

# PHILOSOPHY OF CLINICAL PSYCHOPHARMACOLOGY

Massimiliano Aragona

Association Crossing Dialogues, Rome, Italy

received: 10.10.2012;

revised: 28.11.2012;

accepted: 28.12.2012

## SUMMARY

*The renewal of the philosophical debate in psychiatry is one exciting news of recent years. However, its use in psychopharmacology may be problematic, ranging from self-confinement into the realm of values (which leaves the evidence-based domain unchallenged) to complete rejection of scientific evidence. In this paper philosophy is conceived as a conceptual audit of clinical psychopharmacology. Its function is to criticise the epistemological and methodological problems of current neopositivist, ingenuously realist and evidence-servant psychiatry from within the scientific stance and with the aim of aiding psychopharmacologists in practicing a more self-aware, critical and possibly useful clinical practice.*

*Three examples are discussed to suggest that psychopharmacological practice needs conceptual clarification. At the diagnostic level it is shown that the crisis of the current diagnostic system and the problem of comorbidity strongly influence psychopharmacological results, new conceptualizations more respondent to the psychopharmacological requirements being needed. Heterogeneity of research samples, lack of specificity of psychotropic drugs, difficult generalizability of results, need of a phenomenological study of drug-induced psychopathological changes are discussed herein. At the methodological level the merits and limits of evidence-based practice are considered, arguing that clinicians should know the best available evidence but that guidelines should not be constrictive (due to several methodological biases and rhetorical tricks of which the clinician should be aware, sometimes respondent to extra-scientific, economical requests). At the epistemological level it is shown that the clinical stance is shaped by implicit philosophical beliefs about the mind/body problem (reductionism, dualism, interactionism, pragmatism), and that philosophy can aid physicians to be more aware of their beliefs in order to choose the most useful view and to practice coherently. In conclusion, psychopharmacologists already use methodological audit (e.g. statistical audit); similarly, conceptual clarification is needed in both research planning/evaluation and everyday psychopharmacological practice.*

**Key words:** *philosophy – epistemology – psychopharmacology - evidence-based psychiatry – pragmatism - conceptual analysis*

\* \* \* \* \*

## Introduction

The new philosophy of psychiatry has raised increasing interest in recent years, paralleling the concurrent crisis of the neokraepelinian view of mental disorders, sustained by the so called biological psychiatry (Aragona 2009a). Philosophy of psychiatry was presented as the third revolution in late Twentieth Century psychiatry, its roots being on both phenomenology and analytic philosophy, and proposing a values-based approach. The last is conceived as a direct counterpart for values of the set of practical tools provided by evidence-based practice to support clinical decision-making where complex and conflicting evidence is involved (Fulford & Stanghellini 2008). Although naturally involved in clinical practice, the philosophy of psychiatry risks to be viewed by many only as the humanistic part of our discipline, mainly involved with concerns about personal meanings and values. As a consequence, philosophical inquiry is sometimes perceived as detached from more basic, somatically oriented matters focusing on neurobiological processes and psychopharmacological modulation. In this case, a sharp division between evidence-based and values-based psychiatry risks being misleading, covertly suggesting that evidence is the province of mere facts. Indeed, the logical consequence would be that philosophy has nothing to say to the

clinical psychopharmacologist and that empirical research is the only authorized research in this field. More recently an alternative view tried to integrate “perennial conceptual tensions in the field (objectivity/subjectivity, explanation/understanding, absolutism/relativism)” (Stein 2012), arguing for an integrative approach which should answer the questions raised by the “successes” of modern psychopharmacology: i.e., conceptual, explanatory and moral questions about psychotropics (Stein 2008). This is a naturalistic approach based on the idea that although categories of mental disorders can be shaped by both medical and moral metaphors (thus recognizing the role of culture in their shaping), nevertheless they “are embodied in our brain-minds” (Stein 2012) and “informed by knowledge of their underlying structures and mechanisms” (Stein 2012). Similarly, “decisions about ethics and bioethics are informed by universal biologically-based considerations” (Stein 2012).

The present paper will use a more “old fashioned” philosophy based on conceptual analysis. However, even if it does not rely on the “revolutionary data about the brain and the mind, and especially about medications that act on the brain-mind, (that) will fundamentally change our thoughts about humans” (Stein 2008), it will also try to give a contribution to the clarification of psychopharmacological issues without falling into an anti-scientific stance. In particular, three

themes will be discussed, showing that conceptual/philosophical research should be considered as an integral part of the clinical psychopharmacological approach. They do not cover all possible fields of philosophical contribution to psychopharmacology, just exemplifying how a philosophical analysis of the psychopharmacological practice may work.

## Psychiatric Diagnosis and Comorbidity

Current psychiatry is dominated by the neopositivist assumptions that: a) psychiatric symptoms are objective facts that can be directly observed and described, and b) psychiatric diagnoses are the result of a process of composition of these basic symptoms performed under the control of objective and explicit rules of correspondence (the operative diagnostic criteria). In this view the objectivity of the resulting diagnosis is guaranteed by the process itself (Aragona, in press). Despite the official atheoretical claim, the corollary of this stance is the implicit assumption that psychiatric diagnoses are real natural entities with their proper boundaries, their etiology (although only supposed), and possibly their specific treatment. Moreover, the same view tends to restrict the interpretation of psychiatric comorbidity (for a comprehensive review showing the richness and complexity of this concept see: Jakovljević & Crnčević 2012) to the co-occurrence of two or more natural diseases in the same patient. The consequence of it in clinical psychopharmacology is that, ideally, every mental disorder should be treated with a specific drug and that patients presenting with psychiatric comorbidity should receive a mix of several psychoactive drugs, one for any comorbid disorder (Vella & Aragona 2000). The usual philosophical reply to these assumptions is that psychiatric disorders are not natural entities but social constructs, hence they should not be treated with drugs at all, only somatic diseases being legitimately treatable by means of somatic interventions (i.e., pharmacologically). The problem with this interpretation is that it detaches the philosophical analysis from the practical arena of clinical psychopharmacology, self-confining conceptual research in the humanistic fields and giving the (false) impression that psychopharmacology is just a matter of empirical research. However, there are alternative possibilities. One is to admit that, as clearly shown by the history of psychiatry, mental disorders are constructs (Berrios 2006, Aragona 2009b), and nevertheless to assert that constructs are not to be banished from the scientific discourse; on the contrary, they are an integral part of science. Accordingly, the epistemology of psychiatry shows that the internal rules that guided the conceptual construction of mental disorders have a clear role in their “empirical” performance. For example, it was suggested that many “empirical” problems of the current psychiatric classification (e.g., internal heterogeneity of the diagnoses and consequent lack of specificity of the pharmacological treatments) are

basically due to the way the DSM operative diagnostic criteria were designed: lack of specificity of the enlisted symptoms, lack of qualitative determination and use of a polythetic rule coupled with a quantitative diagnostic threshold (Aragona 2006a). A similar reasoning applies to the problem of comorbidity (Aragona 2009c). For example, consider the practical problem of lack of specificity of current psychopharmacological treatments. It should not be considered as a problem of the drugs themselves to overcome by searching for new more specific treatments: the problem is not in the drug itself but in the heterogeneity of the diagnostic category. If the population to be tested is heterogeneous, then any double blind trial is made on two groups of subjects that formally receive the same diagnosis but that present many differences which are not under control and that could consequently bias the results. How can we assert that the resulting effect is due to the intrinsic activity of the drug and not to a different distribution of an uncontrolled interfering variable that makes subjects more or less respondent to the drug? The same with comorbidity: we know that this phenomenon is so widespread that almost anything co-occurs with almost everything. How should this high comorbidity be controlled in our trials? If we consider all comorbidities the number of potential interfering variables increases to the infinite; if we do not consider comorbidity all these variables may interfere and bias our results; if (as it is usually done) we select pure cases excluding from the study comorbid cases, our results are in big trouble concerning their generalizability to the clinical population (where comorbidity is the rule), and so the risk is to have results that are formally correct but useless for everyday clinical practice. In short, the practical aid of such methodological reasoning about the foundations of the psychiatric classification is to make the clinicians aware of the limits intrinsic to their activity. Pharmacological treatments can (and in some cases must) be administered, but the clinicians should know that they are not acting in the ideal situation of one specific drug for any specific mental disease, and two or more specific drugs together to treat two or more specific disorders casually comorbid. Such a specificity does not exist at all, and drugs are used more pragmatically, for the simple but good reason that they modify some clinical patterns (groups of symptoms that do not match the boundaries of DSM disorders). In an early paper (Vella & Aragona 2000) our group tried to distinguish between pharmacological agents having: a) a purely symptomatic effect (e.g. the effect of benzodiazepines on anxiety), which is usually transitory and on a few superficial symptoms, independently from the syndrome in which they are inscribed, and b) a syndromic effect (e.g. the effect of antidepressants in severe depressions, those depressive forms that were once called “endogenous”), which is on the syndromic pattern as a whole with a typical progression in symptom changes with time, and usually with a more stable effect: for example, the typical recovery from the

typical depressive phase starts from the “vital” part of the syndrome (increase of energy, improvement of psychomotor retardation) while the improvement of the cognitive component comes later (and it is for this reason that clinicians accurately monitor the first phase of treatment for the increased risk of suicide in people with increased energy but still a negative appraisal of their situation).

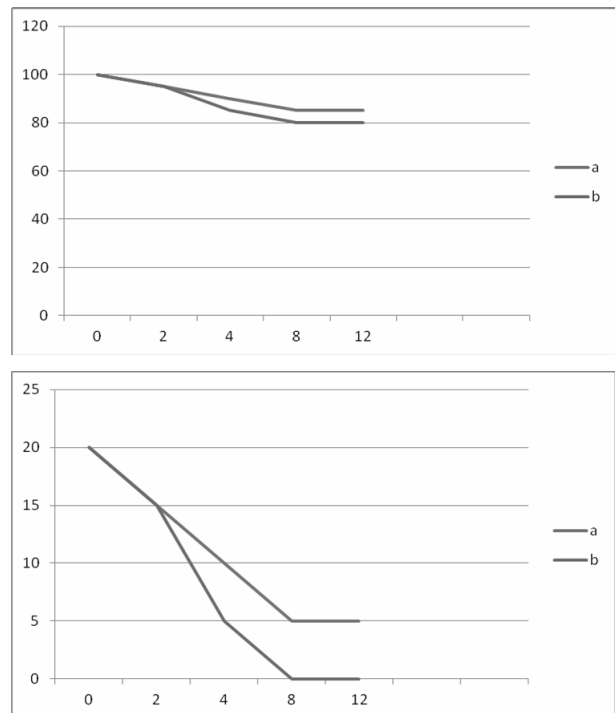
More fine-grained distinctions would probably be useful, this being only a first and still unsatisfying tentative of clarification, but to do this we need less blind “empirical” studies than good phenomenological analyses of what was once called the “psychotropism” of drugs (i.e., the drug induced modification of psychopathological phenomena).

### Merits & Limits of Psychopharmacological Evidence

The evidence-based medicine (EBM) is defined as “the conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients” (Sackett et al. 1996). In short, its function is to contrast a medicine based on authority (ironically called eminence-based medicine) and to stress the importance of considering what treatments have been effectively tested and their measured efficacy. These merits are noteworthy and obviously they need to be preserved. However, the success of EBM has been so striking that it soon became the dominant approach. At this point, the evidence-based international guidelines turned out to be not only a source of important information for the clinician but a constrictive (legally, ethically and deontologically) system that must be applied in everyday psychiatry. As expected, the reaction to this dominance has been complete rejection: conceptual works suggested that EBM has the same structure as a religion (Links 2006) or that it is ethically harmful because it is a “micro-fascist” practice (Holmes et al. 2006). Again, the risk is that conceptual/philosophical analyses do not interact with science to improve it but simply reject it. The alternative is to consider both limits and merits of the evidence-based approach (Aragona 2006b) and to use the acknowledgment of limitations to critically reconsider some clinical issues. The following are a few examples:

- The efficacy of a treatment can appear more relevant than it really is, for example due to the interference of marketing/economical biases. This is not simply the case of the few fraudulent cases of false data occasionally published even in respectful scientific journals; there are also less evident “structural” distortions of the data through methodological tricks. For example, if I am testing a new drug that I already know is as effective as an old one, I can design the study administering the new drug at the best possible dosage (the one with the best balance between therapeutic effect and side effects),

compared to highest doses of the old drug (dosages that are above the therapeutic limit, in such cases increasing the dose does not produce further therapeutic effects while the side effects continue to increase). In this case I know a priori that the new drug will be as effective as the old one but with significantly less side effects. The double blind trial is formally unobjectionable and nevertheless the results are covertly biased (more examples of structural distortions of this kind in Aragona 2009d). Finally, the way results are showed can be manipulated to suggest higher effects than those effectively found. For example, the two graphics in figure 1 show the same results of a 12 weeks trial comparing two drugs, respectively a and b. In the first graphic the real effect is reported and it shows that drug b reduces the symptoms assessed with a self-rating scale from a score of 100 to 80. In the second graphic the same 20 points reduction is transposed in a scale from 20 to 0. Although the effect is the same, the physician who is showed the second graphic is more likely to have the impression that drug b works better compared to his judgment in the case when the first graphic was shown.



**Figure 1.** An example of different graphical presentation of the same results

- Clinicians may be induced to believe also the opposite: that a given drug is less effective than it really is. This is often the case when a pharmacological agent is less studied than others. A methodological asymmetry that is typical of the evidence-based process is to be considered (Aragona 2006b): a) If a drug is tested in a double blind trial and it works better than the placebo (or the gold-standard), we can assert that it works (that there is

evidence supporting it); b) If it is tested with the same method and it does not perform better than the comparison drug, we can assert that it does not work; c) If a drug has not been tested in any clinical trial, we cannot say either that it works or that it does not, simply there is no evidence on it. However, because clinical guidelines and consensus conferences recommend only those therapies on which there is evidence, the reader is (falsely) induced to assume that those drugs that are not suggested by the guidelines do not work.

- Moreover, the efficacy of a treatment can appear more relevant than it really is, due to a bias related to the publishers. Indeed, it is well known that “those trials which show positive results and focus on new drugs are published more easily” (Angellozzi 2008). Accordingly, if a drug shows good results in two trials and negative results in other two trials, but only the first two are published, the reader assumes that it works while in reality the tested effect is conflicting. Some corrections have been proposed, such as the registration of all trials or the publication of all trial designs (both should discourage the researchers sponsored by the industry from concealing negative results), or the launch of a journal’s sections specifically dedicated to “experimental negative results”. However, in general negative results are still at major risk of not being published.
- Finally, clinicians should remember the difference between efficacy and efficaciousness and the problem of generalizing to current clinical activity the results of research trials. In fact, research trials tend to select “pure” cases while in ordinary clinical practice “pure” cases are a rarity. See for example the above discussed case of comorbidity. Another relevant example is that of mania: to be included in the study subjects have to sign informed consent; how many patients with full-blown mania that undergo compulsory treatment in psychiatric hospital would give their consent to a similar trial? The consequence is that randomized controlled trials on mania are usually made on hypomanic patients and the generalizability of their results to severe mania is suspect.

To sum up, philosophical/methodological reflections on the characteristics and limits of evidence-based research and practice should not be used to reject this approach and to propose alternative non-scientific stances. On the contrary, philosophy can help clinicians to discover biases and to be on the alert for possible structural manipulation of available evidence. This should result in a more insightful practice and in the rejection of uncritical, observant compliance to international guidelines: clinical practice must be aware of the best evidence on the matter, but the clinician must also be aware of their limits and be allowed to freely judge (“in science and conscience”) if and how they can be transposed into the peculiar practical situation he/she is actually dealing with.

## **Implicit Philosophical Assumptions Influencing Clinical Practice**

Clinicians are usually not fully aware of their general philosophical beliefs about what is the mind and its relationship with the biological processes of the brain. Their views range from extreme reductionism (there is only the brain and all mental disorders are just brain diseases) to sharp dualism (mind and brain are two distinct entities and drugs act only on the latter), with other relevant possibilities somewhere in between. The following are some of them, together with their practical consequences in clinical psychopharmacology.

### ***Reductionism***

Psychiatric reductionism is grounded on the idea that “mental” phenomena are devoid of a proper ontological status and that what really counts is the underlying level of the (supposed) biological dysfunctions. In different times the basic bodily alterations have been hypothesized to be, respectively, in circumscribed brain areas, in histological abnormalities, in metabolic alterations, in neurotransmitters and their receptors, in the intracellular transduction pathway, in genes. Usually (although not always) the reductionist psychiatrist is also an eliminativist: he/she thinks that the “mental” level of description is not scientific and that it will be eliminated following the advances of research (similarly to what happened with the concept of phlogiston in chemistry). There is nothing contradictory in this line of reasoning, but the clinician should be aware that there are practical consequences in clinical practice. Coherently with his/her model the reductionist psychopharmacologist will take a directive stance in the medical/patient relationship, in which he/she presents him/herself as the expert; he/she will be more interested in the assessment of symptoms and in making the diagnosis than in the consideration of the ongoing relational dynamic; he/she will decide what, how and when the patient must take the drugs (disregarding mutual consensus); he/she will make explicitly the diagnosis, probably explaining that it is due to basic dysfunctions and how the drugs work to bring them back to normal. The advantage of this approach is in those cases in which an assertive stance may improve compliance; moreover, asserting that the patient has a somatic disorder reduces his responsibility and consequently stigmatization (nobody would reproach a patient with amyotrophic lateral sclerosis for not speaking well, while psychotic patients are often reproached for their “symptoms”). However, there are also possible limitations, for examples in those cases in which reducing a life problem to a mere dysfunction overlooks the importance of working on the existential meaning of the psychic distress leading to mental symptoms, their meaning in the context of the patient’s life, and the exploration of possible strategies to deal with such a situation. For example, a patient experiencing panic symptoms that are successfully treated with SSRIs may think that the problem was that he/she

had pathologically decreased levels of serotonin in his brain. If this view is induced/reinforced by the “reductionist” physician, and consequently if he/she does not work psychotherapeutically on the existential meaning of his/her panic “disorder” (changing his/her reactions when faced to trigger stimuli), then a relapse after SSRIs’ discontinuation is predictable. In short, in those cases in which the goal of the treatment is not simply the *restitutio ad integrum*, that is to restore the original functioning, but the patient’s personal growth in the direction of a more flexible way to manage potentially distressful conditions, then the psychopharmacological treatment may need to be integrated with a psychotherapeutic approach. However, for our eliminativist/reductionist clinician the level of meanings is outside his/her view (being scientifically unacceptable); for this reason, he/she cannot coherently suggest to the patient that they should integrate the pharmacological treatment with a psychotherapy and is compelled to limit him/herself to the prescription of the pharmacological treatment.

### **Dualism**

Usually the dualist psychiatrist severs mental disorders in two groups: those with a somatic basis (the “real” mental diseases), and those with a psychogenic origin. The first are to be treated somatically (including the psychopharmacological treatment) and the doctor/patient model of communication is the same as seen above in the case of the reductionist. On the contrary, the latter are considered to be not “real” diseases but individual existential reactions related to the character as well as to the relational and social environment. Here the proposed “treatment” is on these latter variables with educational and social means (the psychotherapeutic option being included). From this perspective, in psychogenic disorders a pharmacological treatment should be excluded. Here again there are advantages and limits. Among the first there are that this approach preserves the advantages of the reductionist stance in the case of severe psychiatric diseases but without overlooking a psychotherapeutic treatment in the “psychogenic” cases. One of the main limits is that the dualistic view coherently excludes the possibility of integrating a psychotherapeutic treatment in the “somatic” cases and conversely a pharmacological treatment in the “psychogenic” cases. The huge amount of evidence showing that combined treatments are very often more efficacious than one kind of therapy alone contrasts with this view.

### **Interactionism**

Among those who tried to avoid the rigidity of the dualist distinction without falling into the reductionist realm there is the very well known bio-psycho-social model (Engel 1980). This model was conceived as a holistic view taking into account all the levels of explanation that might be involved in psychopathology: natural sciences, psychology, sociology, economics, politics, anthropology. Its greatest merit is surely that it does not scotomize important contributions to the study

of psychiatric disorders coming from the humanities. However, critics stressed that the biopsychosocial model simply juxtaposed several viewpoints without synthesizing them into a more comprehensive model assisting the psychiatrist in choosing the best available option(s) (Kecmanović 2011). To sum up, the bio-psycho-social psychiatrist appears to be an open-minded clinician considering all the complex variables related to psychopathological processes but also a person at risk of being the carrier of an eclecticism lacking specificity. It is also for these reasons that critics suggest that “the biopsychosocial model has been accepted as a catch phrase but has not been translated into a new form of clinical practice” (Kecmanović 2011). Of course this description suggesting a lack of specificity and a superficial stance mixing together psychopharmacology, psychotherapy and social interventions is just a caricature of this approach. Interdisciplinary groups with skilled researchers and clinicians who are expert in the involved disciplinary fields and are ready to share their knowledge within the group might be a very good answer to Kecmanović’s concerns. However, eclectic lack of specificity is not the only problem with this approach. In fact, there are also epistemological problems: what is the ontological status of the entities that are interacting? Some concerns focus on the possibility that in this way a covert form of Cartesian ontology is reintroduced.

### **Pragmatism**

This approach shares with interactionism the common need of a model able to consider all the interrelated levels involved in psychopathology, without falling into the ontological problem described above. Brendel (2006) suggests that this approach, whose roots are in the works of William James, John Dewey and Charles S. Pierce, has methods and principles to reconcile the scientific and the humanistic approaches. In the pragmatic approach a possible key principle is that of utility, a concept which was also proposed in the diagnostic debate (Kendell & Jablensky 2003). Moreover, pragmatist views have also been extended to the problem of the validity of psychiatric diagnoses (Rodrigues & Banzato 2009), a problem that until recently was considered the exclusive domain of positivist/realist scientists. In the context of the psychopharmacological approach the pragmatic concept of utility is surely very important, because what matters in the clinical context is above all what works for whom. Accordingly, both clinicians and patients want to know what is the best available therapy, and a pragmatic approach does not confine the choice of the intervention to an a priori defined possibility based on theoretical arguments. The consequence is a more flexible approach that can use any therapeutic device (several drugs but also psychological and social interventions) if they are expected to help. If this is surely an advantage, a possible problem is that this utilitarian approach seems to be based on the same ideas of the Seventeenth Century empiricism, namely that the important thing is

not what causes the disease but what removes it. This approach, which has many points of contact with the evidence-based approach, has the limit that it acts as a facilitating factor for the empirical testing of drugs' efficacy but at the same time does not enhance the research on the explanatory level (it is more focused on what works and how much it does it than on why and how it works). However, a pragmatic stance does not need to be necessarily confined to utilitarianism: being a more general theory suggesting that what we see depends on our pragmatic needs, the pragmatic view is free to focus on anything that is considered relevant, including (but not restricted to) utility. In this version the pragmatic psychopharmacologist is free to be interested in everything he/she believes is relevant for the healing process, theoretical reflections being included when relevant. To sum up, the pragmatic approach is grounded on the following basic features: a) at the epistemological level, the idea is that scientific theories do not need to be true, it is enough if they are suitable for their scientific needs; in van Fraassen's (1980) version, what is important in science is the validity of the models being used, that is whether its models are empirically adequate or not (consequently, two alternative theories are not evaluated on their truthfulness but on their adequacy to answer scientific why-questions); b) at the methodological level, the idea of a unique point of view which is truer and more valid than others is rejected; on the contrary, it is suggested that there are many scientific perspectives, everyone with its own domain of knowledge and methods. The most appropriate perspective is chosen depending on the kind of scientific question that must be answered in a particular occasion; c) at the psychopathological level, psychiatric symptoms and diagnoses are not conceived as real natural entities but as constructs (as ways to organize complex phenomenal pictures, based on specific needs: nosologic, ethical, therapeutic, legal, related to management issues, and so on); d) at the therapeutic level, this or that kind of intervention is justified on the basis of its likelihood to satisfy the clinical aims. As a consequence, there is not a therapy which is absolutely the best; rather, there are many possible interventions and the possibility to choose among them on the basis of concrete needs and aims.

## Conclusion

The renewal of the philosophical debate in psychiatry is one of the most exciting news of recent years (Fulford & Stanghellini 2008). However, when applied to psychopharmacology the achievements of the philosophical analysis are sometimes problematic. In one case the risk is to divide too sharply the evidence-based from the values-based domain, hence confining philosophy mostly to the ethical side of the debate and losing the possibility to really influence the evidence-based debate. In other cases philosophical arguments are used to reject in toto the evidence-based approach, again

excluding philosophy from the scientific debate on psychopharmacology. In this paper an alternative view was defended. By means of three examples it was suggested that philosophy has much to say on psychopharmacological clinical practice and research. Philosophy, here intended as a conceptual audit of psychopharmacology, shows that conceptual clarity is needed because it influences psychopharmacology at least at three levels: the diagnostic, the methodological and the epistemological level.

In the first case, according to the biomedical model the official ratio of psychopharmacological interventions is that the patient has a disorder (i.e., a pathological condition) that deserves treatment. The above discussed problems with a biomedical/realist approach to the diagnosis (showing that mental symptoms and diagnoses are constructs) and with comorbidity suggest that the internal rules guiding the conceptual construction of mental disorders influence their "empirical" performance. Hence, clinical psychopharmacologists should be aware of these limits when dealing with problematic outputs in psychopharmacological practice and consequently consider that more "blindly" empirical research alone will not resolve them. New conceptualizations more respondent to the psychopharmacological requirements are thus needed as well.

The methodological level explored the concept of evidence-based practice and found internal methodological limits in this perspective. The message was that clinicians should know and use the best available evidence while acknowledging their intrinsic limitations. This in order to know what they are doing and to be more free, in their choice, from rhetorical influences that tend to bias psychopharmacological practice to serve extra-scientific reasons (political and economical reasons included).

Finally, the paragraph on the epistemological implicit beliefs of psychopharmacologists showed that physicians may practice psychopharmacology having in mind (implicitly or explicitly) basic epistemological views on what is mental and how it relates to brain processes, and that such views may strongly influence their practical approach. Reductionists will tend to be more assertive and to explain to patients that they have a brain dysfunction that the drug will restore. Dualists will tend to sever organic mental diseases (to be treated pharmacologically) from psychogenic conditions (to be treated with psychotherapy). Interactionists and pragmatists will tend to integrate more treatments in the same patients with more or less epistemological clarity on why they do it. In this case the philosophical audit helps clinicians to be aware of their beliefs and of the deep reasons shaping their clinical practice, suggesting that: a) these are philosophical reasons that cannot be decided by the empirical research but by conceptual clarification; and b) having in mind one of these philosophical models, the possible actions in clinical practice should be coherent and consequent.

In conclusion, in this paper philosophy has been presented as a conceptual audit for clinical psychopharmacology. The idea that philosophy should not be an instrument to reject science or to self-confine conceptual reflections in a severed humanistic domain was defended. Philosophy is important not only at the ethical level (an assumption on which there is already sufficient agreement in the literature) but also at the methodological level. Psychopharmacologists are already familiar with the idea of a methodological audit, for example when they involve experts in statistics in designing research protocols and evaluating the results of previous trials. Similarly, they should familiarize themselves with the idea that conceptual clarification is equally important, in the same activities (research planning and evaluation) as well as in everyday clinical psychopharmacological practice. The three paragraphs of the present paper represent a partial but illustrative exemplification of this point of view.

**Acknowledgements:** None.

**Conflict of interest :** None to declare.

## References

1. Angelozzi A: *Ai confini dell'evidence-based medicine: un'analisi critica (At the boundaries of the evidence-based approach: a critical analysis)*. *Psichiatria di Comunità* 2008; 7:61-70.
2. Aragona M: *Aspettando la rivoluzione. Oltre il DSM-V: le nuove idee sulla diagnosi tra filosofia della scienza e psicopatologia (Waiting for the revolution: the new ideas on the diagnosis, between philosophy of science and psychopathology)*. Editori Riuniti, Rome, 2006a.
3. Aragona M: *Some comments on the problems and merits of the evidence-based practice*. *Int J Evid Based Healthc* 2006b; 4:396-7.
4. Aragona M: *About and beyond comorbidity: does the crisis of the DSM bring on a radical rethinking of descriptive psychopathology?* *Philos Psychiatr Psychol* 2009a; 16:29-33.
5. Aragona M: *The concept of mental disorder and the DSM-V*. *Dial Phil Ment Neuro Sci* 2009b; 2:1-14.
6. Aragona M: *The role of comorbidity in the crisis of the current psychiatric classification system*. *Philos Psychiatr Psychol* 2009c; 16:1-11.
7. Aragona M: *Il mito dei fatti. Una introduzione alla filosofia della psicopatologia (The myth of facts. An introduction to the philosophy of psychopathology)*. *Crossing Dialogues*, Rome, 2009d.
8. Aragona M: *Neopositivism and the DSM psychiatric classification: an epistemological history. Part I. Theoretical comparison*. *Hist Psychiatry* (in press).
9. Berrios GE: *Introduction: the philosophies of psychiatry – conceptual history*. *Hist Psychiatry* 2006; 17:469-486.
10. Brendel DH: *Healing psychiatry: Bridging the science/humanism divide*. MIT Press, Cambridge, MA, 2006.
11. Engel GL: *The clinical application of the biopsychosocial model*. *Am J Psychiatry* 1980; 137:535-44.
12. Fulford KWM & Stanghellini G: *The third revolution: philosophy into practice in twenty-first Century psychiatry*. *Dial Phil Ment Neuro Sci* 2008; 1:5-14.
13. Holmes D, Murray SJ, Perron A, Rail G: *Deconstructing the evidence-based discourse in health sciences: truth, power and fascism*. *Int J Evid Based Healthc* 2006; 4:180-6.
14. Jakovljević M & Crnčević Z: *Comorbidity as an epistemological challenge to modern psychiatry*. *Dial Phil Ment Neuro Sci* 2012; 5:1-13.
15. Kecmanović D: *Conceptual discord in psychiatry: origin, implications and failed attempts to resolve it*. *Psychiatr Danub* 2011; 23:210-22.
16. Kendell R & Jablensky A: *Distinguishing between the validity and utility of psychiatric diagnoses*. *Am J Psychiatry* 2003; 160:4-12.
17. Links M: *Analogies between reading of medical and religious texts*. *BMJ* 2006; 333:1068-70.
18. Rodrigues ACT & Banzato CEM: *A logical-pragmatic perspective on validity*. *Dial Phil Ment Neuro Sci* 2009; 2:40-4.
19. Sackett DL, Rosenberg WM, Gray JA, Haynes RB, Richardson WS: *Evidence based medicine: what it is and what it isn't*. *BMJ* 1996; 312:71.
20. Stein DJ: *Philosophy of psychopharmacology*. Cambridge University Press, Cambridge, 2008.
21. Stein DJ: *Psychopharmacological enhancement: a conceptual framework*. *Philos Ethics Humanit Med* 2012; 7:5.
22. van Fraassen BC: *The scientific image*. Oxford University Press, Oxford and New York, 1980.
23. Vella G & Aragona M: *Metodologia della diagnosi in psicopatologia. Categorie e dimensioni (Methodology of the diagnosis in psychopathology. Categories and dimensions)*. Bollati Boringhieri, Torino, 2000.

Correspondence:

Massimiliano Aragona  
Association Crossing Dialogues  
Via Trapani 20, 00161 Rome, Italy  
E-mail: massimiliano.aragona@uniroma1.it