Narrowing the gap between ICD/DSM and RDoC constructs: possible steps and caveats

One century ago K. Jaspers, discussing the status of research on the neural correlates of mental disorders, stated that “we only know the end links in the chain of causation from soma to psyche and vice versa, and from both these terminal points we endeavour to advance”¹.

There has certainly been some progress from Jaspers’ time. We know today some more links in that chain of causation, on one side and on the other. But the gap does remain and we – clinicians and psychopathologists on one side and neuroscientists on the other – do have to advance from our respective terminal points in order to narrow it.

Indeed, the recent debate following the publication of the DSM-5 and the launch of the Research Domain Criteria (RDoC) project has made it clear that we have today on the one hand a problem with several ICD/DSM constructs, in that they are too distant from the level of inquiry of neurosciences, and on the other a problem with at least part of RDoC constructs, in that they are somewhat distant from the level of “the actual clinical phenomena that bring patients to the clinic”². This gap seems to be particularly sensible in the area of psychoses: according to B. Cuthbert, “one hears informal comments at conferences that psychosis is a ‘black box’ in RDoC”².

How can we, clinicians and psychopathologists, from our side of the chain of causation Jaspers was referring to, contribute to narrow this gap?

Actually, after an initial phase in which the RDoC project was presented (or perceived) as being in competition with current diagnostic systems, a dialogue has started between the US National Institute of Mental Health (NIMH) and both the World Health Organization (WHO) and the American Psychiatric Association (APA) (see also Sanislow³ in this issue of the journal). Particularly productive in this respect have been the WHO/NIMH meeting held in Madrid in February 2014 entitled “Future directions in research diagnostic criteria for mental and behavioural disorders” and the APA/NIMH symposium that took place in Atlanta in May 2016 entitled “DSM-5 and RDoC: moving towards a common agenda for understanding mental disorders”.

Having been the chairperson in the Madrid meeting and the discussant in the Atlanta symposium, I would like to share here with the readers of *World Psychiatry*: a) a list of the possible steps, emerging from those meetings, that we clinicians and psychopathologists can implement in order to contribute to narrow the gap between ICD/DSM and RDoC constructs, and b) a list of caveats that we will have to take into account, because not all of the assumptions which are at the basis of the RDoC project can be fully endorsed by us at the current state of development of our discipline.

A first possible step that we clinicians and psychopathologists can implement in order to narrow the above-mentioned gap is a redefinition and dissection of some complex symptoms

and signs. Indeed, while the characterization of psychiatric syndromes has been repeatedly refined in the past four decades, that of psychiatric symptoms and signs has remained more or less the same, with the result that several symptoms, especially composite and heterogeneous ones (e.g., delusions, hallucinations, anhedonia), are characterized in the DSM-5 glossary in a way that is somewhat outdated and too far from the level of neuroscientific inquiry.

A second possible step is the identification of experiential intermediate phenotypes which can be added to those, mostly behavioural, that are included in the RDoC framework. Primary psychotic experiences – of which aberrant salience, in part corresponding to Jaspers’ delusional atmosphere, represents a good example – may indeed be a more reasonable and meaningful target for neuroscientific inquiry than, say, delusional ideas. Of course, these primary psychotic experiences will have to be characterized in a way that is clear and reliable, and a good example of how this can be done is given by the instrument called Examination of Anomalous Self-Experience (EASE)⁴, developed by J. Parnas and some other European psychopathologists.

A third possible step is a refinement of currently identified dimensions of mental disorder and the delineation of crosswalks between some of those dimensions and some RDoC constructs. This is the aim of the effort that P. Wang and D. Clarke are conducting within the APA⁵, which has been described at the above-mentioned Atlanta symposium.

A fourth possible step is a more precise and detailed characterization of broader dimensional groupings or spectra, such as internalizing and externalizing, but also neuroticism. The research conducted by R. Krueger and his group on these spectra and their neurobiological correlates, also illustrated at the Atlanta symposium, and their recent effort to explore possible interrelationships between those spectra and some RDoC constructs⁶, represent a good example of how this strategy can be productively pursued.

A fifth possible step is the refinement of recent attempts to delineate several stages in the development of some mental disorders, again particularly in the area of psychoses⁷. Some of those stages, especially early ones, may be closer to the level of neuroscientific inquiry than the full-blown syndromes described in the ICD and DSM.

A sixth possible step is an in-depth exploration of the dynamics within networks of symptoms. Recent research in this area, in fact, has suggested that reciprocal interactions may exist between the symptoms of a mental disorder, so that an adverse event may trigger one or more specific symptoms, which may in turn activate other symptoms, that may then modulate the expression of the former⁸. These dynamics can be relevant to neuroscientific inquiry.

This is, of course, a very tentative list, that will need to be extensively refined and to which further elements can be added.

Let's come now to the conceptual caveats, all emerging from the recent rich literature in the field of philosophy of psychiatry. Incidentally, according to T. Kuhn⁹, the "recourse to philosophy and to debate over fundamentals" is one of the symptoms of the "transition from normal to extraordinary science" which characterizes paradigm shifts within a given discipline. There is no doubt that this symptom is quite evident in the current phase of development of our discipline.

The first caveat is that there are several different levels of observation and explanation of abnormal mental phenomena, and there is no *a priori* reason why one of these levels should be regarded as more fundamental than the others¹⁰. Of course, all abnormal experiences and behaviours are likely to be implemented through neural circuits, but this does not mean that the level of neural circuits will necessarily be the most useful and efficient at which to observe and explain those experiences and behaviours. What is the most useful and efficient level will depend upon the purpose for which the observation and explanation is required. If our purpose is to develop new psychotropic drugs, the level of neural circuits is likely to be the most efficient, but if our purpose is to develop new psychotherapies or psychosocial interventions, other levels of observation and explanation may be more useful and efficient.

A second, related but more radical, caveat is that, although all abnormal mental phenomena are likely to be implemented through neural circuits, this does not mean that those circuits will have to be necessarily themselves "faulty", and in need to be "fixed". Some forms of mental dysfunction may involve maladaptive operating rules acquired by learning¹¹, which may be subtended by a reconfiguration of neural activity that is not in itself "dysfunctional", although different from ordinary patterns. In other terms, the level of the dysfunction may be higher than that of neural circuits, and intervening on those circuits may not be an adequate way to act on that dysfunction.

The third caveat was already voiced by K. Jaspers one century ago: "the method of living mosaic – i.e., the idea that disease entities are mosaic-like structures composed from a variety of individual and identical pieces – turns psychopathological investigation and diagnosis into something mechanical and petrifies discovery"¹. In other terms, whether it is really possible to decompose currently identified mental disorders into "pieces" (variables or dimensions), which recur exactly with the same characteristics and presumably with the same neurobiological correlates in all those disorders, remains to be proved. A given symptom may instead have a different meaning and different underlying pathogenetic processes depending on the overall psychopathological context within which it emerges.

A fourth caveat is that, while the problematic issue of test-retest and inter-rater reliability of psychopathological measures has been repeatedly emphasized in the past few years, the possibly even more problematic issue of test-retest and inter-laboratory reliability of neurobiological measures in psychiatry has not been equally emphasized. This is a problem to be taken into account, if the aim is to develop measures to be used in ordinary clinical practice.

On the basis of all this, what tentative conclusions can be drawn, concerning both clinical practice and progress of knowledge?

As to clinical practice, it will be empirical evidence to show whether, or to which extent, the characterization of individual patients in terms of behavioural and neurobiological variables can augment (or, as originally proposed in the RDoC project, even replace) our current psychopathological characterization in the pursuit of what remains our main objective, i.e., the efficient prediction of outcomes, in particular response to treatments. One thing are promises and statements; another thing are facts and data. What we badly need today is the latter: an empirical evidence which is solid, convincing, widely replicated and clinically relevant. The history of biological psychiatry has not started yesterday. We have seen so many biological findings which have been just forgotten after some years, without being either confirmed or disproved, and which have never been even considered for application in ordinary practice.

As to progress of knowledge, clinicians and psychopathologists on one side and neuroscientists on the other are still "exploring from opposite directions", as Jaspers was pointing out, a continent which is largely unknown¹. Neither team can state that its effort is *a priori* more fundamental, worthwhile or scientific than the other, or claim present or future complete control over the territory. The best service we can do to our profession and our patients is to work actively to advance from our respective terminal points in the chain of causation, in a spirit of collaboration and mutual respect.

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