**REVIEW ESSAY**

**Causality, mosaics, and the health sciences**

*Olaf Dammann*

*Dept. of Public Health and Community Medicine*

*Tufts University School of Medicine*

*Boston, MA 02111*

**Olaf.Dammann@tufts.edu**

# Introduction

Thinking about illness causation has a long and rich history in medicine [1-4]. After a hiatus in the 1990s, the last one-and-a-half decades have seen a surge of publications on causality in the biomedical sciences. Interestingly, this surge is visible not only in the medical [5], epidemiological [6], bioinformatics [7], and public health [8] literatures, but also among philosophical publications [9-15]. In this essay, I review and discuss one most recent addition to the literature, *Causality: Philosophical Theory Meets Scientific Practice*, written by philosophers about causality in the sciences, and particularly about the health sciences [16].

“Causality” denotes a relationship between entities that cannot easily be captured by words. Causality is intangible, invisible, and impossible to ascertain beyond lingering doubt. One can describe causation in many different ways, for example, as a deterministic or probabilistic relationship, as a mechanism or process, or as what happens after an intervention [17]. Sometimes, such descriptions contradict one another; sometimes they appear to be compatible.

Looking at many different accounts of causality, arranged like a mosaic,[[1]](#footnote-1) might provide more insight than looking at each one individually. This is, in a nutshell, what Phyllis Illari and Federica Russo propose in *Causality* [16]. In this review, I offer my thoughts about the book, and the authors’ underlying theoretical framework.

The book is an excellent resource, well written and referenced, and a sheer pleasure to read. The authors’ two main goals are to offer an introduction to the philosophy of causality and to be highly accessible. They have clearly achieved both goals. I recommend the text to medical and epidemiology students, fellow epidemiologists, and physicians unfamiliar with the philosophy of causality.

 “Causality” has a somewhat peculiar, but highly effective structure. It is organized into 4 parts (Prelude, Concepts, Approaches, and Mosaic), which I will review in sequence.

# Prelude

In the first chapter, Illari and Russo present five scientific problems they consider important motivations for bringing together philosophical theory and scientific practice: inference (does X cause Y?), prediction (what will happen?), explanation (how does X cause Y?), control (how to control causal systems?), and reasoning about causality. In essence, the entire book is a whirlwind tour through the classical, but mainly recent, literature that attempts to answer these questions. The authors provide what they call “a scientific toolbox for philosophy” in chapter 2 and “a philosophical toolbox for science” in chapter 3; both are helpful in paving the way for the second part of the book.

# Concepts

Part 2 is a series of fifteen chapters on specific notions of causality, e.g., necessary/sufficient, probabilism, counterfactuals, manipulation, processes, mechanisms, information, dispositions, regularity, variation, action, inference, and others. The collection of chapters in Part 2 turns out to be a bit of a tradeoff. The good news is that each one is short (minimum 4, median 8, maximum 13 pages) and refreshingly concise. The not so good news is that each one is so short that it leaves some ground uncovered. I find the first three chapters in Part 2 particularly interesting because they provide a fresh perspective on classic attempts in epidemiology and medicine to get a handle on causation, the problem of criteria for illness causation [18], the problem of causes versus conditions of illness [19], and the problem of illness in individuals versus populations [20].

In chapter 4, Illari and Russo illustrate how closely related the philosophical and epidemiological ways of thinking about causality can be. They show that Ken Rothman’s model of sufficient constellations of insufficient component causes [19] is in fact an epidemiological adaptation of Mackie’s insufficient non-redundant parts of unnecessary but sufficient causes (a.k.a., INUS conditions) [21]. The Rothman model conceives of sufficient causes of illness as constellations of component causes, e.g., C­­s1 ≡ {cc1, cc2, cc3, cc4}, C­­s2 ≡ {cc1, cc4, cc5, cc6}, C­­s3 ≡ {cc1, cc6, cc7, cc8}. If one assumes that C­­s1,2,3 account for similar proportions of all causes of the disease of interest, each one causes one third (33.3%) of all cases. Note that *none* of the three sufficient causes (or better, causal constellations) is *necessary* for the disease to occur. None of the component causes alone is sufficient and only cc1 is necessary, because none of the sufficient causal constellations is complete without cc1. All other component causes are insufficient *and* unnecessary. The philosophical parallel here is that each one of the component causes cc1-8 qualifies as a Mackiean INUS condition.

Illari and Russo turn to semantic differences between the philosophical and scientific terminologies in chapter 5. They write about levels of causation and the not so obvious difference between the type/token distinction in philosophy and the person/population distinction in medicine and epidemiology. While the former distinguishes between events that recur with some regularity (e.g., the phenomenon of earthquakes) and a single instant (one particular earthquake), the latter distinguishes between population-level occurrence (e.g., the annual incidence of myocardial infarcts in Germany in 1995) and person-level occurrences (my Godfather’s myocardial infarct there that same year). Illari and Russo suggest bridging the levels and the terminology by replacing both dyads with the terms “generic” and “single case” [16, p. 41].

In chapter 6, on evidence, Illari and Russo discuss the Hill viewpoints on causal interpretation of associations [18] compared to their own accounts of causal interpretation in the health sciences, i.e., the Russo-Williamson thesis [11, 22][[2]](#footnote-2) and its disambiguation by Illari [24]. They briefly mention evidence-based approaches, including evidence-based medicine, which has been the focus in recent papers written with the Russo-Williamson thesis in mind [14, 26]. The authors critique the idea of “evidence hierarchies” that actually represent hierarchies of methods of evidence gathering, not hierarchies of what we gather evidence of**.**

Chapters 7–18 represent a formidable overview of current philosophical concepts of causality. Chapter 7 covers causal methods, e.g., Bayesian networks, structural and multilevel models, as well as contingency tables. Chapter 8 covers probabilistic accounts of causality, which are of particular importance for medicine, where risk factors only rarely determine disease occurrence, but rather, increase its likelihood. The next chapter addresses a perhaps even more crucial issue in medicine and epidemiology: counterfactual reasoning such as, “Had the patient not received this antibiotic, she would have died in a septic shock.” Counterfactuals provide the underlying logic for all medical intervention; they also represent the motivation to consider randomized trials the *sine qua non* in evidence-based medicine. The so-called “potential outcomes approach” is also rooted in counterfactual reasoning and is currently advertised as “the modern approach” to causal inference in public health [8]. Based on the ideas of Paul Holland [27] and Donald Rubin [28], and developed further by Jamie Robins and Miguel Hernán [29, 30], the potential outcomes approach is rooted in the idea that an individual who is given a certain treatment (or not) could experience a certain outcome (or not). The outcome difference (present vs absent) between the two groups (treated vs. untreated) is then *defined* as being causal if certain additional requirements are fulfilled [31]. The main point of criticism against this causal interpretation is the assumption that the *observed* outcome in a group of *untreated* individuals is a solid estimate of what *would have been* observed (but was not) in the *treated* individual had she not been treated (missing data problem).[[3]](#footnote-3) Chapter 10 discusses the manipulationist stance, which holds that an exposure *E* causes an outcome *O* if and only if changes in *E* that are due to its manipulation are followed by changes in *O.* Taken together, chapters 8-10 are a good basis for those who want to understand the philosophical underpinnings of why the randomized-controlled trial is often considered the gold standard of causal reasoning in medicine.[[4]](#footnote-4)

Chapters 11 and 12 deal with process and mechanism theories of causation, respectively. The authors point out that process theories are rather physics-focused and that their “book is an encouragement to look beyond the realm of physics to understand causality” [16, p. 114]. The review of mechanistic accounts of causality and the recognition that “all of the major mechanists agree that a mechanism gives a *causal* explanation” [16, p. 126] provides a foundation for accounts of illness causation in biomedicine that view disease pathogenesis as part of, but not equivalent to, etiology, because “disease etiology includes the pathogenesis as the disease mechanism that occupies the black box between exposures and outcomes” [26].

In chapter 13, Illari and Russo describe information-theoretical accounts of causation that focus on the kind of connection between cause and effect and that can help explain phenomena of causal production, e.g., in the transmission of infectious diseases. In chapter 14, they introduce views that conceptualize causation as the consequence of “capacities,” “powers,” or “dispositions,” defined as “potentialities that (have) effects under certain conditions” [16, p. 152]. Chapter 15 describes causality as regular patterns, i.e., as the Humean stance that causality is “what actually happens in the world is regular succession” [16, p. 164]. Illari and Russo point out what such a view cannot do, e.g., treat singular instances of cause-effect occurrence as causal. In chapter 16, variational causal reasoning is described, in essence, as the observation (in experimental or observational settings) of “what varies with what,” yielding an overlap with regularity and manipulationist accounts. The authors address action theories of causality in chapter 17, the idea being that “causal relationships are relationships that agents can use to get what they want” [16, p. 187].

Chapter 18 reviews multiple approaches that ask “the question of what causality is by looking to our inferential practices” [16, p. 191]. These recent theories all focus, in a sense, on the anthropocentric aspects of causality: our causal expectations projected out into the world (Helen Beebee), causality as a feature of our epistemic representation of the world (Jon Williamson), causal inferential systems as related to social reasoning practices (Julian Reiss), and “ranking functions,” which account for degrees of belief (Wolfgang Spohn). This final chapter of Part 2 is, in itself, an extremely well-crafted exhibit of cutting-edge work in a branch of philosophy of science that is likely to influence future theories of evidence. This, in turn, will have repercussions for evidence-based medicine and public health.

# Approaches

Part 3 is an unusual and highly interesting component of the book. It begins with a description of what Illari and Russo call “CitS,” the Causation in the Sciences community. Part developmental narrative and part multidisciplinary manifesto for the integration of philosophical and scientific techniques, chapter 19 is basically about the authors’ intellectual motivation for their work.

The next chapter describes how examples of causality are employed in philosophical and scientific texts (philosophical thought experiments or scientific empirical ones). Often these examples are so absurdly unrealistic that it remains unclear how and why such examples can help at all in an argument for or against a certain account of causality that is supposed to matter in real life. Illari and Russo offer helpful suggestions for how to use examples and counterexamples in philosophical arguments.

To promote cross-disciplinary communication, Illari and Russo devote chapter 21 to the introduction and comparison of two approaches to causality in philosophy and the sciences. First, they describe how scientists use models to tackle causation. They suggest that it “is not so much whether models allow us to establish the *truth* of a causal claim, but rather whether they are of any *use* for a given problem” [16, p. 229], especially the problems listed in the Prelude (inference, prediction, explanation, control). Second, they write about the identification by philosophers of so-called truthmakers, i.e., what it is in the world that makes causal claims true. In their comparison of the truthmaking and modeling approaches, Illari and Russo declare that

we’ll know whether [a] proposition is in fact true or not by checking the world out there.… But prior to empirical investigation, why should we suppose that the reality we are interested in has a rigid constraint on what the causal relation and its relata must be? Indeed, the reasons for pluralism about causality we examine … suggest that we have evidence that it cannot be. Allying this with the arguments in the truthmakers literature, we suggest the release of the ‘One Truth’ straightjacket on the truthmakers approach. The resulting picture changes enormously!” [16, p. 233]

Illari and Russo suggest that the collaboration between philosophers and scientists can be much more fruitful if they start by allowing for as many *kinds* of causation as there are *accounts* of causation. The last chapter in this part is a cogent argument in support of the idea to integrate epistemological, metaphysical, methodological, semantic, and usage-related questions about causation and to think about the ways the answers to these questions inform each other.

# Mosaic

In Part 4, Illari and Russo proceed in two steps. First, they defend their version of pluralism. They offer what they call a “cheerful conceptual pluralism”, which draws upon *“all the developed accounts and theories in the literature”* [16, p. 256]. They call their form of pluralism “the pluralist mosaic,” borrowing the term from Carl Craver,[[5]](#footnote-5) who used it to suggest that “the mosaic unity of neuroscience is achieved both through interfield integration at a given level and through integration across levels in a hierarchy of mechanisms” [35, p. 228]. Craver’s “mosaic unity” (of neuroscience) captures the scenario in which different fields of neuroscience contribute different kinds of evidence with different forms of constraints that are “integrated piecemeal as research progresses” [35, p. 231]. But building on Craver, Illari and Russo envision an even broader mosaicism, one of integration across the sciences and philosophy, across types of causing, concepts of causation, types of inferences, sources of evidence, and across methods for causal inference. They are pragmatists in that they are task-oriented: “different concepts help in building causal knowledge, in virtue of their usefulness for specific tasks” [16, p. 257].

In their final chapter, Illari and Russo offer an example of how causal mosaicism can be used in practice. They describe “exposomics” as “the science of exposure” [16, p. 260][[6]](#footnote-6) performed by multiple research groups from various fields, rooted in molecular epidemiology using biomarkers of exposure, internal responses, and outcome [37], integrating knowledge from all biomedical, basic, and social sciences [16, p. 264]. They demonstrate how to select accounts of causality that might be useful when assembled as a mosaic. I share the authors’ hope that this approach will help us to “think better about science” [16, p. 271].

# Comments

Although the book’s subtitle is “philosophical theory meets scientific practice,” the book is mainly about causality and causal inferences in medicine and the health sciences, particularly in epidemiology. It might have helped to advertise this more clearly, since scientists working outside the health field might expect the book to address science a bit more broadly.

The book would have benefitted from a close reading by an epidemiologist to avoid several errors. For example, when discussing “observational methods” in chapter 2, Illari and Russo describe (mainly epidemiological) study types. In the section on case-control studies they write that “those who have been exposed to some factor (the cases) are compared to those who have not been exposed (the controls)” [16, p. 11]. Although this error has no impact on their overall thesis, it is plainly wrong. The exposed are compared to the unexposed in cohort studies, not in case-control studies.

Again in chapter 4, Illari and Russo struggle with intricacies of epidemiological terminology and concepts. They describe effect modification as “the ‘characteristics’ of diseases (the effect or effects) measured at different levels of other variables (the causes)” [16, p. 28]. In fact, effect (measure) modification refers to differences between subgroups defined by the effect modifier variable with regard to the magnitude of measures of association (estimates of effect) between risk factor and disease. A simple terminological difference between philosophy and epidemiology explains this misunderstanding. While philosophers talk about cause and effect, epidemiologists talk about exposures (risk factors) and outcomes (diseases). Thus, for philosophers, the effect *is* the outcome; for epidemiologists, the effect is what happens between exposure (to risk factors) and outcome (disease) occurrence. Such misunderstandings could have been avoided, but they certainly do not invalidate the text.

The main question, however, is what contribution their pluralist *mosaic* approach to causality will make for scientists and philosophers. I think that it is important to note that the mosaic approach is just that: an *approach* to how to use all available accounts of causality in an attempt to help clarify (identify and refine) scientific questions and problems [16, p. 270]. It is *not* a novel account of causality that can be compared to the currently available accounts referred to in Part 2 of the book. Thus, the proposed model is one that might help scientists and philosophers *to do better work* by helping them to *think better about their work*.

Illari and Russo have provided us with an excellent overview and comprehensive discussion of causality in the sciences, and with cogent arguments in support of the “usefulness of a *plurality* of methods and concepts for causal analysis” [16, p. 57]. Despite its few conceptual and terminological wrinkles, the book is a great contribution to the literature that informs interdisciplinary collaboration between philosophers and scientists.

# Acknowledgements

I am grateful to James A. Marcum for more than just a few suggestions on how to improve this paper.

# References

1. Feezer, L.W. 1921. *Theories concerning the causation of disease.* *American Journal of Public Health* 11(10): 908-912.

2. Susser, M.W. 1973. *Causal thinking in the health sciences: Concepts and strategies of epidemiology.* Oxford: Oxford University Press.

3. Nordenfelt, L., and B.I.B. Lindahl. 1984. *Health, disease, and causal explanations in medicine*. Dordrecht: Kluwer Academic Publishers.

4. Evans, A.S. 1993. *Causation and disease: A chronological journey*. New York: Plenum.

5. Kerry, R., T.E. Eriksen, S.A. Lie, S.D. Mumford, R.L. Anjum. 2012. Causation and evidence-based practice: an ontological review. *Journal of Evaluation in Clinical Practice* 18(5): 1006-1012.

6. Parascandola, M., and D.L. Weed. 2001. Causation in epidemiology*.* *Journal of Epidemiology and Community Health* 55(12): 905-912.

7. Mandel, J., N.M. Palfreyman, J.A. Lopez, W. Dubitzky. 2004. Representing bioinformatics causality*.* *Briefings in Bioinformatics* **5**(3): 270-283.

8. Glass, T.A., S.N. Goodman, M.A. Hernán, J.M. Samet. 2013. Causal inference in public health*.* *Annual Review Public Health* 34: 61-75.

9. Thagard, P. 1999. *How scientists explain disease*. Princeton: Princeton University Press.

10. Thygesen, L.C., G.S. Andersen, and H. Andersen. 2005. A philosophical analysis of the Hill criteria*.* *Journal of Epidemiology and Community Health* 59(6): 512-516.

11. Russo, F., and J. Williamson. 2007. Interpreting causality in the health sciences*.* *International Studies in the Philosophy of Science* 21(2): 157-170.

12. Woodward, J. 2010. Causation in biology: Stability, specificity, and the choice of levels of explanation*.* *Biology and Philosophy* 25(3): 287-318.

13. Kincaid, H. 2011. Causal modelling, mechanism, and probability in epidemiology. In *Causality in the sciences*, ed. P. Illari, F. Russo, and J. Williamson, 70-90. Oxford: Oxford University Press.

14. Clarke, B., D. Gillies, P. Illari, F. Russo, J. Williamson. 2013. The evidence that evidence-based medicine omits. *Preventive Medicine* 57(6): 745-747.

15. Broadbent, A. 2013. *Philosophy of epidemiology*. Houndmills, UK: Palgrave Macmillan.

16. Illari, P.M., and F. Russo. 2014. *Causality: Philosophical theory meets scientific practice*. Oxford: Oxford University Press.

17. Beebee, H., C. Hitchcock, and P.C. Menzies. 2009. *The Oxford handbook of causation*. Oxford: Oxford University Press.

18. Hill, A.B. 1965. The environment and disease: Association or causation? *Proceedings of the Royal Society of Medicine* 58: 295-300.

19. Rothman, K.J. 1976. Causes*.* *American Journal of Epidemiology* 104: 87-92.

20. Rose, G. 1985. Sick individuals and sick populations. International Journal of Epidemiology 14(1): 32-38.

21. Mackie, J.L. 1965. Causes and conditions. *American Philosophical Quarterly* **2**(4): 245-264.

22. Russo, F., and J. Williamson. 2011. Generic versus single-case causality: the case of autopsy*.* *European Journal for Philosophy of Science* 1: 47-69.

23. Broadbent, A. 2011. Inferring causation in epidemiology: Mechanisms, black boxes, and contrasts. In *Causality in the sciences*, ed. P.M. Illari, F. Russo, and J. Williamson, 45-69. Oxford: Oxford University Press.

24. Illari, P.M. 2011. Mechanistic evidence: Disambiguating the Russo–Williamson thesis. *International Studies in the Philosophy of Science* 25: 139–157.

25. Claveau, F. 2012. The Russo-Williamson Theses in the social sciences: Causal inference drawing on two types of evidence*.* Studies in History and Philosophy of Biological and Biomedical Sciences 43(4): 806-813.

26. Fiorentino, A.R., and O. Dammann. 2015. Evidence, Disease, and Causation: An Epidemiologic Perspective on the Russo-Williamson Thesis*.* *Studies in History and Philosophy of Biological and Biomedical Sciences* **54**: 1-9.

27. Holland, P.W. 1986. Statistics and Causal Inference*.* *Journal of the American Statistical Association* 81(396): 945-960.

28. Rubin, D.B. 2005. Causal inference using potential outcomes: Design, modeling, decisions. *Journal of the American Statistical Association* 100(469): 322-331.

29. Hernan, M.A., and J.M. Robins. 2006. Estimating causal effects from epidemiologic data. *Journal of Epidemiology and Community Health* 60: 578-586.

30. Hernan, M.A., and J.M. Robins. 2006. Instruments for causal inference: An epidemiologist's dream? *Epidemiology* 17(4): 360-372.

31. Imbens, G., and D.B. Rubin. 2015. *Causal inference for statistics, social, and biomedical sciences: An introduction.* Cambridge: Cambridge University Press.

32. Worrall, J. 2007. Why there's no cause to randomize. *British Journal for the Philosophy of Science* 58(3): 451-488.

33. Lewis, D.K. 1986. *Philosophical papers, volume II*. New York: Oxford University Press.

34. Charlton, B.G. 1996. Attribution of causation in epidemiology: chain or mosaic? *Journal of Clinical Epidemiology* 49(1): 105-107.

35. Craver, C.F. 2007. *Explaining the brain*. Oxford: Oxford University Press.

36. Coughlin, S.S. 2014. Toward a road map for global-omics: A primer on -omic technologies. American Journal of Epidemiology 180(12): 1188-1195.

37. Schulte, P.A., and F.P. Perera. 1993. *Molecular epidemiology: Principles and practices.* San Diego: Academic Press.

1. Illari and Russo use the “mosaic” metaphor for their proposed causal pluralist mosaic (see below and their chapter 23). [↑](#footnote-ref-1)
2. In brief, the Russo-Williamson thesis suggests that causal claims in the health sciences need to be supported by evidence of difference-making *and* evidence of mechanism. This framework, which has come to be called the Russo-Williamson thesis [11, 22], has recently been discussed and criticized by philosophers [23-26]. [↑](#footnote-ref-2)
3. Details of the potential outcomes approach are far beyond the scope of this essay; they can be found in recently published concise [8] and comprehensive [31] treatments of the topic. [↑](#footnote-ref-3)
4. For an interesting alternative position, see [32]. [↑](#footnote-ref-4)
5. Craver was not the first to use the “mosaic” metaphor [33, p. ix; 34]. [↑](#footnote-ref-5)
6. If this is indeed what those who do exposomics research think they do, “exposomics” is a misnomer and should be replaced with “exposology.” If, instead, exposomics is what the term denotes, it is the idea to consider *all* lifetime exposures and their individual and joint health effects (see [36] for a concise discussion of exposomics in light of multiple other –omics approaches). [↑](#footnote-ref-6)