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# A review of the current nomenclature for psychotropic agents and an introduction to the Neuroscience-based Nomenclature

Joseph Zohar<sup>a,\*</sup>, Stephen Stahl<sup>b</sup>, Hans-Jurgen Moller<sup>c</sup>,  
Pierre Blier<sup>d</sup>, David Kupfer<sup>e</sup>, Shigeto Yamawaki<sup>f</sup>,  
Hiroyuki Uchida<sup>g</sup>, Michael Spedding<sup>h</sup>, Guy M. Goodwin<sup>i</sup>,  
David Nutt<sup>j</sup>

<sup>a</sup>Department of Psychiatry, Sheba Medical Center, and Sackler School of Medicine, Tel Aviv University, Tel Hashomer, Israel

<sup>b</sup>Department of Psychiatry, University of California, San Diego, USA

<sup>c</sup>Department of Psychiatry and Psychotherapy, Ludwig-Maximilians-University, Munich, Germany

<sup>d</sup>Department of Psychiatry and Cellular & Molecular Medicine, University of Ottawa, Canada K1Z 7K4

<sup>e</sup>Department of Psychiatry, University of Pittsburgh Medical Center, Pittsburgh, PA, USA

<sup>f</sup>Department of Psychiatry and Neurosciences, Institute of Biomedical & Health Sciences, Hiroshima University, Hiroshima, Japan

<sup>g</sup>Department of Neuropsychiatry, Keio University School of Medicine, Tokyo, Japan

<sup>h</sup>Spedding Research Solutions SARL, 6 Rue Ampere, Le Vesinet 78110, France

<sup>i</sup>University Department, Warneford Hospital, Oxford OX3 7JX, UK

<sup>j</sup>Centre for Neuropsychopharmacology Division of Brain Sciences Imperial College, London, UK

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Adherence

## Abstract

Neuroscience based Nomenclature (NbN) is a new system of classifying psychotropic drugs by their pharmacological profile. The NbN was developed to replace the current indication-based nomenclature and to provide an up-to-date and more useful framework to better inform pharmacological decisions. NbN provides updated relevant and specific scientific, regulatory and clinical information, aiming to support rational and lucid prescribing. This pharmacologically driven nomenclature, which highlights pharmacological domains and modes of action, may also increase drug adherence as it clarifies the rationale for selecting a specific psychotropic agent.

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\*Corresponding author. Tel.: +972 35186207; fax: +972 35186091.

E-mail address: [jzohar@post.tau.ac.il](mailto:jzohar@post.tau.ac.il) (J. Zohar).

## 1. Current nomenclature

In clinical practice and in the scientific literature we usually refer to different classes of medications by names based on indication (e.g. antidepressant, antipsychotic, anxiolytic, hypnotic, mood stabilizer, stimulant etc.). This follows the convention established by WHO's Drug Utilization Research Group (DURG) in the so-called ATC (Anatomical-Therapeutic-Chemical) classification system first published in 1976 and still used by the WHO Collaborating Center for Drug Statistics Methodology (WHOCC) to present drug utilisation data ([Introduction to drug utilization research, 2003](#)).

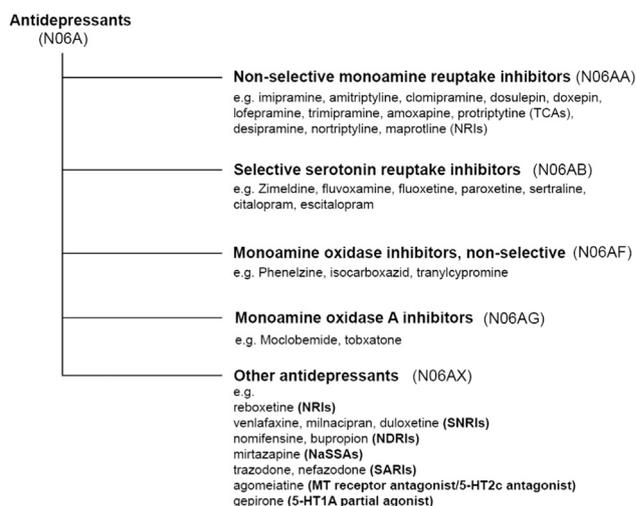
The WHO ATC nomenclature for the nervous system is presented in [Figure 1](#).

In the ATC, the relevant medications for psychiatric practice are classified under the nervous system as the anatomical category. Subsequent sub-divisions occur by broad indication. Thus, as shown in [Figure 1](#), the “psycho-analeptics” include anti-dementia drugs, antidepressants, psycho-stimulants, and psycholeptics and analeptics in combination. It is not surprising that the term psycho-analeptic is neither used nor understood, with its meaning “to exert a stimulating effect on the mind”. However, it sets the tone for a confused and confusing approach to classification, by stating a property for drugs that is too imprecise to be useful. The further grouping by anti-dementia, anti-depressant, psychostimulants and combinations is partly by indication and partly by imprecise drug action.

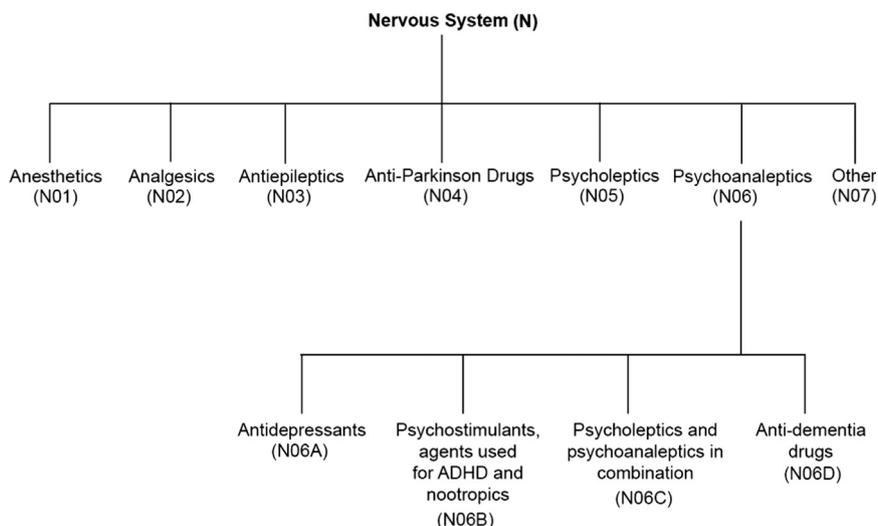
The next level of classification can be illustrated by referring to “antidepressants” ([Figure 2](#)).

This level of classification represents in part a combination of structure and indication, such as for the TCAs (tricyclic antidepressants). For other drugs, pharmacological targets and modes of action are the basis for classification; e.g. SSRIs-selective serotonin reuptake inhibitors and MAOIs-monoamine oxidase inhibitors. The nomenclature is also obviously unsatisfactory since it classifies four groups of agents according to their modes of action and the rest fall under “others”. Moreover, it has not been updated - zimeldine and nomifensine have been removed worldwide but yet are still mentioned.

The arbitrary and the ultimately unhelpful nature of this scheme in itself merits substantial re-thinking. However, like any indication-based nomenclature, it leads to further problems for patients and for prescribers. Awkward clinical situations are bound to arise because the indications are not specific and exclusive. Thus, we prescribe “antidepressants” for anxiety disorders ([Klein, 1964](#); [Baldwin et al. 2014](#)) and “antipsychotics” for depression and anxiety ([Komossa et al., 2010](#); [Zohar and Allgulander, 2011](#)). Almost all clinicians have been faced with questions from patients with an anxiety disorder such as “Doctor, I am not depressed so why are you giving me antidepressants?” The gap is growing even wider in the case of “antipsychotics” given for depression (or anxiety). “Doctor, is my situation so bad that you give me antipsychotics?” Although particular drugs are correctly used for different diagnoses, situations in which the drug names do not match the clinical conditions for which they were prescribed, may instill doubts in patients



**Figure 2** Current antidepressant nomenclature under the WHO system (adapted from Guidelines for ATC classification and DDD assignment 2015 ([WHO Collaborating Centre for Drug Statistics Methodology, 2014](#))).



**Figure 1** Current nomenclature for psychotropic drugs under the WHO system (adapted from Guidelines for ATC classification and DDD assignment 2015) ([WHO Collaborating Centre for Drug Statistics Methodology, 2014](#)).

about their use (Howland, 2014). Moreover, the discrepancies between the current naming of psychotropics and their clinical uses may have negative consequences on medication adherence (Demyttenaere, 2001).

For clinicians, as for patients, an indication based nomenclature may provide apparent simplicity but this comes at a cost. It obviously does not provide relevant pharmacological anchors to assist clinicians in making informed choices, neither for the first nor for subsequent pharmacological steps, when switching, augmenting or combining is needed.

Finally, the current nomenclature has proved a fertile ground for creative use of language to market new compounds. We have seen the invention of categories like “atypical antipsychotics”, “second generation drugs”, and other more specific classes such as SNRIs (not, as would be logical, selective noradrenergic reuptake inhibitors, but are actually serotonin and noradrenaline reuptake inhibitors) and a NaSSA (noradrenergic and specific serotonergic antidepressant).

Surprisingly, the current nomenclature has not been systematically reviewed for 60 years and is largely based on concepts and knowledge from the 1960s. For psychiatric diagnosis this would be comparable to using the DSM II or ICD 6; hence it is no wonder that key and significant concepts and findings in neuroscience are not embedded. As an example, imipramine is classified as an “antidepressant”, since its additional therapeutic benefit in panic disorder was discovered 16 years after it was approved for depression (Klein, 1964). Another more recent example is the term “atypical antipsychotic”, which mainly reflects the date the drugs were marketed rather than their relevant pharmacological characteristics. Grouping them together under the “copywriter’s” invention of “second generation antipsychotics” may be a brilliant marketing strategy but it does not provide relevant information for the clinician and can be confusing for patients when they are used to indications other than psychosis (Zohar and Allgulander, 2011).

Our expectations from a psychotropic nomenclature are that it should:

- (1) Be based on contemporary scientific knowledge.
- (2) Help clinicians to make informed choices when working out the next “pharmacological step”.
- (3) Provide a system that does not conflict with the use of medications.
- (4) Be future proof and to accommodate new types of compounds.

Since none of these criteria are met by the current nomenclature, we support a set of major suggestions to update and improve the system.

## 2. Approaches to changing psychotropic nomenclature

In an editorial entitled “Beyond psychoanalectics - can we improve antidepressant drug nomenclature?” David Nutt (2009) observed that the current terminology “has grown in a random way...” and lacks therapeutic information and

educational value, since it classifies many of the newer medications in the “other” category. That editorial sowed the seeds of the NbN, by introducing an early pharmacology-driven nomenclature for drugs for depression.

## 3. NbN - Neuroscience based Nomenclature

In 2008, the taskforce for psychotropic nomenclature was established. The core group was composed of representatives from 5 international organizations, with specific expertise in psychopharmacology

**ECNP** - European College of Neuropsychopharmacology  
**ACNP** - American College of Neuropsychopharmacology  
**AsCNP** - Asian College of Neuropsychopharmacology  
**CINP** - International College of Neuropsychopharmacology  
**IUPHAR** - International Union of Basic and Clinical Pharmacology

The mission was “to examine ways of improving the current nomenclature in psychopharmacology”.

In 2014 this group published “A proposal for an updated neuropsychopharmacological nomenclature”, which presented the concept of pharmacologically driven nomenclature (ENP (2014) 24, 1005-1014).

Over the last 4 years, the multi-axial nomenclature was presented at major meetings held in four continents: Europe (ECNP Paris, Vienna, Barcelona, Berlin), EPA (Prague); Asia (JSNP Okinawa); Brazil (Brazilian Association of Psychiatry/ Brazilian Congress of Psychiatry); and Africa (South African Biological Psychiatry Congress), and also tested via the internet by a North American audience (Stahl, 2013; Zohar et al., 2014a).

Based on extensive work and feedback from colleagues, a modified nomenclature was developed (Zohar et al., 2014b). This new version of the nomenclature received positive scientific reviews in several journals (Stahl, 2013; Howland, 2014).

## 4. Where we are now

The current NbN is pharmacologically driven and focuses on pharmacological domains and modes of action. It now includes 108 compounds, which represent the vast majority of psychotropics used worldwide.

In those compounds, 11 pharmacological domains were identified. These *pharmacological domains* reflect current knowledge and understanding regarding the neurotransmitters / molecules / systems that are modified (Table 1).

In relation to these pharmacological domains it is recognized that drugs can have actions on more than one system. In those cases the relevant domains will be specified in a hierarchical order.

The NbN include (in addition to the pharmacological domains) the modes/mechanisms of action. Based on the 108 compounds included in the nomenclature, 10 modes of action were identified (Table 2).

When a drug has more than one clinically relevant mode of action it is defined as multimodal (MM) and the respected modes are listed.

NbN also includes 4 additional dimensions: *approved indication, efficacy and side effects, practical notes and neurobiology* (Table 3).

## 5. How to use NbN

“Translating” the former nomenclature to NbN is done via a free app (NbN from the Apple Store <https://itunes.apple.com/us/app/nbn-neuroscience-based-nomenclature/id927272449?mt=8> and Google Play <https://play.google.com/store/apps/details?id=il.co.inmanage.nbnomenclature>) that was launched in October 2014.

To use this app for a specific medication one searches the medication name (generic or brand). It is also possible to search via the pharmacology, mode of action, approved indication, efficacy and side effects, and former terminology. Moreover, any of these can be combined. For example, it is possible to search all the medications that were approved for the indication of major depressive disorder,

**Table 1** Pharmacological domains.

1.	Acetylcholine
2.	Dopamine
3.	GABA
4.	Glutamate
5.	Histamine
6.	Ion Channel
7.	Lithium mimetic
8.	Melatonin
9.	Norepinephrine
10.	Opioid
11.	Serotonin

**Table 2** Modes/mechanisms of actions (MoA).

1.	Receptor agonist
2.	Receptor partial agonist
3.	Receptor antagonist
4.	Reuptake inhibitor
5.	Reuptake inhibitor and releaser
6.	Reuptake inhibitor and receptor antagonist
7.	Enzyme inhibitor
8.	Ion channel blocker
9.	Positive allosteric modulator (PAM)
10.	Enzyme modulator

**Table 3** 4 Additional dimensions.

1 <b>Approved indications</b>	Based on the recommendations of the major regulatory bodies (e.g. FDA, EMA, etc.)
2 <b>Efficacy and side effects</b>	Aimed to highlight situations in which there is evidence to support additional indication(s) (as well as approved indications), for example well supported expert guidelines. In the side effects section, only prevalent or life-changing side-effects are listed.
3 <b>Practical note</b>	Summarizes the clinical knowledge that has been “filtered” through the taskforce “sieve”.
4 <b>Neurobiology</b>	Derived from empirical data and divided into preclinical and clinical sections, with an emphasis on the latter.

and whose primary pharmacological domain is norepinephrine. Once a specific compound is identified, by swiping (to the left) compounds with similar pharmacological characteristics line up.

To test the usefulness of the NbN in scientific publications, the taskforce decided to “translate” to NbN nomenclature, two recent articles (Vieta, 2014; Miurai et al., 2014) that were published using the former terminology. “Translating” group terminology e.g. antidepressants, antipsychotics (Neuroleptics, Major tranquilizers), Anxiolytics, Hypnotics, Mood Stabilizers and Stimulants turns out to be more challenging than “translating” specific medications. To address this matter, the taskforce developed a glossary (Table 4).

The glossary has two goals: a) to ensure smooth transition of group terms from the former terminology to NbN and b) to assist author/clinicians/lecturers in the use of more precise terminology. For example, if a medication for anxiety (the former term anxiolytic) is mentioned, does it refer to a GABA-PAM, a 5HT1A receptor partial agonist (buspirone), a glutamate voltage-gated Ca channel blocker (gabapentin or pregabalin) or a histamine receptor antagonist (hydroxyzine)? Or to some or all of them? In the case of medications for psychosis (focusing on what are referred to as ‘second generation antipsychotics’), are we referring to a dopamine and serotonin receptor antagonist, a dopamine D2 receptor antagonist, a dopamine and serotonin receptor antagonist and other receptor antagonist, a dopamine and serotonin receptor partial agonist, or a dopamine and serotonin receptor antagonist and norepinephrine reuptake inhibitor? When the glossary was used to “translate” two additional reviews (Samara et al. 2014; von Wolff et al. 2013) it was able to demonstrate smooth transition from the former nomenclature to NbN.

## 6. Discussion

NbN is a pharmacological driven nomenclature of psychotropic agents that addresses the following expectations from a modern classification:

- (1) Embeds contemporary neuroscience advances.
- (2) Helps clinicians to make informed decisions about prescribing.
- (3) Presents a naming system that clarifies the rationale for selecting a specific psychotropic; thus, facilitating the relaying of information to patients (and enhancing adherence).

Table 4 NbN glossary.\*

Former terminology	NbN		Drugs
<b>Indication-based</b>	<b>(Pharmacological-based)</b>		
	<b>Pharmacology</b>	<b>Mode of action</b>	
		MM; multimodal (e.g. more than one mode)	
<b>Antidepressants</b>	<b>Drugs for depression</b>		
(TCA)	Norepinephrine	Reuptake inhibitor (NET)	Desipramine
	Norepinephrine, Serotonin	Reuptake inhibitor (NET and SERT)	Protriptyline, lofepramine, amoxapine, nortriptyline
	Norepinephrine	Reuptake inhibitor (SERT and NET)	Imipramine, dosulepin,
	Serotonin	Reuptake inhibitor (SERT)	Comipramine
	Serotonin, Norepinephrine	MM; reuptake inhibitor (SERT and NET), 5-HT <sub>2</sub> receptor antagonist	Amitriptyline
	Norepinephrine, Serotonin	MM; reuptake inhibitor (NET and SERT), 5-HT <sub>2</sub> receptor antagonist	Doxepin
	Serotonin, dopamine	Receptor antagonist (5-HT <sub>2</sub> and D <sub>2</sub> )	Trimipramine
(MAOI)	Serotonin, norepinephrine, dopamine	Enzyme inhibitor (MAO-A and -B) Reversible enzyme inhibitor (MAO-A)	Isocarboxazid, phenelzine Moclobemide
		MM; enzyme inhibitor (MAO-A and -B), releaser (DAT, NET)	Tranlycypromine
	Dopamine, norepinephrine, serotonin	Enzyme inhibitor (MAO-B and -A)	Selegiline
(SSRI)	Serotonin	Reuptake inhibitor (SERT)	Citalopram, escitalopram, fluoxetine, fluvoxamine, paroxetine, sertraline
(SNRI)	Serotonin, norepinephrine	Reuptake inhibitor (SERT and NET)	Venlafaxine, duloxetine
	Norepinephrine, serotonin	Reuptake inhibitor (NET and SERT)	Milnacipran
<b>Stimulants</b>	Dopamine and norepinephrine	Reuptake inhibitors and release	Amphetamine (D) and (D,L), lisdexamfetamine, methylphenidate (D) and (D, L)
<b>Antipsychotics</b>	<b>Drugs for psychosis</b>		
Typical (1st generation)	Dopamine	Receptor antagonist (D <sub>2</sub> )	Flupenthixol, fluphenazine, haloperidol, perphenazine, pimozide, pipotiazine, sulpiride, trifluoperazine, zuclopenthixol
	Dopamine, serotonin	Receptor antagonist (D <sub>2</sub> , 5-HT <sub>2</sub> )	Chlorpromazine, thioridazine
Atypical (2nd generation)	Dopamine	Receptor antagonist (D <sub>2</sub> )	Amisulpiride
	Dopamine, serotonin	Receptor antagonist (D <sub>2</sub> , 5-HT <sub>2</sub> )	lloperidone, loxapine, lurasidone, olanzapine, perospirone, sertindole, ziprasidone, zotepine
	Dopamine, serotonin	Receptor partial agonist (D <sub>2</sub> , 5-HT <sub>1A</sub> )	Aripiprazole
	Dopamine, serotonin, noradrenaline	Receptor antagonist (D <sub>2</sub> , 5-HT <sub>2</sub> , NE alpha-2)	Asenapine, clozapine, risperidone, paliperidone
		MM; receptor antagonist (D <sub>2</sub> , 5-HT <sub>2</sub> ) and reuptake inhibitor (NET)(metabolite)	Quetiapine
<b>Anxiolytics</b>	<b>Drugs for anxiety</b>		
	GABA	Positive allosteric modulator (GABA-A receptor, benzodiazepine site)	Alprazolam, chlordiazepoxide, clonazepam, clorazepate, diazepam, flunitrazepam, lorazepam, oxazepam
	Serotonin	Receptor partial agonist (5-HT <sub>1A</sub> )	Buspirone
	Glutamate		Gabapentin, pregabalin

Table 4 (continued)

Former terminology	NbN	Drugs
	Voltage-gated calcium channel blocker	
	Histamine Receptor antagonist (H1)	Hydroxyzine
<b>Hypnotics</b> (Benzodiazepine)	<b>Drugs for insomnia</b> GABA	Estazolam, eszopiclone, flunitrazepam, lormetazepam, midazolam, quazepam, temazepam, triazolam, zaleplon, zolpidem, zopiclone
	Melatonin Receptor agonist (M1, M2)	Melatonin, ramelteon
<b>Mood stabilizers</b>	<b>Drugs for relapse prevention</b> Glutamate	Carbamazepine, oxcarbazepine
	Glutamate	Lamotrigine
	Glutamate	Valproate Lithium

\*The glossary includes only the psychotropics relevant to former terminology. Newer medications or psychotropics not included here could be found in NbN by their name.

- (4) Enables new types of pharmacological domains and/or modes of action to fit logically into the schema.

As NbN is based on scientific knowledge, and emphasizes rational psychopharmacology hence it may foster psychoeducation and reduce confusion and distrust in patients and in the general public.

In addition to the named pharmacological domain and mode of action, NbN provides four additional dimensions: approved indications, efficacy and side effects, practical notes and neurobiology, which are accessible (via a free app) to clinicians; and brings updated cutting-edge, impartial and useful information.

NbN currently includes 108 compounds, which cover the vast majority of psychotropics worldwide. It expands our psychopharmacological vocabulary, as it enables combinations of 11 pharmacological domains (Table 1) and 10 modes of action (Table 2), and is able to accommodate new discoveries. For example, if a new target (Umbricht et al., 2014) or a new mode of is identified, NbN can be expanded in a meaningful way to address and incorporate such new developments.

The NbN is different from other existing nomenclature as it is solely pharmacologically based (focusing on pharmacological domain and mode of action) and reflects contemporary clinical and scientific knowledge. An integral part of NbN is the NbN app; a free and convenient tool which will be updated at least on a yearly basis.

A certain limitation of NbN is that in many cases neither the exact nor the mode of action that is relevant to the therapeutic effect is entirely clear. However, from the outset, NbN aims to reflect the contemporary pharmacological knowledge base, and to acknowledge its limitations in representing the ultimate scientific truth. The taskforce that assembled NbN could have taken the stand that our current knowledge base is not enough to define primary pharmacology and mechanisms of action. However, the contemporary view is that it is better to present a scientific interpretation based on knowledge than to wait for definitive conclusions. After all, clinicians need to treat their

patients now, and cannot postpone treatment until all the facts are known. Moreover, the NbN is designed as a living document, which can (and will) be updated yearly as new developments emerge and new medications are approved.

Another limitation of NbN is that it does not include fixed combinations. This actually reflects the generally negative view of the committee regarding this type of prescribing practice.

The taskforce is aware that in the current form (first edition) of the nomenclature, there are omissions (e.g. many of the brand names are missing, possible critical drug-drug interactions, data on activity at cytochromes, treatment of overdose, etc. are far from being completed). However, the taskforce encourages our colleagues to send feedback (via the NbN app), and acknowledges "Collective Colleagues Wisdom" (CCW) as a major asset to shape and improve the proposed nomenclature.

A further limitation is that in its current form, the NbN focuses on psychopharmacology, while other brain related medications such as medications for epilepsy, migraine, movement disorders, chronic pain, narcolepsy and excessive daytime sleepiness are currently missing. The taskforce recognizes this gap and a special committee was recently established to work on these neuropharmacological medications (chaired by Gitte Moos Knudsen).

Further, the NbN focuses on adult psychopharmacology. The need to address the specific aspects of the pediatric population is not addressed in the NbN in its current form. To respond to this gap a pediatric section has been established (chaired by Celso Arango).

The level of evidence is not the same for the 4 dimensions of NbN. The first - approved indication - is essentially based on the regulatory decisions of different national (e.g. FDA) or international bodies (e.g. EMA). The second and third dimensions (efficacy and side effects practical notes) reflect the opinions and interpretations of the taskforce on clinical issues, as well as empirical data. For efficacy, inclusion criteria were positive single large RCT, "heavy solid weight" clinical data and carefully crafted guidelines. Side effects were included only if they were prevalent (more than 10%) or life-changing/life-threatening.

The practical notes comprise clinical knowledge that has “filtered” through practice, by means of the taskforce “clinical sieve”, presented in a nut shell.

The fourth dimension - neurobiology - reflects empirical data, which will be updated, together with other sections of the NbN, on a regular basis.

Medications that are included in the NbN are, in principle, medications with approved CNS indications. However, as this is the first edition, the taskforce welcomes suggestions for additional medications to be included in the future. (Please check our website <http://nbnomenclature.org/>.)

In summary, the mission and the focus of the NbN taskforce is to embed current neuroscience advances in NbN. The scope is to harness NbN to help clinicians in their decisions regarding the next rational “psychopharmacology step”. The intention is to provide a comprehensive and coherent naming system that clarifies the rationale for prescription (and hence might increase adherence). The expectation is that scientific journal will require authors to use the new nomenclature. Feedback from our colleagues will be an important component in updating and fine tuning of NbN.

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Funding for this project was provided by the ECNP; the ECNP had no further role in the project design; in the collection, analysis and interpretation of data; in the writing of the manuscript; and in the decision to submit the paper for publication.

## Contributors

Joseph Zohar designed the concept, collect the data, coordinated the meetings and wrote the paper.

Stephen Stahl went over a substantial number of the agents, added comments, actively participated in at least 4 face-to face meetings, numerous teleconferences and email correspondences. He reviewed several drafts of this paper, made comments and directly contributed to the final version.

Hans-Jurgen Moller went over a substantial number of the agents, added comments, actively participated in at least 4 face-to face meetings, numerous teleconferences and email correspondences. He reviewed several drafts of this paper, made comments and directly contributed to the final version.

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Shigeto Yamawaki went over a substantial number of the agents, added comments, actively participated in at least 4 face-to face meetings, numerous teleconferences and email correspondences. He reviewed several drafts of this paper, made comments and directly contributed to the final version.

Hiroyuki Uchida went over a substantial number of the agents, added comments, actively participated in at least 4 face-to face meetings, numerous teleconferences and email correspondences.

He reviewed several drafts of this paper, made comments and directly contributed to the final version.

Michael Spedding participated in the classification framework, actively participated in at least 4 face-to face meetings, numerous teleconferences and email correspondences. He reviewed several drafts of this paper, made comments and directly contributed to the final version.

Guy M. Goodwin went over a substantial number of the agents, added comments, actively participated in at least 4 face-to face meetings, numerous teleconferences and email correspondences. He reviewed several drafts of this paper, made comments and directly contributed to the final version.

David Nutt went over a substantial number of the agents, added comments, actively participated in at least 4 face-to face meetings, numerous teleconferences and email correspondences. He reviewed several drafts of this paper, made comments and directly contributed to the final version.

All authors contributed to and have approved the final manuscript.

## Conflict of interest

Joseph Zohar has received grant/research support from Lundbeck, Servier, Brainsway and Pfizer, has served as a consultant or on advisory boards for Servier, Pfizer, Abbott, Lilly, Actelion, AstraZeneca and Roche, and has served on speakers' bureaus for Lundbeck, Roch, Lilly Servier, Pfizer and Abbott.

Stephen Stahl has received support from Astra Zeneca, Avanir, Biomarin, Forum, Allergan, Jazz, Lundbeck, Neuronetics, Noveida, Orexigen, Otsuka, PamLabs, Servier, Shire, Sunovion, Taisho, Takeda, Trius, Janssen, AssureX, Lilly, JayMac, Mylan, Novartis, Pfizer, Roche, Teva and Valeant.

Hans-Jurgen Moller served in the last 2 years as consultant, advisor or CME speaker for Astra-Zeneca, Janssen, Lundbeck, Otsuka, Schwabe, Servier.

Pierre Blier has received research grants and/or honoraria for participating in advisory boards and/or giving lectures for Astra Zeneca, Bristol Myers, Eli Lilly, Forest, Janssen, Lundbeck, Medscape, Meda-Valeant, Merck, Otsuka, Pfizer, Servier, Sunovion, Takeda.

David Kupfer Copyright, Pittsburgh Sleep Qual. Index; Advisory Board, Servier International; Equity Interest, AliphCom, Psych. Assessments, Inc., Health Rhythms, Inc.

Shigeto Yamawaki noting to declare.

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Michael Spedding has worked for, and is currently consultant for, Les Laboratoires Servier. He is CEO of Spedding Research Solutions SARL, an independent consultancy and research company, and is secretary General for IUPHAR.

Guy M. Goodwin holds shares in P1vital and has served in the last 2 years as consultant, advisor or CME speaker for AstraZeneca, Abbvie, Cephalon/Teva, Convergence, Eli Lilly, GSK, Lundbeck, Medscape, Merck, Otsuka, P1Vital, Servier, Sunovion, Takeda.

David Nutt is an advisor to British National Formulary, MRC, GMC, Department of Health, is President of the European Brain Council, past President of the British Neuroscience Association and European College of Neuropsychopharmacology, chair of the Independent Scientific Committee on Drugs (UK), is a member of the International Center for Science in Drug Policy, advisor to Swedish government on drug, alcohol and tobacco research, editor of the Journal of Psychopharmacology, sits on advisory Boards at

Lundbeck, MSD, Nalparm, Orexigen, Shire, has received speaking honoraria (in addition to above) from BMS/Otsuka, GSK, Lilly, Janssen, Servier, is a member of the Lundbeck International Neuroscience Foundation, has received grants or clinical trial payments from P1vital, MRC, NHS, Lundbeck, has share options with P1vital, has been expert witness in a number of legal cases relating to psychotropic drugs, and has edited/written 27 books, some purchased by pharmaceutical companies.

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