empirical methods to make as sure as possible that we really observe a causal effect in an empirical sense, and not spurious correlations (e.g., due to a common cause of both the suspected cause and its suspected outcome). Having ruled out potential confounders, the identification of the social mechanism(s) by which the causal effect is hypothesized to be passed on can be pursued.

2. Causal Identification

Causal identification is something that has to be established by theoretical assumptions *prior* to statistical analysis. Keele (2015) discusses causal identification in the tradition of the potential outcomes framework (Holland 1986; Rubin 1974) which estimates the effect of a treatment T on an outcome Y by adopting a counterfactual view on causality. Theoretically of interest would be the difference in outcome Y for individual i in the treatment condition ($T = 1$) compared to the same individual being untreated ($T = 0$). However, as each individual is only observed in one condition, the individual-level causal effect is approximated by the average treatment effect (ATE) defined as "the average difference in the pair of potential outcomes averaged over the entire population of interest" (Keele 2015, 316). The unobservability of the individual-level treatment effect is also referred to as the *fundamental problem of causal inference* (Holland 1986, 947). In order to use the ATE as an estimate of the individual-level causal effect, several *identification assumptions* are necessary:

- the treatment status must be independent of the potential outcomes,
- there are no hidden forms of treatment (consistency assumption),
- a subject's potential outcome is not affected by other subjects' exposure to the treatment (no spillover effects).

Following this theoretical reasoning about identification *assumptions*, several identification *strategies* are possible. In the following, I will review the potential of experimental, quantitative, qualitative, and simulation methods (also known as agent-based modeling) concerning the identification of causal effects (for a more extensive overview, neglecting qualitative methods and ABM though, see Keele 2015, 318–24).

2.1 Experiments and Natural Experiments

The most important method for the natural (and some social) sciences is the *experiment* randomizing observations along treatment groups and thereby isolating the treatment from the now randomly-distributed side conditions. Importantly, application or manipulation of the treatment can be controlled by the experimental researcher (Dunning 2007, 282). The advantage of the experiment is its high internal validity, but it comes at the price of comparably low external validity (cf. Bohnet 2009): most experiments rely on a selective group of individuals (in most cases undergraduate psychology students), and the artificial laboratory situation which in most cases circumvents the possibility of communication between subjects hardly maps our notion of everyday social interaction. Hence, a causal effect that has been identified in an experimental setup must not necessarily hold in real-world settings.

A more realistic scenario is the *natural experiment* which tries to identify treatment assignment within a real-world setting. Thus, having established internal validity in a randomized experiment, a natural experiment may be a plausible second step to test for the external validity of a causal effect. Important to mention, natural experiments are *observational* and not true experimental data as treatment variables cannot necessarily be manipulated by the analyst (Dunning 2007, 283). Neither are they 'quasi experiments' for the very reason that in contrast to the latter, the hallmark of natural experiments is a plausible "as if" (Dunning 2007, 289) or exogeneity assumption (Sekhon/Titiunik 2012, 35f.) of random assignment which can be defined as follows: 1) The treatment must be independent of both observables and unobservables that might affect the outcome; 2) people do not change treatment condition as a consequence of the treatment; and 3) the treatment and control group should be balanced (Dunning 2007, 284). Typical examples of establishing as-if randomization are price lotteries (Doherty/Gerber/Green 2006), jurisdictional borders (Card/Krueger 1994), or regression discontinuity designs (Angrist/Lavy 1999). While matching techniques can be used to ensure that treatment and control group are balanced on observables (Conniffe/Gash/O Connell 2000), the risk that between-group differences with respect to unobserved characteristics dis-

tort or account for the postulated causal effect of the treatment on the outcome constitutes "the Achilles' heel of natural experiments" (Dunning 2007, 289).[2]

2.2 Quantitative Data

2.2.1 Causal Identification

When it comes to approximation of causal effects with *observational quantitative data*, there is no actual (quasi)experimental assignment of individuals into a particular treatment status. Yet, particular identification strategies for observational data share the aim to minimize differences between treatment and control group in order to ensure that a causal effect between two balanced sub-samples can be estimated (Brückner 2011, 697ff.; Keele 2015, 321f.). An assumption full of preconditions which yet is often implicitly made in practice without any further justification is the case of *selection on observables*. Here, the researcher assumes that once she controls for a range of covariates measured in the data, there are no differences between treatment and control group. Typical *statistical* strategies to be performed under this assumption are regression analysis, matching, or weighting approaches. Again, is important to note that selection on observables is a theoretical identification assumption that cannot be tested with empirical data.

Another identification assumption supposes selection on *unobservables*. Here, (also) unmeasured characteristics would be responsible for individuals to select into a particular treatment. In that case, conventional regression and even matching techniques are not suited to ensure balanced grouping between treated and the non-treated individuals. Instead, instrumental variable (IV) strategies, selection models (SM), or Differences-In-Differences (DID) estimators have to be used (cf. Ceruli 2015, chapter 3). The IV approach assumes that selection on observables can be controlled for by including an instrument that affects selection into the treatment, but not the outcome of interest. SMs control for the selection process by simultaneously estimating one equation for selection into the treatment, and one treatment equation of primary interest which is thereby controlled for the selection process. Either an instrument in sense of the IV approach, or other identification assumptions such as joint normality of the error terms of the two equations are required. Finally, DID estimators rely on panel data and estimate the difference between two time points for both the treated and the non-treated. Similar to fixed-effects regression, it is assumed that unobserved heterogeneity will be canceled out once within-group changes over time are compared between groups.

2.2.2 Causal Heterogeneity

A crucial question in both experimental and quantitative-observational research is about whether a causal effect is homogenous or heterogeneous among the

[2] The issue of causal heterogeneity will be discussed in *section 2.2*.

treated. Recall that in quantitative research, we usually fit additive models of the following functional form:

$$y_i = \beta_0 + \beta_1 x_1 + \beta_2 x_2 + \ldots + \beta_n x_n + \epsilon \tag{1}$$

As it has been recognized by both qualitative (cf. Braumoeller 2003, 210) and ABM researchers (Bruch/Atwell 2015, 188), additivity is a strong assumption, and in many instances, a simplification of the actual mechanism underlying the causal effect. Yet, this issue has also been recognized by quantitative researchers who no longer ignore the potential heterogeneity of a (treatment) effect on a particular outcome but acknowledge that the former might vary over the range of another predictor. As aptly verbalized by Xie (2013, 6263), "heterogeneity is the rule rather than the exception in social science [...] I do not believe that we can ever assume within-group homogeneity in social sciences".

One technique that is easily implemented in conventional quantitative analysis even without consideration of the potential-outcome framework are *moderator models* which can be represented statistically as follows:

$$y_i = \beta_0 + \beta_1 x_1 + \beta_2 x_2 + \beta_3 x_1 x_2 + \ldots + \beta_n x_n + \epsilon \tag{2}$$

Here, the term $\beta_3 x_1 x_2$ represents the interaction term capturing this conditional variance. Importantly, in moderator models, not only partial regression weights for each of the predictors x_1 and x_2 can be deduced, but also conditional significance levels (Brambor/Clark/Golder 2006; Friedrich 1982). Hence, a treatment effect that was found to be statistically significant for one group or over a particular range of a continuous moderator variable must not necessarily be statistically significant for another group or over another range.

In the potential outcome framework, Imai et al. (2011) distinguish two ways an ACME may be moderated. First, the treatment effect may be different for individuals exhibiting different pre-treatment characteristics (i.e., psychological dispositions). This may be accounted for by including interaction terms between the treatment and *observed pre-treatment covariates*. Second, it may be possible that the treatment effect is moderated by the *mediator*.[3] In that case, the interaction term between treatment and mediator would be included in the two equations predicting the outcome according to the ACME framework outlined above.

While the idea of mediation analysis as a technique to test for social mechanisms will be elaborated on in *section 3*, the crucial argument for now reads as follows: it is already an issue of causal identification to analyze treatment effect heterogeneity *before* the social mechanism(s) accounting for it can be specified. To provide an example: imagine a student sample in which we find a significant effect of parental education on students' educational transitions which remains stable after controlling for a vector of relevant covariates. Yet, moderation analysis reveals that the social background effect is only significant for boys, but not for girls. Consequently, as a second step, we would search for the particular social mechanism by which this heterogeneous effect is passed on.

[3] On moderated moderation also see Preacher et al. (2007).

2.3 Qualitative Data

2.3.1 Causal Identification

In (comparative) *qualitative research*, "it is difficult to capture weak causalities, assess the relative strength of causal factors, assess statistical significance, control for other variables, capture central tendencies and variability, make assertions on the general distribution of types and assess representativity in general" (Stolz, this volume, 265). Yet, Mill's method of agreement and method of difference can be used to obtain a closure by eliminating either potential necessary or sufficient causes of a nominal outcome in small-N analysis. With the method of agreement, the outcome occurs in all cases. Thus, any potential cause that is not observed in all cases cannot be necessary. In contrast, with the method of difference, the outcome occurs in some cases but not in others. Hence, any potential cause that is observed in all cases cannot be sufficient since it did not always bring about the outcome. Both methods suit the counterfactual framework of causal identification as they compare the actual distribution of events with a fictitious distribution that would have to be expected if a treatment was necessary or sufficient, respectively (for a similar argument see Sekhon 2004).

Yet, Mill's methods are not immune against specification error, for "it is always possible that explanatory variables not considered in the analysis might avoid elimination if they were included. In addition, variables eliminated in the Boolean reduction procedure might *not* have been eliminated if other variables had been introduced in the analysis." (Mahoney 2000, 395) In general, qualitative research requires methods of identification very similar to those of quantitative methods. For instance, if a qualitative researcher wants to make more inferences than observations are available, or two explanatory variables are perfectly correlated with each other, the research design is as indeterminate as in underidentified or multicollinear quantitative analysis (King/Keohane/Verba 1994, 119–24). On the other hand, once a qualitative model is identified, an undeniable strength of qualitative research is to get close to the generative processes, i.e. the underlying mechanisms(s) of a causal effect (details to follow).

2.3.2 Causal Heterogeneity

A common method in qualitative research to account for causal heterogeneity is the *qualitative comparative analysis* (QCA). QCA builds on Mill's methods of necessary and sufficient conditions, but it is explicitly acknowledged that one and the same outcome may be instantiated by several distinct causes. Therefore, it turns the view that causation should be analyzed backwards from multiple instances of an outcome to the alternative analysis of combinations of causal conditions (Ragin 1999, 1228).

The first step in QCA is to construct a truth table containing all possible combinations of causal conditions which exponentially increases with the number of conditions. To give an example, if each condition has 2 categories (e.g., 'present' vs. 'absent'), and we have 4 conditions, we must analyze $2^4 = 16$ causal conditions. The next step amounts to identifying and interpreting the causal conditions that are sufficient for the outcome. *Table 1* shows a hypothetical

truth table with four causal conditions (**A**, **B**, **C**, **D**) and one outcome (**Y**). We can see that the presence of both **A** and **B** and the absence of **C** is jointly sufficient to bring about **Y** (in formal terms: **A*B*c**).

A	**B**	**C**	**D**	**Y**
no	no	no	no	No
no	no	no	yes	?
no	no	yes	no	?
no	yes	no	no	No
no	yes	no	yes	No
no	yes	yes	no	?
no	yes	yes	yes	No
yes	no	no	no	?
yes	no	no	yes	?
yes	no	yes	no	?
yes	no	yes	yes	?
yes	yes	no	no	yes
yes	yes	no	yes	yes
yes	yes	yes	no	?
yes	yes	yes	yes	?

Table 1: Truth table example. Source: Ragin/Sonnett 2005, 191.

Yet, we could also think of other scenarios where, say, **A*B** is sufficient to bring about the outcome, but so is **C*d**, which we would write as **A*B** + **C*d**. This is what Mackie (1965) called an INUS condition which is itself "an insufficient but necessary part of a condition which is itself unnecessary but sufficient for the result" (245). Thus, both **A*B** and **C*d** may each be *sufficient* to realize **Y**, but none combination is a *necessary* combination for **Y** to be brought about.

Hence, QCA is a technique that allows to incorporate causal complexities that go beyond conventional additive models which are still often used in mainstream quantitative analysis. It is also suited to capture higher-order complexities that go beyond two- or three-way interactions which are usually the maximum considered to be implementable in multivariate regression models (Dawson/Richter 2006).[4]

[4]In the QCA literature, terms like 'causal complexity' or 'conjunctive causation' are more common than the term 'causal heterogeneity'. Yet, the example from *section 2.2* may serve as an illustration that all these terms refer to the same idea: If the result of a small-N QCA shows that the conditions 'high parental education' and 'gender: male' must both be satisfied to explain the outcome 'low achievement', this is equivalent to a large-N moderation analysis which finds that the effect of parental education on achievement is significant for male students only. In neither case would gender alone be a sufficient condition to explain the outcome.

2.4 Agent-based Modeling

2.4.1 Causal Identification

Agent-based modeling (ABM) is a technique which is usually pled for by analytical sociologists when it comes to the analysis of social mechanisms (Hedström 2005, chapter 6; Manzo 2014). ABM made its way into mechanism-based research as a reaction to bad-practice quantitative research (e.g., the 'causal modeling' or path analysis tradition; see Freedman 1987) always standing at the risk of mixing up correlations (or regression coefficients) with causation (Hedström 2005, chapter 5). Another advantage of ABM is of course that the transformational mechanisms connecting the micro to the macro level again can be investigated much better than in quantitative research which in many cases has to assume the mutual independence of observations (i.e., it cannot be modeled that observations may influence each other).[5] While Berk (2008, 291f.) already proposed credibility criteria to evaluate the performance of ABMs, it is notable that the community of analytical sociologists remains silent on the strategies of causal identification mainly developed by quantitative econometricians. Yet, a very recent publication from epidemiology (Marshall/Galea 2015) provided a first account to causal identification of ABM under the potential outcome framework. Concretely, ABMs can be assumed to represent an *in silicio* laboratory (Marshall/Galea 2015, 93) where model runs can be regarded as distinct treatments the results of which can be compared in order to arrive at a causal effect estimate under counterfactual conditions. The causal effect grounds on four variables (or matrices): $\mathbf{S}^t$ represents the agent population at time t; agents' interactions are captured by an agent-agent interaction matrix $\mathbf{K}^t$; apart from interaction (i.e., independent of other agents), agents find themselves in an environment $\mathbf{E}^t$; and a set of rules $\mathbf{Z}$ specifies how agents update their desires or beliefs, interact with other agents or react to the environment. Now, "agent-based modeling permits the examination of ensembles of counterfactual policy and programmatic scenarios, which may represent different populations (agent states), social interactions, environments, or combinations thereof" (Marshall/Galea 2015, 95): When counterfactual scenario A is denoted as $\mathbf{Z}_A$, $\mathbf{S}_A^T$, $\mathbf{K}_A^T$, $\mathbf{E}_A^T$, and counterfactual scenario B as $\mathbf{Z}_B$, $\mathbf{S}_B^T$, $\mathbf{K}_B^T$, $\mathbf{E}_B^T$, then the causal effect can be estimated as the difference in the expectation of outcome O obtained in a Monte Carlo simulation under a number of runs R.

Less formally, and in contrast to other methods, ABM can approximate the transformational mechanisms linking micro-level input conditions to macro-level outcomes by deliberately manipulating the value range of the input parameters (actors' desires and beliefs as well as their initial opportunity structure).

[5] One exception to the exclusion of mutual interdependence in quantitative models are multilevel analyses (Hox 2010) assuming that aggregated individual-level characteristics form a context which may influence individuals apart from their own characteristics (also see Brückner 2011, 672, 675). For instance, the Big-Fish-Little-Pond hypothesis postulates that *regardless of their own achievement*, students in high-achievement school classes show a lower academic self-concept than students in low-achievement classes (Marsh et al. 2008). Another exception are network models (Snijders/de Bunt/Steglich 2010; Snijders 2011)—which of course require that information on network structure has been surveyed in advance.

Thereby, it is possible to test which particular combination of parameters *can* bring about the explanandum in question. The identification and consistency assumption are satisfied due to the *in silicio* laboratory character, and spillover effects (e.g. Wersching 2010) can be either excluded or modeled and investigated. Yet, similar to all other methods reviewed above, the question necessary to answer when it comes to mechanism-based explanations is *how* the causal effect is brought about (*see section 3.3*).

2.4.2 Causal Heterogeneity

Marshall and Galea's (2015) potential-outcome notation of causal effects in agent-based modeling already points to some of the capacities of ABM to account for causal heterogeneity. Still in contrast to many applications of quantitative methods which omit to include interaction effects in their models, ABM inherently obliges researchers to model causal heterogeneity explicitly. Not only can causal complexities be modeled by different (counterfactual) scenarios of environments, actor networks and updating rules (Marshall/Galea 2015)—but ABM also allows for population heterogeneity in terms of *different* distributions of preferences and beliefs within the same model: "[T]he generativist wants an account of the configuration's attainment by a decentralized system of heterogeneous autonomous agents." (Epstein 1999, 43) Thus, adopting the counterfactual position, heterogeneity can be addressed in ABMs by deliberately changing agents' situational embeddedness, their preferences, beliefs, and decision rules. Furthermore, it is known that transformational mechanisms are not independent from the number of cases, which is why further sensitivity analyses may be required (Bruch/Atwell 2015, 198ff.; Manzo 2014, 36).

3. Empirical Methods for the Analysis of Social Mechanisms

While the assumptions of the potential-outcome framework are *necessary* to identify a causal effect, they are by no means *sufficient* to tell something about the underlying mechanism. Yet, similar to the issue of causal identification, also the endeavor to provide an answer to the *how*- and *why*-question of a causal effect suffers from disciplinary boundaries. In the following, I argue that having established causal identification, each of the empirical methods discussed so far disposes of suitable techniques to analyze what was above referred to as *IMO* relations, i.e., the generative process *how* the connection between an input or predictor and an output or dependent variable is brought about via a social mechanism.

3.1 Experimental and Observational Data

Although observational data require more sophisticated tools than experimental data in order to arrive at balanced samples, both data types share a technique that can be used to test for *IMO* relations: *mediation analysis*. Imai et al. (2011)

provide a methodological approximation to causal mechanisms within the potential outcome framework: "We define a causal mechanism as a process whereby one variable T causally affects another Y through an intermediate variable or a mediator M that operationalizes the hypothesized mechanism." (768) Hence, a straightforward way to operationalize social mechanisms in quantitative studies is to translate the input-mechanism-output framework into the potential outcome framework by stating that *a treatment affects an outcome via its mediator.*

One of the most famous definitions of mediation analysis was formulated by Baron and Kenny (1986, 1177; also see *figure 1*):

> "A variable functions as a mediator when it meets the following conditions: (a) variations in levels of the independent variable significantly account for variations in the presumed mediator (i.e., Path a), (b) variations in the mediator significantly account for variations in the dependent variable (i.e., Path b), and (c) when Paths a and b are controlled, a previously significant relation between the independent and dependent variables is no longer significant, with the strongest demonstration of mediation occurring when Path c is zero."

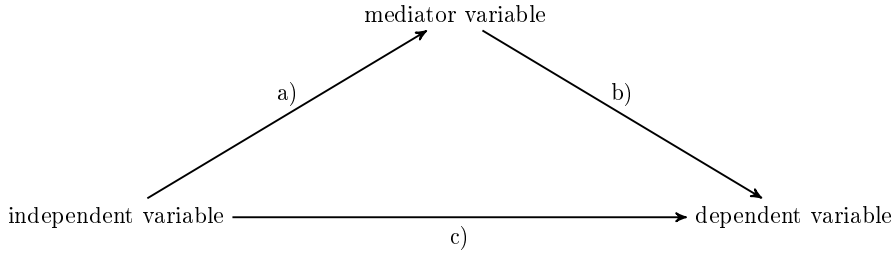

Figure 1: A simple mediator model. Source: Baron/Kenny 1986, 1176.

It is further common to distinguish between a direct, an indirect, and the total effect of a predictor (or treatment) on its outcome. In *figure 1*, the direct effect is denoted by path c, the indirect effect is the product of paths a and b, and the total effect is the sum of both the direct and the indirect effect, i.e. $c + a * b$ (Hayes/Preacher/Myers 2011).

While *figure 1* shows the most simple mediator model consisting of just one interfering variable, also more complex scenarios are possible (cf. *figure 2*). Diagram *(b)* shows the effect of treatment T on outcome Y being mediated by two different mediators M and N which are not related to each other. In other words, diagram *(b)* shows a convenient representation of two potential mechanisms of a treatment effect that should be tested against each other. In diagram *(c)*, the constraint of *(b)* is relaxed and mediator N is allowed to have an effect on mediator M.

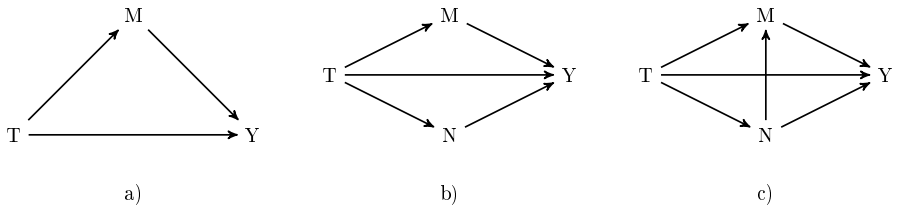

Figure 2: Diagrams representing various causal mechanisms. Adapted from Imai et al. (2011, 769).

However, it is important to note that *(c)* is *not identified* since M is affected by both the treatment and an additional mediator N. In more formal terms, Imai et al. note that *(c)* does not satisfy the assumption of *sequential ignorability* which can be divided into two parts (Imai et al. 2011, 770). *First*, as outlined above, the assignment of the treatment status has to be independent of both the outcome and potential mediators. This can either be assured by randomized experiments, or by conditioning on covariates in regression analysis or matching. *Second*, also the mediator must be ignorable given the treatment status (treated vs. non-treated) and pre-treatment covariates. This assumption is often neglected in observational studies, and it cannot be guaranteed by randomizing both treatment assignment and mediators.[6] In case of diagram *(c)* of *figure 2*, the second condition of the sequential ignorability assumption is violated because M is affected by post-treatment covariate N. Thus, to be fulfilled, it must be excluded that there are unmeasured pre-treatment or any post-treatment covariates affecting the mediator of interest (M in *figure 2*).

Given a mediator model can be causally identified in sense of *figure 2*, how should it be estimated statistically? Imai et al. (2011) extend the potential outcomes framework on mediation analysis. First, the outcome is predicted by the treatment, the mediator, and all pre-treatment covariates, and the mediator is predicted by the treatment and the pre-treatment covariates. Second, predicted values for the mediator under both treatment and control condition are generated. Third, the outcome is predicted under the treatment condition, but one time for the mediator taking predicted values estimated under the treatment condition, and another time for the mediator taking predicted values estimated under the control condition. The difference between the latter two predictions is called the Average Causal Mediation Effect (ACME) and represents an overarching framework that can be implemented within several statistical conditions.

In *section 2.2*, we already learned about moderator models as a quantitative technique to account for causal heterogeneity. A special case of moderation analysis are models where the treatment is not moderated by pre-treatment covariates but where we have an interaction between treatment and *mediator* (Imai et al. 2011, 784). Preacher et al. (2007) distinguish five ways mediation might coincide with moderation at the same time (*figure 3*; examples by myself):

[6] It is possible that individuals with a positive effect of the treatment on the mediator show a negative effect of the mediator on the outcome.

A) The independent variable is also the moderator. Example: The effect of social background on student achievement is mediated by academic self-concept, but only/ stronger for low achievement students.
B) Path a is moderated by W. Example: The effect of social background on student achievement is mediated by academic self-concept, while the effect of social background on academic self-concept is moderated by gender.
C) Path b is moderated by W. Example: The effect of social background on student achievement is mediated by academic self-concept, while the effect of academic self-concept on student achievement is moderated by gender.
D) Path a is moderated by W and path b is moderated by Z. Example: The effect of social background on student achievement is mediated by academic self-concept, while the effect of social background on academic self-concept is moderated by gender, and the effect of academic self-concept on student achievement is moderated by migration status.
E) Paths a and b are moderated by W. Example: The effect of social background on student achievement is mediated by academic self-concept, while both the effect of social background on academic self-concept and the effect of academic self-concept on achievement are moderated by gender.

3.2 Qualitative Data

The crucial step of qualitative data analysis to take a closer look at the social mechanism underlying a causal effect is a move from the *between-case* level (which is the level of QCA) to the *within-case* level (Mahoney 2000, 409ff.; Rohlfing/Zuber 2014). As causal inference based on small-N analyses may stand even more at risk of considering spurious correlations for causal effects, a closer look at the within level may identify the social mechanisms that are assumed to bring about the hypothesized causal effect (Mahoney 2000, 412f.). The most established method to analyze *IMO* relations is *process tracing*. In particular, two tests can be distinguished, *hoop tests* and *smoking gun tests*:

> "A hoop test proposes that a given piece of evidence—namely, a specific causal-process observation—must be present for a hypothesis to be valid. Failing a hoop test eliminates a hypothesis, but passing a hoop test does not confirm a hypothesis. Smoking gun tests, by contrast, propose that if a given piece of evidence—namely, a specific CPO [i.e., a causal process observation; *DB*]—is present, then the hypothesis must be valid. Passing a smoking gun test lends decisive support in favor of a hypothesis, though failing a smoking gun test does not eliminate a hypothesis." (Mahoney 2012, 571f.)

Van Evera's (1997, 31–32) illustrations of both tests read as follows: imagine a man is accused of having committed a murder. The question of whether the suspect was in the state in which the murder happened would amount to a hoop test. It is a necessary condition for having committed the murder, but not a sufficient one. In contrast, detecting the suspect holding a smoking revolver in his hand shortly after the murder happened would amount to a smoking gun

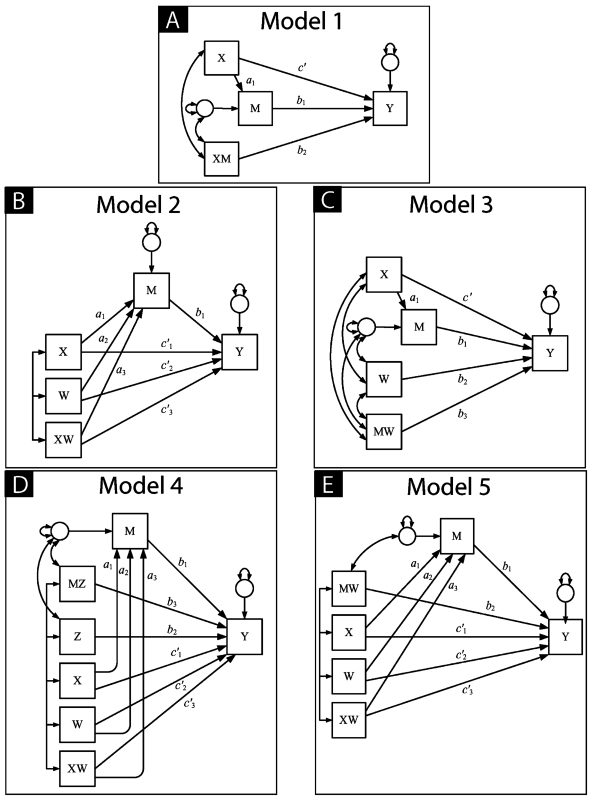

Figure 3: Path diagrams of five possible ways of moderated mediation (Preacher et al. 2007, 194).

test as it can be considered to be a sufficient condition for having committed the murder.

Importantly, both types of tests can be connected to the analysis of *IMO* relations. On the one hand, a *hoop test* on mechanisms can be used to reject the Null that a cause *cannot* be *necessary* for an outcome. If a researcher holds a hypothesis that a potential cause X is *necessary* for an outcome Y, she may first deduce from theory that a social mechanism M can be supposed to be a *sufficient* condition for Y. If then X is found to be necessary for M, the underlying hypothesis successfully passed the hoop test: "Logically speaking, X *cannot be necessary for* Y *unless it is necessary for all intervening conditions that are sufficient for* Y." (Mahoney 2012, 579)

On the other hand, a hoop test on mechanisms can also be used to reject the Null that a cause *cannot* be *sufficient* for the outcome. If X is sufficient for M, it is established that X can be sufficient for Y: "X *cannot be sufficient for* Y *if it is not sufficient for all intervening conditions that are necessary for* Y." (Mahoney 2012, 580)

Turning to the *smoking gun test*, on the one hand, it can be used to establish that a cause X *must* be *necessary* for an outcome Y. A smoking gun test is passed if both the cause X is shown to be necessary for a mechanism M and the mechanism M is shown to be necessary for the outcome Y: "It would be logically impossible for the cause to be necessary for the mechanism but not the outcome." (Mahoney 2012, 581)

On the other hand, a smoking gun test can be used to establish a *sufficient* cause X on an outcome Y as well. Quite in analogy to necessity, the smoking gun test is passed if it is shown that X is sufficient for a mechanism M and M is in turn sufficient for the outcome Y. "The assumption is that if the cause is sufficient for a mechanism that is known to be sufficient for the outcome, the cause itself must be sufficient for the outcome. It would be logically impossible for the cause to be sufficient for the mechanism but not the outcome." (Mahoney 2012, 582)

In sum, smoking gun tests and hoop tests are process-tracing methods that apply the logic of necessary and sufficient conditions on the identification of mechanisms on the *within-case* level.

3.3 Agent-based Modeling

The most important pledge of agent-based modelers concerning the analysis of social mechanisms is that ABM is not only able to analyze situational and action formation mechanisms, but also allows the shedding of light on what I would call *the dark side of Coleman's boat*, i.e. transformational mechanisms (e.g., Manzo 2007, 40). As actors are embedded within situations, equipped with a set of preferences and beliefs constituting their logic of selection, and, most important, also allowed to *interact* in order to aggregate from the micro to the macro level (Manzo 2014, 31), ABMs are inherently processual and thereby capable of analyzing *IMO* relations.

Yet, the crucial challenge for each ABM is whether not only an internally plausible, but also a *realistic* mechanism is established: "[T]he overall goal is not merely to get a validation of simulation results, but to empirically test theoretical mechanisms behind the model. Empirical data are needed both to build sound micro specifications of the model and to validate macro results of simulation (*sic!*)." (Boero/Squazzoni 2005, 2) Or in the words of Manzo (2014, 35), "[...] agent-based modeling cannot provide the proof that the mechanism(s) represented by the generative model under scrunity is(are) the mechanism(s) underlying the high-level regularities to be explained." This leads us to *empirically calibrated* ABMs and their potential to address both *IMO* relations and causal heterogeneity.

In the logic of agent-based modeling, situational and action formation mechanisms can be regarded as antecedent to the transformational mechanism of primary interest. Yet, unsurprisingly, the macro-level outcome of a transformational mechanism to be simulated is only valid in empirical terms (i.e., apart from pure theoretical abstractions; see below) if the preceding situational and action-formation mechanisms do not entirely deviate from actors' real-world pref-

erences, beliefs, decision rules, and situations they are located in: "[G]iven that a possible infinite amount of micro specifications (and, consequently, an infinite amount of possible explanations!) can be found capable of generating the k_a [i.e., the outcome generated by artificial data; *DB*] close to the k_r [i.e., the outcome] of interest, what else, if not empirical data and knowledge about the micro level, is indispensable to understand which causal mechanism is behind the phenomenon of interest?" (Boero/Squazzoni 2005, 3)

Concretely, three scenarios with different demands for 'empirical richness' can be distinguished, for each of which various data types such as experimental, quantitative, or qualitative data can be used (cf. Boero/Squazzoni 2005, 6ff.). In *case-based models*, the aim is to find a transformational mechanism for a specific case, sometimes to be followed by policy recommendations. Case-based models can be enriched by finding appropriate macro and micro data about a particular time-space. For instance, imagine that the impact of a potential policy on reduction of contagion effects concerning youth deviance in a particular urban district should be analyzed. In that case, the model could be enriched by quantitative district-level information on the initial distribution of youth deviance and the degree towards which it is known to affect youths with particular preferences and beliefs.[7] *Typifications* should be applied to a wider range of empirical phenomena sharing common properties. The idea is to unveil social mechanisms that can be used to explain a similar set of empirical phenomena. For instance, in her ethnographic analysis of dissent in organizations, Vaughan (2009, 697) found that in prison, the workplace, and the family, the same set of five mechanisms (which she labeled 'social location', 'power dependence relations', 'norms of loyalty', 'organizational retaliation', and 'social support') explained behavior such as resisting authority, speaking out against illegal, illegitimate, or immoral action etc. Agent-based modelers could draw on this evidence to test to what extent some transformational mechanism of mutual influence might help to explain the empirically observed rates of organizational dissent.

Third, *theoretical abstractions* have a more generative demand in that they strive to find new insights and propositions for theoretical puzzles unsolved hitherto. Typically, they neither refer to particular spatio-temporal circumstances, nor do they try to explain a limited set of empirical phenomena. The most famous theoretical abstractions are Schelling's (1978) segregation model and Axelrod's TIT-for-TAT model of cooperative behavior in repeated games of strictly rational actions (Axelrod/Hamilton 1981; Axelrod 1984). The idea of the latter is not that strictly rational behavior of actors is an exceptional realistic assumption—but that *even in that case*, an equilibrium of stable cooperative behavior is *possible*. For theoretical abstractions, "the goal is not to reproduce existing patterns or even to anchor agents' behavior, characteristics, or environment in empirical knowledge. Rather, the models are generative; they develop new ways of thinking about a problem and provide a great deal of theoretical stimulation for existing empirical research." (Bruch/Atwell 2015, 192)

[7] Of course, quantitative data usually assume observations to be independent from each other apart from the 'pure' contextual effect.

Yet, the last part of the quotation points to an important issue: even for theoretical abstractions, it does not suffice to build an ad-hoc ABM without consideration of the empirical literature. Quite in contrast, the most seminal models such as the ones by Schelling and Axelrod built on a strong body of existing empirical research (Boero/Squazzoni 2005, 15). Of course, the *extent* towards which empirical enrichment is required varies between all three types. Theoretical abstractions need only a little while case-based models require quite rich empirical information (Bruch/Atwell 2015). However, when it comes to external validation, a very abstract ABM might require "an analysis of empirical data that is completely separate from the agent-based model" (Bruch/Atwell 2015, 207).

Finally, also for the issue of causal heterogeneity, more realist simulation models are useful. First, similar to the question whether the assumptions of the underlying situational and action-formation mechanisms are realistic, I argue that assumptions about population heterogeneity in general and about heterogeneous effects of similar situations on agents with different dispositions in particular benefit from empirical calibration as well. As discussed in the section on treatment effect heterogeneity and moderator models, elaborate experimental and quantitative techniques are available to address this issue. Furthermore, qualitative comparative analysis (QCA) may account for higher-order causal complexities such as INUS conditions that are, just to recall, "an insufficient but necessary part of a condition which is itself unnecessary but sufficient for the result" (Mackie 1965, 245). Hence, before setting up an ABM that is supposed to consider causal heterogeneity in a realistic way, simulation modelers should apply the available quantitative and qualitative methods for causal heterogeneity to empirical data and use these external results to implement the heterogeneous real-world mechanisms into the agent-based model.

Second, a known issue in agent-based modeling is what the philosophy of science calls the problem of *multiple realizability* (Bickle 2013): analytically, an infinite amount of micro specifications can bring about the same macro-level outcome (Boero/Squazzoni 2005, 4; Manzo 2014, 35). It was argued by Ragin (1999, 1227f.) that QCA is a suitable technique to deal with these kinds of causal complexities. Hence, having already set up an empirically-calibrated ABM, QCA can be used to examine how different *combinations* of counterfactual conditions—which have been built up in accordance with the empirical literature—may bring about the macro-level outcome of interest (see Franzmann/Schmidt, in this volume, 227–256).

4. Summary and Conclusion

The aim of this outlook was to set the ground for a common framework of causal identification and empirical tests of social mechanisms for experimental, quantitative, qualitative, and simulation analyses (agent-based modeling). The main arguments are summarized in *table 2*.

I am aware of the fact that this chapter could only touch upon a small subset of techniques for each of the empirical methods reviewed, and any expert in their respective field might legitimately accuse my presentation to be at least incomprehensive. Notwithstanding any qualified objection, critics might still second my proposition that a unified view on better linkages between questions of model identification on the one hand and empirical implementations of the crucial ideas of mechanism-based explanations on the other hand might make a contribution to advance the underlying research program.

	Causal identifcation	Causal heterogeneity	Input-mechanism-output relations
Experimental data	Randomization	Moderation analysis	Mediation analysis
Observational quantitative data	Selection on observables: regression, matching, weighting; selection on unobservables: instrumental variables, selection models, differences-in-differences	Moderation analysis	Mediation analysis
Comparative qualitative data	Mill's methods of difference and agreement; more cases than conditions available	Qualitative comparative analysis (QCA) with causal complexities	Process tracing methods (hoop tests; smoking gun tests)
Agent-based modeling	Comparison of counterfactual simulation scenarios	Pre-calibration tests for causal heterogeneity (moderation models) and causal complexities (QCA); post-calibration QCA of ABM results	Empirically calibrated situational and action formation mechanisms

Table 2: Overview on techniques for causal identification, IMO relations and causal heterogeneity in experimental, quantitative, qualitative, and simulation methods.

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