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Some Remarks on Effect Studies of Physician Dissatisfaction

The topic of several papers in this special issue (e.g. Angerer & Weigl, 2015; Casalino & Crosson, 2015; Konrad, 2015) is causes and/or consequences of physician (dis-)satisfaction. This topic is important for several reasons. First, regarding the doctors themselves, we want healthy doctors because they as all other individuals deserve a good life. Secondly, for the patients we want healthy doctors because healthy doctors presumably are better doctors. Thirdly, for the society we want doctors to stand in work life for as a long time as possible and deliver good health care quality.

When that is been said, physicians do probably not belong to the most vulnerable occupations or professions. Based on register data, a comparison of 25 professions (occupations demanding higher education) showed that workers in low-status caring professions were more exposed to risk of disability pension than individuals with other professional education backgrounds, including physicians (Tufte, 2013). Possible explanations are mechanisms related to selection effects (who chooses various professional educations), physical and mental job strain, and professional ethics.

Since most of the papers in this special issue focus on detecting causal relationships, I would like to present a few general comments from the viewpoint of research methodology and philosophy of science. I argue that we ought to reflect on the *concept of causality*. What do we mean when we conclude that there is a causal relationship between various phenomena? Do we base this conclusion on a conception of robust regularity, counter-factuality, intervention, causal capacity or mechanism? In my opinion, a mechanism approach to causality is most adequate. Moreover, I argue that we must be conscious of the level of explanation. Mechanism approaches to causality usually stress the importance of a micro-foundation in scientific explanation. I point, however, to the importance of including and studying the relevance of the *meso-level*, that is, the level of social groups and organization, for the relation between cause and outcome. Finally, we ought to include a conception of *context and intervening mechanisms* in our studies of causal relations. We need to understand when contexts trigger and prevent specific causal mechanisms.

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The concept of causality

There are in principle two ways one can detect causal relationships, by introducing control variables (also known as third variables) in multivariate analysis of observational data, or by conducting interventional studies (experiments). Interventional studies have in general higher internal validity, that is, ability to identify a true causal relationship. The distinction between the control variable method and exper-

iment is related to the great philosophical discussion of causality. Researchers usually steer clear of philosophical discussions, but in this case it may enlighten us.

There are several conceptions of causality (cf. Beebee, Hitchcock & Menzies, 2009). One conception is that causality implies *robust regularity*. If there is a causal relationship between physician discontent and bad quality of health care, we expect to discover a regularity between these events, that is, when we observe discontented doctors we also expect to observe bad health care. Presumably, the relationship is not perfect or deterministic, but we expect at least there to be a probabilistic relationship, a correlation. Among the problems with this conception of causality is the fact that correlation is not identical to causation.

The control variable method for detecting causal explanations goes with the regularity view of causality. We must, nevertheless, be aware of several pitfalls. The first pitfall is *selection effects*: Are there social processes ahead of our observations that may influence the composition of individuals in the categories defines by our research variable(s)? The result of selection is that presumed comparable categories actually are not comparable. Selections have a tendency to sneak in through the backdoor both in observational and interventional (quasi-experimental) studies. For instance if one compares different departments or clinics, these clinics may from the outset have different kinds of physicians or patients. If there is a selection, and if we do not control for the variables describing this selection, we may detect a spurious relationship between physician distress and quality of health care. If one clinic serves patients in a poor district and another in a wealthy district, the patients and doctors probably vary substantially between the clinics, and this fact may induce correlations between work conditions, physicians health and quality of treatment (for instance in the form of incidents of malpractice.

Secondly, and more generally, we have to be aware of potentially unclosed "*backdoors*" (Pearl, 2009): Are important "backdoors" closed (or not reopened), that is, have one controlled for confounding variables that causes correlations between other variables? The important point is how to avoid making causal inferences from spurious correlations. The main strategies are random controlled tests (RCT), various strategies for controlling for third variables (multiple regression, matching, etc.), instrumental variables, complete set of mechanisms (Morgan & Winship, 2007).

Thirdly, we must be conscious of the causal direction: Is the relationship reverse or reciprocal? It could be the case that bad health care quality causes physician discomfort rather than, or in addition to, the opposite. One example could be that physicians in a clinic for some reasons (not dissatisfaction) initially delivers bad quality of care. This may cause physical and mental stress reactions for the physicians, but also precautions at the organizational and management level that affect the psychosocial work conditions. This model with reversed causality can also explain a correlation between doctor discomfort and quality of health care in observational studies. If these mechanisms exist, they bring about an endogeneity problem, making it hard to estimate the true causal relationships—unless one is in the position of instrumental variables that make it possible to estimate the true causal effect.

Of course, in interventional studies the question of the causal direction is not a problem because of the manipulation of the independent variable. In quasi-experiments however, there may be unobserved heterogeneity between control group and experimental group. Other observational designs that may increase the possibility of detecting causal directions is of course longitudinal studies, in particular panel studies.

Another conception of causality is *counter-factual*. The view is that causality implies that the effect had not occurred if the cause did not occur. If doctor dissatisfaction affects health care quality negatively, without doctor dissatisfaction there would be only good health care quality. The problem is, however, that dissatisfac-

tion can have a negative, causal influence on quality—even if we observe bad health care when doctors are satisfied or good quality when doctors are dissatisfied.

Doctor dissatisfaction might be a necessary, but not sufficient, cause of bad health quality. Therefore, we will observe cases of good quality even when doctors are dissatisfied. The reason is that doctor dissatisfaction is some cases is not enough to imply bad quality. Other causes may prevent the effect (bad health care) from occurring. It may also be that doctor satisfaction is a sufficient, but not necessary, cause of bad health care. Then we will observe cases of bad quality even when doctors are satisfied, because other causes also may induce bad health care.

We will probably expect that doctor discontent is an INUS condition for bad health care, that is, "insufficient but necessary parts of a condition which is itself unnecessary but sufficient for result" (Mackie, 1965, p. 245). Doctor dissatisfaction is not a cause that operates in a vacuum. It must presumably be part of several other conditions (for instance bad control routines etc.) to cause bad health care quality. This package of conditions is sufficient to influence health quality, but other packages of causes may also give rise to bad health quality.

Actually, even the INUS-condition may be asking too much. We may observe causes being unnecessary and insufficient parts of a condition that is unnecessary and insufficient for the occurrence of the effect. Doctor dissatisfaction combined with several contextual factors may increase the risk of bad health care, but other conditions may also cause bad health quality. Moreover, additional conditions may also prevent the negative influence of doctor dissatisfaction. There are always several conditions and mechanisms at work simultaneously. Outcomes are usually not influenced a single condition, but by several intervening conditions. Trying to single out one particular condition or mechanism is usually a challenge.

A third candidate related to the counter-factional definition, but still different from it, is the *manipulation* definition of causality. In this view, causality implies (at least in principle) the possibility of bringing out the effect by manipulating the cause. If we could reduce the satisfaction of doctors (or substitute existing doctors with identical although dissatisfied doctors) we would expect bad health quality to occur. The rationale behind this is clear, if we can identify critical aspects in the psychosocial work conditions that strongly affect physicians health and quality of patient care, we may also be in the position of manipulating these factors in order to improve both quality and care.

Manipulation is the principle behind the experiment. In the wake of the success of evidence based practice and policy-making, randomised controlled trials tower as the gold standard of knowledge, scorning the relevance of everyday knowledge. No other research design imply the same degree of internal validity. Nevertheless, one gets the impression of too great optimism (or even naivety) for the evaluation of effects in experimental or observational studies. Randomized and double (even triple) blind studies almost ensures isolation and detection of real causal effects.

In social experiments, as a rule, fixing confounding processes is next to impossible. Firstly, we are hardly ever capable of ensuring that manipulation is identical between, or even within, test cases and control cases. There is usually too much going on simultaneously. Working conditions, management policies, influence from co-workers, patients, and so on, give rise to a multitude of combinations that one hardly can control for by randomization. What we can hope for is that the cases are sufficiently similar. This matter concerns the construct validity of the test variable. Often there will be variability in this variable that is not due to variation in our theoretical concept, thus inducing more or less severe bias in our estimates. Another danger is disregard of the context. Often one seeks universal effects, but outcomes are often result of a conglomerate of context triggers and intervening mechanisms. There is a trade-off between the internal and the external validity of an experiment. In order to create good internal validity the experimenter has to withdraw from real life situations because they contain too many factors we ought

to hold fixed. Thus, the external validity suffers.

Pawson (2013) claims that in general the average effects of interventions are often approach zero. If one studies effects of interventions over various contexts, the likelihood of observing no average effect is rather high. Zero effect studies often conceal the fact that the effect in specific contexts can be stupendous in either positive or negative direction. This is a criticism of RCT's in general but it probably strikes meta-analyses even harder. Thus, experiments and meta-analyses do not represent the gold standard without reservation.

The mechanism approach

The discussion above points to the importance of mechanism explanations. Mechanism explanations are not opposed to either of the causal approaches above. The main difference is the focus on fine-grained explanations. While experiments are not appropriate to study the causal story, that is, why or how the cause influences the effect, the mechanism approach focuses entirely on this story. The mantra is "no causal explanation without mechanisms". A mechanism is a detailed story of the causal process from cause to effect. An illustrative example from medicine is the difference between knowing *that* a medicine has an effect (on an illness) and knowing *why/how* a medicine affects the disease. Related to the subject of physician dissatisfaction, we want to know why/how physicians become dissatisfied and why/how dissatisfied doctors are bad doctors.

The mechanism approach emphasizes the importance of a micro level foundation of explanations. The basic tenet is that no social or human event can occur without individuals that are acting and interacting (Hedström, 2005). Thus, we need to study the *desires, beliefs and action opportunities* that affects individual actions and interactions between individuals. Our explanations must have a plausible psychological (and biological) basis (Elster, 2007). In my opinion, Konrad (2015) and Angerer & Weigl (2015) aim at outlining mechanisms between doctor dissatisfaction and bad health care quality. There are several psychological theories that could explain the relationship, or make it more plausible. Lack of focus can be one mechanism: Discontented physicians become more occupied with their own emotions or with troublesome working conditions, and less attentive towards the story of the client. Blaming the victim is another mechanism: Distressed and in particular burned-out physicians may blame the client for their problems and thus be less responsive to the needs of the client. It can however, be difficult to determine the right candidate(s).

However, several social scientists and philosophers have questioned this reductionist approach to explanations (Risjord, 2014). While recognising that only individuals act and interact, there is an increasing focus on the meso-level (groups of individuals, organisations, etc.) as an important mediating level between macro and micro. Thus, in my opinion, it is appropriate to focus on how working environment moderate the effect of doctor distress on quality of health care. Medical clinics may for instance be organised in way that physicians cannot communicate discontent to management. Another intensifying mechanism relates to fact that physicians may work with patients in isolation from other colleagues. Informal norms among physicians may also influence on the relation between discontent and quality of health care.

There is a potential pitfall here, namely the ecological inference fallacy. One risk in studies of clinics and departments is that relationships on department level are not necessarily corresponding relationships at the individual level. When studies find that average or aggregate level of stress correlates with levels of malpractice at the department, this does not necessarily imply that there is a corresponding relationship at the individual level. In short, we do not know whether the dissatis-

fied or the satisfied doctors make the errors or not. Moreover, we do not know the direction of the relationship.

Of outmost importance within the social mechanism approach is the focus on context and intervening mechanisms. Intervening mechanisms are mechanisms that work in the same or opposite directions. Lack of focus and blaming the victim are mechanisms that work in the same direction. Ethical norms stressing that patient considerations are paramount to all other considerations, will work in the opposite direction.

Some contextual features trigger, prevent, reinforce or attenuate the effect of particular mechanisms. Open communication between management and physicians and less isolation may for instance attenuate, or perhaps even prevent, mechanisms like blaming the victim and lack of focus. As we have seen, lack of sensitivity towards contexts is the reason why RCT's and meta-analysis may fail. We have to interpret the results. One must identify deviant cases and study their contexts in order to get an impression of the real causal process. Usually, we ought to supplement experimental data with rich quantitative and/or qualitative data that permits the identification of context-mechanism connections that are relevant for the outcome. The "gold standard" would then be a mixed research design: experiments (or observational studies) combined with qualitative case studies.

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