

## **DRAFT VERSION**

### **EBM *versus* EBP?**

#### **Complexity and evidence-based practices.**

*Leen De Vreese*

#### **Abstract**

In the debate on the vices and virtues of evidence-based psychiatry the main focus of the critics lies on the differences between physical and mental diseases. I propose to interpret the limitations of the evidence-based approach not as the result of differences between physical and mental diseases as such, but as the result of different kinds of diseases on the whole. These different kinds of diseases differ in the degree of complexity in the constellation of their explanatorily relevant causes. My argument is that it is this complexity of diseases, rather than their being mental or physical, what makes it on the one hand useful, but on the other hand intricate, to apply evidence-based practices to further our knowledge about how to deal with them. The latter should force us to take a humble approach to what we can achieve using evidence-based approaches in medicine and psychiatry alike.

#### **Key Words:**

complexity, disease causation, explanatory relevance, mental disease, physical disease, pragmatism, medical practice, medical knowledge

## **1. Introduction**

During the last decades, the vices and virtues of evidence-based medicine (EBM) have widely been covered in the medical and philosophical literature. Critics have mainly focused on the epistemological presuppositions of, and methodological problems for, EBM. The vices and virtues of evidence-based psychiatry (EBP) form the subject for a largely separate debate, in which the main focus of the critics lies on the differences between physical and mental diseases. A central argument that is recurring in different forms is that the uniqueness of the individual is so important in psychiatry that evidence-based approaches cannot work as well as in somatic medicine. It is not my aim to argue here against the cogency of this claim as such (as is done explicitly by Ayob 2008), but rather to offer an alternative perspective to the discussion. I will propose to interpret the limitations of the evidence-based approach not as the result of the differences between psychiatry and medicine and its subjects as such, but as the consequence of a more basic difference in the diverging complexity of different kinds of diseases, and the related divergence in relevant causal structures.

In section 2, I give a short introduction to EBM and EBP, and comment on some recent attempts to grasp the basis for discussion on EBP. In section 3, I challenge the basic underlying idea that is guiding most of the critics in the debate on EBP, namely that psychiatric diseases are in some sense importantly different from physical diseases and that we should therefore evaluate the merits and demerits of evidence-based approaches separately for medicine and for psychiatry. Instead, I will present a pragmatic approach to ‘disease’

covering both physical and mental diseases, and argue on the basis of that approach that what matters is not whether diseases are mental or physical, but rather how complex they are. I further define the kind of complexity I am referring to as “the complexity of the *explanatorily relevant* causal structures leading to the disease”. In section 4, I show how this kind of complexity confronts us with the actual limits of our medical knowledge, primarily with respect to the most complex diseases. In section 5, I argue that, contradictorily, the methods that are favored by EBM can be very handy tools to circumvent these gaps in our knowledge on complex diseases, but that meanwhile their evidence is also the most assailable in such cases. In section 6, I come to final conclusions.

## **2. EBM: a (too) short introduction to basics and presuppositions**

In the beginning of the ‘90s, the internist Gordon Guyatt (Guyatt 1992) introduced the term “evidence-based medicine”. Since that point in time, the importance of this idea(l) has steadily grown. Currently, the idea is widespread and generally accepted as “the golden standard” in medicine, and also widely instructed as such in medical training. The best-known and most cited definition of EBM is Sackett et al.’s (1996, 71) characterization of EBM as “the conscientious, explicit and judicious use of current best evidence in making decisions about the care of individual patients”. Strauss gave the following clarification of what best research evidence is:

“(…) valid and clinically relevant research, often from the basic science of medicine, but especially from patient-centered clinical research into the accuracy and precision of diagnostic tests (including the clinical examination), the power of prognostic markers, and the

efficacy and safety of therapeutic, rehabilitative, and preventive regimens.” (Strauss et al. 2005,1; cf. Gupta 2007, 14)

These general definitions are filled in by way of an evidence hierarchy: a list of research methods ordered from those that are supposed to provide the best evidence at the top to those whose evidence is lower rated at the bottom. This list has appeared in slightly different forms in the literature, but lists the evidence roughly as follows:

- . Evidence from systematic reviews of randomized controlled trials (RCTs).
- . Evidence from single randomized controlled trials.
- . Evidence from a systematic review of observational studies.
- . Evidence from single observational studies.
- . Evidence from the basic sciences.
- . Evidence from unsystematic clinical observations.

Defenders of EBM do not reject the methods that stand at the bottom of the list, but they are convinced that one should only turn to these methods when there is no evidence available (or possible) that is based on the higher rated methods. The basic aim of the EBM approach has always been to provide the best possible care on the basis of the best available evidence. However, the introduction of the evidence hierarchy has led to a very narrow view on what good evidence in medicine consists in. Since the moment that EBM became the generally accepted paradigm within medicine, most of the attention seems to have gone to studies using the highly valued methods, at the expense of the less valued ones, which gives a lot of people the impression that - in the end - all there is in medicine to value according to the evidence-based movement are randomized controlled trials. Additionally, the gap between the theoretical ideals of EBM and their application in practice is not easily bridged, which

forms an important basis for further worries about the credibility of the evidence-based approach.

As Gupta (2007) argues, there is no official description of what evidence-based psychiatry is, but it is clear that the main idea is that EBP results from the direct application of the EBM-guidelines to psychiatry. While the proponents clearly expect the same merits of EBP for psychiatry as of EBM for medicine (namely better care on the basis of the best available evidence), opponents rely in their counterarguments primarily on the view that an evidence-based approach cannot conform to the peculiarities of psychiatry (see Gupta 2007 for an overview of arguments). Recently, more nuanced standpoints have been offered, trying to get a grip on the basis for the diverging views on EBP (e.g., Ayob 2008, Bolton 2008, Falkum 2008, Wifstad 2008). These papers focus on the inescapable limitations of evidence-based methods that result from the classical problems of the tension between the general and the individual in medical science, and between the art of medicine and the science of medicine. A basic intuition that is discussed somehow in all of these papers is that the uniqueness of the individual, and hence the role of the art of medicine rather than the science, is so important in psychiatry that evidence-based approaches cannot work as well as in somatic medicine. Hence, also these authors defending a more nuanced view towards EBP rely on the assumption that psychiatric diseases are in some sense importantly different from physical diseases and that that we should therefore evaluate the merits and demerits of evidence-based approaches separately for medicine and for psychiatry. In psychiatry, much more than in medicine, one would be confronted with the limitations of a method, such as the RCT, that searches for generalizations:

“... In this way, the problems inherent in the determination of causes and RCT

methodology in particular hit particularly hard in psychiatry, and especially in psychotherapy. The reason is simply that mental health problems, by and large, in contrast with physical health problems, by and large, do involve the person as a whole, not some subpersonal biological organ, perhaps the total, or at least a large scale life narrative, which tend toward the unique and therefore nongeneralizable.” (Bolton, 2008, p. 163)

I am convinced that there is some truth in this view. However, rather than explaining the problems for EBP in terms of differences between psychiatry and medicine and its subjects as such, I will propose to see them as the consequence of a more basic difference in the diverging complexity of different kinds of diseases in general, and the related divergence in relevant causal structures.

### **3. A pragmatic ‘disease’ concept, psychiatry and complexity**

#### *Background and introductory comments*

In this section, I will challenge the basic idea that underlies the argumentation of most of the critics in the debate on EBP. As I said in the previous section, it is largely taken for granted that psychiatric diseases are in some sense importantly different from physical diseases and that we should therefore evaluate the merits and demerits of evidence-based approaches separately for medicine and for psychiatry

<sup>1</sup>. The difference between the situation in somatic medicine and psychiatry is often described as following from the fact that in psychiatry, contrary to medicine, the evidence is applied to “people”, or to “the whole person” instead of to “bodies”. This is nonetheless a problematic simplification. In medicine, one is supposed also to be concerned with the person behind the

body, and in psychiatry, we also try to find solutions at the level of “the body”, and not (only) of “the whole person”. Additionally, there is growing evidence that psychological factors (such as stress) do play an important role in the onset of bodily diseases (see for example Grassarth-Maticcek and Eysenck 1995, Krantz and McCeney 2002, Stojanovich 2010) and it is widely recognized that at least some bodily processes have to underlie the symptoms of mental diseases. Additionally, one cannot always clearly delineate the psychological from the biological, since both are undeniably intertwined (see e.g. Sharpe 2006). Therefore, I will argue that it is better to focus on differences between different kinds of diseases in general than on differences between mental and physical diseases in particular. More precisely, I will focus on differences in the complexity of the relevant causal structures that lead to diseases and their symptoms. In my argumentation, I rely on a pragmatic approach to the disease concept, covering both mental and physical diseases in the same way, but discerning different kinds of diseases on the whole. Precisely the differences in the complexity of the explanatorily relevant causal structures of these different kinds of diseases are important for the evaluation of the usefulness of an evidence-based approach. Although I cannot go into full detail about the pragmatic approach to “disease” here (see De Vreese, to appear), I will explain the basic intuitions, in order to make its importance for the argument in this paper clear.

Some introductory comments are needed for a good understanding of the approach that follows. First, it is contradictory that most approaches to the disease concept that can be found in the literature aim for a unified approach, covering both mental and physical diseases, while in debates about other topics (such as the debate on evidence-based practices), mental and physical diseases are often taken apart. In reaction to that, I propose a pragmatic approach

that covers both mental and physical diseases (cf. De Vreese, to appear), but meanwhile offers a way to understand the differences between different kinds of diseases on the whole. Hence, my approach leads to pluralism, instead of to a unified definition of ‘disease’. However, the approach does not necessarily preclude the possibility of a unifying definition, which then abstracts from the differences between kinds of diseases. My conviction is nonetheless that we can learn much more from a pragmatic, pluralist approach which recognizes the differences, for example when thinking about the vices and virtues of evidence-based practices (as will become clear from the remainder of this paper).

My proposal should further be read as an explication of the different ways in which we can understand the notion of ‘disease’, starting from the meaning of the notion in its broadest sense. This is in the first place a descriptive endeavor, which does not have direct normative implications. It cannot simply be used as a justification for one or another “genuine” interpretation of the notion ‘disease’. The approach should rather be seen as a first basis for reflection about, amongst others, the justification of our use of the term ‘disease’ in specific cases. To fully appreciate what the approach has to offer, readers will also have to distance themselves from their intuitive interpretation of the notion, which might, for example, be narrower, excluding some of the disease kinds discerned below.

### *Pragmatic approach*

The pragmatic approach starts from the conviction that ‘disease’ is not a strict, theoretical concept, but a *practical* term. Our use of this term is determined by two *interacting* factors: (A) value-laden considerations about the (un)desirability of certain physiological and/or psychological states and (B) discoveries of a/the bodily and/or psychological cause(s) which

is/are explanatorily relevant in view of possible medical interventions that can prevent, cure, or at least improve undesired states. Let me explain the import of these two aspects further.

*A. Undesirable states:* We cannot start assembling facts about diseases and disease causation if we do not have at least a basic idea of the bodily and psychological states we are talking about when using the word ‘disease’. Certain states are conceived of as eligible for being disease states on the basis that we disvalue them because they deviate from certain idealized states of our body and mind. This will be based on comparison between the various bodily and mental states that we encounter in general. Further, for considering a state as an eligible ‘disease’ state a consensus is needed on the disvalue of this state and the kind of state that should replace it (which excludes purely personal preferences as a basis for calling certain states ‘diseases’, since in that case there is no consensus on which kind of state is to be preferred, think for example about being tall or small, having bigger or smaller breasts, being a realist or an idealist). If medical science finds relevant difference-makers that explain the differences in the outcome (cf. (B) below), “eligible” diseases will be recognized as “genuine” diseases that should ideally be preventable or curable by medical means, which then becomes the goal that medicine sets itself. It is very important here to see that it is not nature but we ourselves who make the basic distinction between desirable and undesirable states that might need medical attention, on the basis of value-laden criteria. However, this does not alter the fact that there will be some real differences in causal processes underlying these distinctions. Medicine investigates these underlying causal facts (cf. Miller Brown 1985), and decides whether they are of a kind that can justify a ‘disease’ label. This brings us to the B part of my analysis.

*B. Disease causation:* It is not the goal of medicine to give the full and complex

description of the whole causal setting that leads up to a disvalued state. Medicine is a practical science focusing primarily on manipulable causes (cf. Kincaid 2008). This means that medicine makes selections of *relevant* causal factors instead of focusing on the whole causal history leading to diseases. Hence, whether (a) certain cause(s) should be selected as *the* difference-making cause(s) between the desired and the undesired state is decided on the basis of their *explanatory relevance for medicine*. It is important here to notice that I speak in terms of the “explanatory relevance” of causes, since in any case, a lot of causes will be involved, but this does not mean that we have to know and cite them all. Only those that are relevant in view of the medical goal of explaining and intervening will attract the attention of medical science. For example, while psychological factors might be important for our susceptibility to certain physical diseases, we do not always need to know the details of how these psychological factors play a role in order to be able to explain and treat these diseases.<sup>2</sup> We can often solve the problem at the physical level and also know often well how to do that, which makes it unnecessary to know the details about the causal influence of the psychological factors. For most mental diseases, to the contrary, it seems much less evident to deny the psychological factors that might have caused it in order to intervene adequately. Unfortunately, we more often lack the knowledge about how to solve mental problems and how to directly intervene in their causes.

To sum up: on the one hand, we have our considerations about desirable and undesirable states; on the other hand, we have medical knowledge about the relevant, underlying causes that lead to certain states. However, these two factors do not stand apart, and are also not just two conditions that should simply both be fulfilled to ascertain a ‘disease’ state. It is crucial to see that what matters is the ongoing interaction between these

two factors. Finding explanatorily relevant difference-makers for an eligible ‘disease’ state, which make it possible medically to intervene, can form an important confirmation of the ‘disease’ status of a state. Suppose, for example, that a clear biological cause would be found that could explain chronic fatigue syndrome (CFS). This would make it possible clearly to discern patients suffering from CFS and those whose fatigue cannot be explained as a symptom of CFS. Further, such a finding could help us in finding an adequate treatment for the disease. And by consequence, the disease status of CFS would become much less disputable. On the other hand, a lack of clarity on explanatorily relevant difference-makers for an eligible disease state will heighten doubt on the aptness of the disease label.

### *Different kinds of ‘disease’*

Concerning both factors, we might be unclear. We can hesitate on whether we should consider certain deviances as eligible diseases (think for example about alcoholism), and we can also have limited scientific underpinnings for our value-considerations (think for example about ADHD). There will always be different extents to which A (the value considerations leading to an eligible ‘disease’ state) fits B (the scientific knowledge concerning the relevant cause(s) of this state). This leads to different kinds of diseases according to the kind of causal explanation that can be given. Nick Haslam (2002) proposed a very interesting classification of psychiatric diseases which fits in nicely with this idea, and which to my mind can be generalized to diseases in general. I do not claim that his list is accurate or exhaustive, but I am nonetheless convinced that it is at least a good first approximation of the different meanings of ‘disease’. For a thorough understanding of what Haslam’s classification is all about, I would like to refer the reader to the original paper of Haslam (2002). I try to

summarize the basics here.

Haslam presents five kinds of disease categories:

1. *the non-kind category*: concerns characteristics that are normally distributed among people and according to which people differ only by degree. Any binary distinction between diseased and non-diseased people in this category is arbitrary. Haslam (2002) argues that ‘diseases’ in this category are no real diseases. Examples are neuroticism (Haslam 2002) and flap-ears.

2. *the practical kind category*: this category only differs from the first one in that a pragmatic distinction for diagnosis is imposed, as justified by scientific findings, for example in the case of high blood pressure where the medical community agreed on some thresholds for medical intervention in view of long-term risks. This pragmatic decision justifies the disease label.

3. *the fuzzy kind category*: with respect to any disease in the fuzzy kind category, one can discern three groups of people: the unambiguous group of non-diseased people, the unambiguous group of diseased people, and an ambiguous group of people in between for which the disease status is not clear. Arteriosclerosis is an example of a physical disease in this category: children almost never suffer from arteriosclerosis, old people often clearly suffer from the disease, and then there is a group of (middle-aged) people who have a (very) mild form of the disease but do not suffer from any harmful effects.

4. *the discrete kind category*: this category covers diseases for which membership is dichotomous (hence, one is clearly diseased, or clearly not diseased), but is not definable on the basis of a single, causally determining essential property. For example, autoimmune diseases.

5. *the natural kind category*: in this category, one finds these diseases for which membership is dichotomous and definable on the basis of an essential, specific cause. The clearest

examples are infectious diseases. But also cancer is an example: suffering from malignant out-of-control cell growth is the defining cause here.

The diseases in the different categories are supposed to result from different constellations of relevant causes. In what I call the “continuous kinds” (i.e. category 1 and 2), and to a lesser extent also in the first of the “non-continuous kinds” (i.e. category 3), the diseases seem to result from a complicated constellation of causal influences. Small differences in the causes should then be known to be able to explain the (gradual) differences in the outcomes. In other words, to understand the differences on the basis of which to intervene in the development of these diseases, one will need the knowledge of a big amount of relevant causes and their interactions. This is not the case for the disease kinds that stand at the bottom of the above list. They can be explained by reference to a limited amount of relevant causes, or even a single one. Where to classify a specific disease, always depends on our current knowledge. Diseases can thus be reclassified on the basis of growing knowledge. In the ideal case, all diseases will once be explained by reference to a limited amount of clear causes that can be intervened on. However, it is also possible that some ‘diseases’ are just the result of very complex interactions and can never be explained by reference to a limited amount of relevant causes.

I relate all this to what I label the “complexity” of diseases

<sup>3</sup>. The more complex the amount and interaction of the explanatorily relevant causal factors, the more complex the disease is. It is true that mental diseases are, according to our current knowledge, more often of a complex kind. However, as Haslam (2002) showed, not all of them are. According to a pragmatic approach along these lines, what makes the difference is not whether the disease is mental or physical, but the complexity of the relevant causal

structures leading to the disease. There are physical and mental diseases of all kinds and both “mental” and “physical” diseases are probably always influenced (though, not necessarily relevantly) by “mental” and “physical” aspects of certain kinds. All this makes the intuitive distinction between “mental” and “physical” diseases arbitrary (see also Brülde & Radovic 2006) and unconvincing as an argument against the usefulness of EBP.

#### **4. Complexity and our limited knowledge**

It is true that a lot of mental diseases confront us with the challenges of complexity. However, complexity should not be understood as a peculiar feature of mental disease. Some authors (e.g. Maier 2006) use the complexity of humans in a very general sense as an argument against EBP (presupposing, again, some kind of uniqueness to follow from this). Mental diseases are then portrayed as being especially difficult to handle because of their unpredictability. However, it is not clear how this argument of general complexity of the human constitution justifies the demarcation between mental and physical diseases and their (un)predictability, since complexity in this general sense is always involved, even in the development of diseases that we understand well. What one should do is try to specify why this complexity is a bigger problem in some cases than in others. My argument is precisely that this should be explained in terms of the complexity of *explanatorily relevant* causal structures, and has nothing to do with the differences between mental and physical diseases as such. That we should more often turn to the art of medicine than to the science of medicine in psychiatry, seems to me the result of this complexity, and is not especially related with the special features of a mental disease. Also the problem of generalization can better be seen as

the result of this complexity, rather than as a special feature of psychiatry. Further, it is important to notice that complexity as I define it, does not necessarily entail uniqueness, the latter forming the basis for some authors to argue against the possibility of EBP. The important point here is that it is not because a disease is caused in a complex way, that one should presuppose some kind of uniqueness to be at work, making us unable ever to come to grips with the (gradual differences in the) complex interactions of causal influences and their effects, in such a way that this knowledge would be generalizable enough to understand (and intervene in) different instances. However, having this knowledge at our disposal would clearly be an ideal situation. The current, factual situation is that we do not (yet) have enough knowledge of a lot of complex diseases to even start developing very precise, directed interventions into their causes. In other words, complex diseases confront us with the limits of our knowledge about the complex interactions that are possible in, and between, our mind and body, and that result in a myriad of possible bodily and mental conditions.

Moreover, complexity can play at different levels: at the level of the proximate causes of the disease as such (which forms the basis for the classification of disease kinds above), at the level of the more distant causes (e.g. the causal influence of the risk factors), and at the level of the possible interventions in the symptoms of the disease. Even if one has a thorough understanding of the proximate causation of a disease, it remains a question whether medical practitioners really *can* directly intervene in the proximate causes. This will not only depend on the complexity as such, but also on, amongst others, the stance of the medical know-how. If an intervention in the proximate causes of the disease is impossible (e.g. when we cannot stop the out-of-control cell growth in the case of cancer), medicine can turn to the more distant causes (which will nonetheless often offer only preventive measures and no adequate

treatment options), or to the symptoms (to relieve the effects of the disease rather than to treat the disease itself). In part of the cases, we have good knowledge of how we can accurately influence the causal processes involved in the symptoms (e.g. when using painkillers to relieve the pain that follows from a disease). In other (and often more complex) cases, neither the difference-making causes nor the exact way in which we can intervene in the symptoms are well known. This is for example the case when we use stimulating drugs to intervene in ADHD, or when using blood-pressure lowering drugs to treat migraine. We should however be careful. The (accidental) finding that certain treatments work to change certain disvalued states, can lead to the unjustified conviction that we are intervening at the level of the complex causation of the disease as such, which medicine then should be able to spell out more clearly in the future. This way of reasoning can be flawed, and can give rise to medicalization on the basis of what is sometimes called the ‘treatability fallacy’: “if we are able to treat it, than there should be a defining cause which confirms the disease status of the condition.” However, we hereby forget that what we are “treating” might not be the disease as such, but just its symptoms, or even worse, the “symptoms” of a state that we have labeled “disease” on unclear grounds. That such treatments might further have important, and possibly unrecognized, side effects - which might be even worse than the initial “disease” itself - is hereby an important further worry.

To conclude, complex diseases confront us with the limits of our actual medical knowledge. One can wonder whether we will ever be able to get the same level of understanding of the complex diseases as we now have of a lot of less complex diseases. We can imagine that we will indeed be able to find “simple” explanations for some “complex” conditions in the future (transforming them in less complex diseases), such as was the case for

Huntington disease. However, it is not unimaginable that there will always be conditions left that are too complex for us to get a detailed understanding of. Fine tuning our causal knowledge in medicine to that extent that we come to grips with the causation of all possible diseases, might be just too difficult to attain by our human knowledge. It is up to the future to decide on this. However, what is sure is that we currently miss a lot of knowledge to have such deep understanding of our bodies and minds. And that is precisely where the methods that are favored by evidence-based approaches seem to come in handy.

## **5. Complexity, evidence-based approaches and practice**

The question that follows for complex diseases<sup>4</sup> is how to deal with our lack of knowledge about their causation. The methods that are favored by EBM can be very handy tools to circumvent these gaps in our knowledge. This is precisely their strength: they can offer useful causal knowledge where deeper insights in the underlying causal mechanisms are missing. In that sense, we can potentially learn a lot from these methods. However, this is no reason for medicine to canonize the methods that are highly valued by EBM. While these methods can be very helpful and useful, it is just as much important that their evidence is also very assailable. The literature on EBM has plenty of critical comments on the limitations of this approach. They show that we should also be humble and careful when using these methods and when applying their evidence. Ironically, the more complex the disease state we try to understand, and hence, the more we seem to be in need of methods that can circumvent our knowledge gaps, the more assailable the evidence that results from these methods will be. In other words, where we are most in need of the highly valued methods of EBM/EBP, they also

seem the least reliable. On the other hand, as Mitra critically argues, where the situation is clear, RCTs are not needed either:

“(...) surely, we do not expect the RCT to deliver a therapeutic breakthrough by itself, in the same way that we would not expect a weighing machine, no matter how sophisticated, to deliver a miraculous drug that will abolish obesity. It is equally clear that if indeed there were to be a revolutionary drug or intervention, the RCT would be neither ethically permissible nor required. The very fact that so many thousands of RCTs are being conducted each year is itself a testimony that medical advances of today, if any, are destined to be of doubtful or marginal clinical utility.” (Mitra, 2009, pp. 504-505).

To sum up, the less complex the causation of a disease, the more apt the use of methods that search for generalizations in the absence of underlying causal knowledge will be. After all, the problem with RCTs is precisely the knowledge base from which we generalize - namely one that potentially lacks in much important knowledge - rather than the generalization as such. But meanwhile, the clearer our grip on a certain bodily or mental state becomes, the less we might be in need for such methods, because it then might be clear enough from our theoretical insights in what sense and to what extent the evidence can be generalized. This brings us in a contradictory situation that cannot easily be escaped. Additionally, while RCTs bring us some knowledge, the impact of the findings is often limited. A lot of the RCTs that are currently carried out only detect small differences and/or only answer very specific questions, that moreover primarily focus on the effects of interventions such as drug treatment, rather than on the search for causal explanations. Although we cannot know to what extent we will ever be able to get a grip on complex diseases, it is clear that we should not expect that too much of the pioneering knowledge will

come from RCTs (cf. Mitra 2009). Therefore, the reflex to promote even more RCTs in response to our limited knowledge about complex diseases (cf. Ayob 2008), seems to be shortsighted. Instead, we should value each method that might contribute to the progress of medicine equally, and meanwhile we should recognize the limits of all of them equally. Basic research into causal mechanisms, for example, might be very necessary for medical discoveries regarding diseases, their causes, and their treatments (see also De Vreese 2008) and should therefore not be pushed into the background, although the long-term benefits from such research is also not predictable beforehand. As Mitra continues:

“This is not to say that the RCT itself is at fault or that it should no longer be practiced. (...) The problem is the high value that we place on it, such that the RCT has become synonymous with clinical research in general, as if it is the only form of clinical enquiry.” (Mitra, 2009, pp. 504-505)

Finally, the pressure of practice in medicine is not to be underestimated in the EBM/EBP story. Society currently has high expectations of medical practice. This is partly due to how medicine has presented itself in the past decades. However, although medicine has made tremendous improvements in the last century, it clearly has no solutions for all human health threats. That a lot of attention goes to evidence-based methods can also be understood from this perspective. These “black box” methods give us some certainties where there would otherwise only be uncertainties. Their evidence clearly helps practitioners, who feel obliged to do at least something about the disease states that they are confronted with in practice:

“(…), I feel more confident in my medical decisions when I can say “the data show this” or “the data show that”. Even when I have to say “there are no data for this,” I feel my decision is more valid. (Padrino, 2002).

Although this sense of certainty is a clear merit for medical practitioners struggling with specific questions, we should be careful that such feelings do not result in a false, general sense of certainty behind which we hide. Further, medical science should critically think about its general objectives when preferring certain methods (cf. Nordin 1999). If the primary goal is to do pioneering work leading to interesting medical progress, the focus on evidence-based approaches might be misleading for two reasons. First, it might overshoot itself by putting too much effort in gaining limited, fragmentary knowledge. Secondly, it does this at the expense of other methods, which leads to underestimations of the (possible) usefulness of their evidence.

However, we might currently have reached some limits, and in anticipation of further important findings, the only way to make some progress, or at least to be as informed as possible, might be by means of the favored EBM methods. It is after all not too difficult to criticize the shortcomings of these methods, but it is much more difficult to offer alternatives to accurately study, in the first place, complex diseases. The only alternative we have, in general, is to value all methods that have proven their usefulness for medical science in the past, to recognize their limits and try to ameliorate them, and to bring their evidence together in our further search for knowledge. In brief: we should learn to live with the limits of our methods and our knowledge. Recognizing this will teach us to value RCTs as one of the possible methods, rather than the preferable one.

## **6. Conclusion**

In this paper, I challenged a basic presupposition that most of the participants in the debate

about EBP share, namely that psychiatric diseases are in some sense importantly different from physical diseases and that we should therefore evaluate the merits and demerits of evidence-based approaches separately for medicine and for psychiatry. Instead, I argued that it is not in the first place being mental or being physical which makes the difference for weighing the usefulness of evidence-based methods, but rather the degree of complexity. However, since complexity in general is involved in any instance of disease, it is necessary to specify why complexity is a bigger problem in some cases than in others. My argument is that this should be explained in terms of the complexity of the *relevant* causal structures of a disease in view of explanation and intervention. This approach offers another perspective on the basis of which we can understand opposite claims about what we can achieve by means of evidence-based practices, both in medicine and psychiatry. The methods that are favored by the evidence-based approach might be the most appropriate to get some grip on complex diseases and possible interventions. However, the complexity of these diseases also makes the evidence that results from these methods very assailable. It is true that mental diseases are, according to our current knowledge, more often of a complex kind and therefore form a bigger challenge. However, focusing on the differences between mental and physical diseases alone leads to a misguided discussion about the usefulness of EBP.

## **Endnotes**

<sup>1</sup> Except maybe for Tanenbaum (2005), who explicitly argues that the controversies she describes for evidence-based practices in mental health care also pertain to EBM.

<sup>2</sup> I do not argue here that it is not of any interest at all to the medical sciences in general also to study the role of psychological and social causes in the development of physical diseases. As I already pointed out, there is growing evidence that psychological causes play a role in the onset of physical diseases. However, such causes will often not be the first to be cited in answers to explanation-seeking questions that have the treatment and intervention of physical diseases in individuals as their epistemic interest (see also De Vreese et al. 2010). To what extent it is really the task of medicine to study and intervene in more distant psychological causes, is further debatable and depends on how one demarcates the medical enterprise (cf. Nordin 1999). All this is thus also a matter of the relevance of certain causes given certain interests, goals and means.

<sup>3</sup> I use “complexity” and “complex diseases” as shorthand to respectively “complexity of explanatorily relevant causal structures” and “diseases resulting from a complex constellation of explanatorily relevant causal influences.”

<sup>4</sup> The same holds to a certain extent for less complex diseases for which we nonetheless need insights in the more complex causal role of more distant causes (e.g. the causes of out-of-control cell growth in different forms of cancer). The difference here is that we nonetheless have a good grip of the target, which is the proximate cause that defines the disease. Such a clear causal target is missing in the continuous disease kinds.

## **References**

Ayob, G. (2008). Do people defy generalizations? Examining the case against evidence-based medicine in psychiatry. *Philosophy, Psychiatry & Psychology* 15(2): 167-174.

- Brülde, B. and F. Radovic. (2006). What is mental about mental disorder? *Philosophy, Psychiatry, & Psychology*, 13(2): 99-116.
- Bolton, D. (2008). The epistemology of randomized, controlled trials and application in psychiatry. *Philosophy, Psychiatry & Psychology* 15(2): 159-165.
- De Vreese, L., E. Weber and J. Van Bouwel. 2010. Explanatory pluralism in the medical sciences: theory and practice. *Theoretical Medicine and Bioethics*, 31: 371-390.
- De Vreese, L. 2008. Causal (mis)understanding and the search for scientific explanations: a case study from the history of medicine. *Studies in History and Philosophy of Biological and Biomedical Sciences*, 39: 14-24.
- De Vreese, L. (to appear). Rethinking the concept of disease debate: a pragmatist alternative.
- Falkum, E. 2008. Phronesis and techne: the debate on evidence-based medicine in psychiatry and psychotherapy. *Philosophy, Psychiatry & Psychology* 15, no. 2: 141-149.
- Grossart-Maticek, R. and H. J. Eysenck. 1995. Self-Regulation and mortality from cancer, coronary heart disease, and other causes: a prospective study. *Personality and Individual Differences*, 19, no. 6: 781-795.
- Gupta, M. 2007. Does evidence-based medicine apply to psychiatry? *Theoretical Medicine and Bioethics*, 28: 103-120.
- Guyatt, G., J. Cairns, D. Churchill, D. Cook, B. Haynes, J. Hirsh, J. Irvine, M. Levine, M. Levine, J. Nishikawa, D. Sackett, P. Brill-Edwards, H. Gerstein, J. Gibson, R. Jaeschke, A. Kerigan, A. Neville, A. Panju, A. Detsky, M. Enkin, P. Frid, M. Gerrity, A. Laupacis, V. Lawrence, J. Menard, V. Moyer, C. Mulrow, P. Links, A. Oxman, J. Sinclair, P. Tugwell. 1992. Evidence-based medicine. A new approach to teaching the practice of medicine. *The*

*Journal of the American Medical Association*, 268:2420-2425.

Haslam, N. 2002. Kinds of kinds: a conceptual taxonomy of psychiatric categories, *Philosophy, Psychiatry & Psychology*, 9, no. 3: 203-217.

Kincaid, H. 2008. Do We Need Theory to Study Disease? Lessons from cancer research and their implications for mental illness, *Perspectives in Biology and Medicine*, 51, no. 3: 367-378.

Krantz, D. S. and M. K. McCeney. 2002. Effects of psychological and social factors on organic disease: a critical assessment of research on coronary heart disease. *Annual Review of Psychology*, 53: 341-369.

Maier, T. 2006. Evidence-based psychiatry: understanding the limitations of a method. *Journal of Evaluation in Clinical Practice*, 12, no. 3: 325-329.

Miller Brown, W. (1985), On defining 'disease'. *The Journal of Medicine and Philosophy*, 10, no. 4: 311-328.

Mitra, I. 2009. Why is modern medicine stuck in a rut? *Perspectives in Biology and Medicine*, 52, no. 4: 500-517.

Nordin, I. 1999. The limits of medical practice. *Theoretical Medicine and Bioethics*, 20, no. 2: 105-123.

Padrino, S. 2002. Evidence-based psychiatry: fad or fundamental? *Psychiatric news* 37, no. 16: 13.

Sackett D. L., W. M. C. Rosenberg, J. A. Muir Gray J.A., R. B. Haynes and W S. Richardson. 1996. Evidence-based medicine: what it is and what it isn't. *British Medical Journal*, 312: 71-72.

- Sharpe, M. 2006. Bodily symptoms: a new approach. *Psychiatry*, 5, no. 2: 39-42.
- Stojanovich, L. 2010. Stress and autoimmunity. *Autoimmunity Reviews* 9, no. 5: A271-A276.
- Strauss S. E., W. S. Richardson, P. Glasziou and R. B. Haynes. 2005. *Evidence-based medicine: How to Practice and Teach EBM*, 3rd edition, Philadelphia: Elsevier Churchill Livingstone.
- Tanenbaum, S. J. 2005. Evidence-based practice as mental health policy: three controversies and a caveat. *Health Affairs* 24, no. 1: 163-173.
- Wifstad, A. 2008. External and internal evidence in clinical judgment: the evidence-based medicine attitude. *Philosophy, Psychiatry & Psychology* 15, no. 2: 135-139.