Jacob Stegenga: Medical Nihilism. Oxford: Oxford University Press, 2018, xiv + 227, ISBN 978-0–19–874704–8.

:: **Giovanni Sanavio**

Abstract
This review critically assesses the main claim and argument of Jacob Stegenga's book Medical Nihilism, published in 2018 by Oxford University Press. It considers some problematic aspects of the application of Bayes' theorem in support of the author's thesis, and it discusses a brief selection of unclear consequences.

Keywords
Medical Nihilism; Effectiveness; Bayes' Theorem; Book Review

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In his book *Medical Nihilism*, published in 2018 by Oxford University Press, Jacob Stegenga aims to defend the claim that "our confidence in the effectiveness of medical interventions ought to be low" (Stegenga, 2018 *Medical Nihilism*, Oxford University Press, p.11). This work is part of a wider line of research in clinical epistemology criticizing the unwarranted trust that ordinary people, patients, and healthcare professionals seem to place in the effectiveness of many clinical practices. This skeptical stance radically questions the optimism of evidence-based medicine (EBM), which assesses the quality of clinical evidence through fixed hierarchies of reliability, celebrating the use of randomized control trials (RCTs) and metaanalyses while downplaying mechanistic reasoning (see Devanesan, 2020: *Medical nihilism: the limits of a decontextualized critique of medicine*, Studies in History and Philosophy of Biological and Biomedical Sciences, 79, DOI: 10.1016/j.shpsc.2019.101189).
Through an extensive list of examples, Stegenga observes that the use and prescrip-

tion of several medical interventions, including treatments associated with severe side effects, seems to rely on unpersuasive evidence-bases. His critique targets the very core of clinical medicine, claiming that the effectiveness of medical interventions is poorly assessed whenever it is not clearly and readily appreciable, thus motivating distrust. However, it is fundamental to emphasize that his argument is neither simplistic nor anti-scientific. The author does not in fact deny the existence of some unquestionably effective medical interventions, specific for certain diseases, nor that well-conducted medical scientific research can produce beneficial results.

Stegenga acknowledges that different classes of medical interventions are associated with different degrees of effectiveness: some interventions are highly effective, others moderately effective, minimally effective, or not effective at all (Stegenga, 2018: p.184). The primary target of Stegenga's critique are thus the classes of interventions ranging from moderately effective to wholly ineffective, later identified with pharmacological interventions (i.e. administration of drugs) and related testing methodologies. This focus on drugs is plausibly motivated by the author's hybrid conception of health and disease, according to which a disease is a causally determined physical state that is also negatively valued, typically in terms of harm or discomfort. On this view, a medical intervention qualifies as effective only if it targets the causal basis of a disease or mitigates its associated discomfort. Pharmacological interventions fit this conception particularly well, as they are readily amenable to evaluation through RCTs. This is especially salient given Stegenga's insistence that assessments of effectiveness should also account for the potential harms (in the form of side effects) that can be caused by such interventions.

Measuring the effectiveness of pharmacological interventions is an operation susceptible to many biases at the level of the design of RCTs'. Stegenga proceeds identifying different stages of clinical testing which in his view may be permeable to such biases, including the selection of outcome measures, the analysis and comparison of results, and the determination of statistical procedures generalizing these results beyond the tested population (*ivi*: p.114). More broadly, he argues that trials designed to assess the effectiveness of drugs exhibit malleability, a characteristic defined by the extent to which their execution depends on discretionary decisions potentially influenced by idiosyncratic and subjective factors. Malleability is then an undesirable feature of clinical trials, as it compromises their objectivity and replicability.

The Reasoner 20(1), 2026. https://doi.org/10.54103/1757-0522/30707

Structure of the "master argument"

Towards the end of the book, Stegenga develops an argument, which he calls the "master argument", to conclude that there are no adequate epistemic grounds to support the effectiveness of most medical (i.e. pharmacological) interventions. To make his position more formally explicit, he introduces H as the hypothesis that a certain medical intervention, call it I, is effective. The probability $P(H)$ is therefore the probability that I is effective, which he interprets as a degree of belief based on all the relevant available evidence. Stegenga then introduces E as the available evidence regarding I and $P(E)$ as the degree of belief in the evidence of the effectiveness of I (*ivi*: p.168).

Motivated by the high malleability of commonly published clinical analyses, Stegenga assumes that, for most of them, the value of $P(E)$ is high while $P(H)$ is low. This means that it is generally admissible that the effectiveness of I is evident, even though it is not very plausible that I is truly effective. However, even when limiting the scope of investigation to pharmacological interventions, it cannot be taken for granted that the evidence E supporting H has a uniform and precise meaning.

The author's insistence on the pervasiveness of biases in RCTs' design stages seem to force an interpretation of E as evidence of correlation, since this is the only type of evidence that lends itself to be manipulated by personal idiosyncrasies and flawed statistical analyses. The case becomes considerably more complicated if E is interpreted as evidence of mechanism, given that RCTs motivated by this type of evidence tends to test research hypotheses on more carefully selected groups of subjects – this is to avoid excessive dilution of the observed effect on subjects who may not respond adequately to the therapy. In such cases, it is plausible to admit a low value of $P(E)$, especially if it is not known a priori on which subjects the intervention will be effective. For the low value of $P(H)$, Stegenga follows the pessimistic argument that, given that many therapies considered very effective in the past have proved useless or harmful over time, the probability that a new therapy will prove to be definitively very effective is low (here, the notions of "evidence of correlation" and "evidence of mechanism" are meant according to the account presented in Shan and Williamson, 2023: *Evidential Pluralism in the Social Sciences*, Routledge, pp. 3-30).

From these assumptions about $P(E)$ and $P(H)$ Stegenga derives that the value of the conditional probability $P(E|H)$ must also be low. This means attributing little value to the belief that the supposed truth of H contributes to make E more probable. To ensure the arithmetic consistency of this assumption, the author is forced to further clarify the meaning of E, stating that this is evidence of "a poor effec-

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tiveness of I" (Stegenga, 2018: p.176). Setting aside the legitimacy of this move, it is nevertheless possible to comply with the author and ensure that the assumption on the high value of $P(E)$ remains unchanged. At this point, it is intuitive that the conditional probability $P(H|E)$, which Stegenga calculates via Bayes' theorem, can only represent the low support of H given by E, thus formally motivating his main claim. Applying the formula:

$$P(H|E) = \frac{P(H) \cdot P(E|H)}{P(E)}$$

it is clear that the value of $P(H|E)$ must be low for elementary arithmetic reasons. In fact, it is the result of the product of two small numbers, both smaller than 1, divided by a number that is also smaller than 1 but greater than both. This is the conclusion of the "master argument".

What is promised and what is delivered

The application of Bayes' theorem seems to justify the lack of support that E provides to H. It therefore seems surprising that, given the formal clarity with which the "master argument" is set out and the arithmetic necessity of the conclusion, the nihilistic thesis is not incontrovertibly accepted by everyone. The problem emerges clearly considering the conditions under which this operation results in a low posterior probability $P(H|E)$, such as to rationally motivate distrust in the effectiveness of I. Appropriate and intuitive requirements are that effectiveness must be adequately measured, and it must be evident that the subjects included in the trial exhibit the symptoms of the disease to be treated. Failures to apply Stegenga's argument depend on the fact that the assumptions on which it is based are not always valid, and that the interpretations of the probabilities involved are not always consistent (a similar point is considered in Gillies, 2018: *Should we distrust medical interventions?* Metascience, 28, DOI: 10.1007/s11016-019-00396-z, pp. 273-276).

Given the reasons leading the author to question the effectiveness of pharmacological interventions, it is obvious that such evidence cannot support any hypothesis of effectiveness. The application of Bayes' theorem merely confirms this fact. Stegenga's point could therefore be that, limited to the classes of medical interventions he considers, the evidence reported in support of their effectiveness is in fact probabilistically greater than that of their actual effectiveness. The conclusion that the high effectiveness of I is not adequately represented by the reported evidence is obvious and there is no need of justifying it using Bayes' theorem. On the contrary, the application of the theorem results unclear.

The Reasoner 20(1), 2026. https://doi.org/10.54103/1757-0522/30707

One further limitation of Stegenga's analysis is that it fails to consider the relevance of moderately effective medical interventions and interventions that are considered to be effective even without proper assessment. In fact, although the argument is not so naive as to reduce all existing therapies to either very effective or completely ineffective, it is still possible to find counterexamples and borderline situations challenging the overall analysis. An interesting case in this sense is the evaluation of effectiveness of oncological treatments: it is difficult to define a notion of effectiveness for therapies aiming to help patients live along with chronic conditions in the absence of definitive cures. A further complication is that the conception of side effects as negative effectiveness values can contribute to underestimate the effectiveness measures of certain containment therapies. Noticeably, chemotherapy is often a potential cause of pain and physical dysfunction for patients due to its duration and intensity, but on these bases it seems irrational to conclude that it is less effective in curing cancer.

With respect to the problematic interpretation of E as evidence of mechanism, it is sufficient to observe that the quality of clinical decisions cannot obviously depend solely on statistical factors. The act of evaluating the effectiveness of a medical intervention is in many cases a complex process in which eminently clinical motivations are combined with statistical and experimental results. These considerations should be carefully considered by those who wish to endorse medical nihilism.

Giovanni Sanavio
https://orcid.org/0009-0000-3214-260X
Istituto Universitario di Studi Superiori di Pavia

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