Biological Psychiatry

Biological Psychiatry and *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging* Adopt Neuroscience-Based Nomenclature

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Biological Psychiatry and *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging* are pleased to adopt the neurobiology mechanism–based psychopharmacology nomenclature (Neuroscience-based Nomenclature [NbN]) proposed by the Nomenclature Task Force (1), which comprised five leading international organizations: the European College of Neuropsychopharmacology, International College of Neuropsychopharmacology, American College of Neuropsychopharmacology, Asian College of Neuropsychopharmacology, and International Union of Basic and Clinical Pharmacology (http://nbnomenclature.org/).

The NbN attempts to address a long-standing problem in psychopharmacology, namely, that its nomenclature could be interpreted to suggest erroneously that drugs alter behavior directly, without specific intervening effects on the brain. Our pharmacologic nomenclature has additional limitations. For example, it falsely conveys a notion of functional specificity. For example, "antipsychotics" are used not only to treat psychotic disorders but also to treat mood disorders, delirium, and other conditions, reflecting the fact that many of these drugs also have antidepressant, anxiolytic, and hypnotic effects. Thus, the gap between the nomenclature and the clinical usage is confusing and potentially stigmatizing. For example, it is common for patients to be concerned about the implications of receiving a prescription of the antipsychotic quetiapine for the treatment of insomnia. The current nomenclature also poorly captures important differences between drugs within a class; for example, the concept of an "atypical" antipsychotic was based on the capacity of a drug to treat psychosis without inducing extrapyramidal symptoms. Yet clozapine, the prototypical atypical antipsychotic, produces akathisia, and other atypical antipsychotics (as well as typical antipsychotics) produce various forms of extrapyramidal symptoms to varying degrees. To a large extent, these limitations reflect the fact that the current nomenclature is not based on the pharmacology or mechanism of action of psychotropic medications.

The NbN is a nomenclature system that organizes medications based on their known pharmacologic actions. It strives to be precise and flexible. New drug targets and mechanisms of action are added on an ongoing basis. It currently lists 108 compounds, representing a broad range of medication in clinical psychopharmacology practice. The NbN characterizes drugs by pharmacologic domain and mode of action. It also includes four layers of additional information: 1) indications approved by regulatory bodies (e.g., US Food and Drug Administration, European Medicines Agency), 2) profile of efficacy and side effects, 3) "practical note," and 4) neurobiology. Following this approach, as an example, the NbN classifies desipramine as 1) a norepinephrine reuptake inhibitor, 2) having approval for the treatment of major depressive disorder, 3) having efficacy for depression associated with the expected side effects and being a substrate for CYP2D6, and 4) having a variety of secondary targets and effects on brain chemistry and signaling (2).

We recognize that there will be challenges to adopting this approach beyond adjusting to abandoning established conventions. For example, medications are grouped on the basis of conventions regarding their primary mechanisms of action. However, this grouping presumes that we actually know with confidence the mechanisms through which medications produce their clinical effects, and this may not always (or even often) be the case. For example, haloperidol is a prototype dopamine D_2 receptor–preferring receptor antagonist. This conventional characterization ignores the fact that haloperidol has similar potency (within an order of magnitude) at σ_1 receptors, which may contribute to its antipsychotic effects (3) as well as several other actions. The NbN approach is particularly difficult in terms of how we characterize drugs—for example, lithium and bupropion—whose initial mechanisms of clinical efficacy remain unknown. Moreover, most psychotropic drugs produce their full clinical effects after long-term, not acute, administration, and our understanding of the drug-induced plasticity that mediates this delayed efficacy is even more poorly understood.

Using the NbN will require authors to change the way in which they write their articles. *Biological Psychiatry* and *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging* will adopt the NbN as of July 2016. To make all new articles searchable by NbN, the NbN of the substances that the article covers should replace the older terminology in the keywords of the article. To "translate" between old and new nomenclature, we recommend the official application available free of cost (on Google Play at https://play.google.com/store/apps/details?id=il.co.inmanage.nbnomenclature or Apple Store at https://itunes.apple.com/us/app/nbn-neuroscience-based-nomenclature/id927272449?mt=8) that provides instructions for authors (http://nbnomenclature.org/authors). Instructions for the NbN will also appear in the journal Guide for Authors.

The NbN effort is relatively new. It will evolve with clinical and research practice. We applaud the effort to develop a clearer nomenclature for psychopharmacology. We, the

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editors, are pleased that *Biological Psychiatry* and *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging* will participate in this initiative.

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