

Thomas A. Ban and Carlos R. Hojaij: Historical Dictionary in Neuropsychopharmacology

Collated by Mateo Kreiker

A comprehensive vocabulary of terms/words used in the different areas of research in neuropsychopharmacology and in education and clinical practice with psychotropic drugs.

Each entry is self-contained and fully comprehensible without reference to other work and includes, as much as possible, the name of the person who coined the term (word) and the publication in which it first appeared; provides the original definition of the term and changes in the definition, if applicable; describes how the definition was derived (observation, experimentation, logic, etc.); and indicates the first application of the term in neuropsychopharmacology. Each statement is referenced with the original publication. Comments on entries are restricted to correction of factual information, and queries regarding how the concept was derived if unclear. Entries are listed in alphabetical order of terms/words. Comments and replies follow the respective entry and are kept open indefinitely.

Introduction by Carlos R Hojaij

I have the honor to coordinate Project One of INHN: “**Historical Dictionary of Neuropsychopharmacology.**”

The main purpose of a dictionary is the orderly presentation of words/concepts related to a particular area of knowledge. Clarity of these concepts is of utmost importance, since it is through these words/ concepts that we construct and interpret our reality.

Preparation of a Historical Dictionary of Neuropsychopharmacology will provide us with an opportunity to examine and clarify the meaning of words/concepts used in neuropsychopharmacology and ascertain that they communicate them clearly.

While preparing this Introduction I found that several terms, like, psycho, pharmaco, neuro, etc. appear in several different combinations. For example: neuropharmacology, psychopharmacology, neuropsychopharmacology, pharmacopsychology, pharmacopsychopathology, pharmacopsychiatry, behavioral pharmacology, pediatric pharmacology, geriatric pharmacology, pharmacogenetics, etc. The picture looks quite chaotic,

but on closer examination, it seems to reflect a constant increase in the number of perspectives or even disciplines involved in studies of the mind via the brain.

Another initial observation I had was that at a certain point in time, the term “neuropsychopharmacology” replaced “psychopharmacology.” It probably reflects the early expectations from neuroscience to provide the basic underpinning for the clinical aspects of the field.

While preparing this dictionary, we should keep in mind the words of Lothar Kalinowsky and Hanns Hippus in their book, *Somatic Treatments in Psychiatry*, written in 1971, 42 years ago: “The great advances achieved during the last decades in the effective treatment of mental diseases are due to the fact that psychiatrists utilized their clinical observations for their therapeutic experiments”.

In his Foreword of *The Dictionary of Modern Medicine*, its editor, George Lundberg wrote: “When I use a word, I may mean it to say exactly what I mean it to mean. Okay. But how will anyone else know what I mean? That is the reason for dictionaries.” In preparing our Dictionary we should keep in mind Lundberg’s words as well.

With so many partially overlapping terms, and with changes in their meaning over time, it will be necessary to define each of them from the time they emerged to the present. It will be necessary to have an open dictionary in which the last inserted concept of today will not be the oldest tomorrow. It is extremely important to keep the Dictionary open, also to accommodate divergence and confrontation as truth and reality come via confrontation; compromise suppresses authenticity.

We should try to create a *Historical Dictionary* that will be passed from generation to generation. I would appreciate any suggestion you may have that could improve our project.

Please see our first “vignette” posted. You are warmly invited to send me via e-mail (crhojaj@biologicalpsychiatry.com.au) comments on this vignette, as well as new vignettes for consideration for posting on our website for comments from members of INHN.

Let’s craft a great opus together!

References:

Kalinowsky L, Hippus H. *Tratamientos Somáticos en Psiquiatria*. (Translated from the 2nd edition of the original English by Teixidor, R. Vidal.) Barcelona: Editorial Científico-Médica; 1972, p. 1

Lundberg G. Foreword. In: Lundberg G, editor. *The Dictionary of Modern Medicine*. Basel: Editiones Roche; 1992.

December 12, 2013

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DEFINITIONS

Active reflex (Joseph Knoll)

The term “active reflex” was coined by Joseph Knoll, in 1956 in the fifth part of his paper on “Experimental studies on the higher nervous activity of animals,” published in *Acta Physiologica Hungarica*. One year later, in 1957, in the sixth part of the same paper, he defined it as a conditioned motor chain reflex, analogues to conditioned chain reflexes developed by Frolov and Fursikov in Ivan Petrovich Pavlov’s laboratories, in which the conditional stimulus of a well-established conditioned reflex served as an unconditional stimulus of the consecutive conditioned reflex in the chain (Ban 1964; Pavlov 1927). The properties of the “active reflex” were defined and presented in a monograph by Knoll (1969). A behavioral pharmacological test, with the capability to differentiate “tranquilizers” by their selectiveness of blocking the “active reflex” from known central nervous system depressants, like the barbiturates, was first published in 1958-1959 (Knoll and Knoll 1958, 1959).

References:

Ban TA. Conditioning in Psychiatry. Chicago: Aldine; 1964, p.23.

Knoll J. Experimental studies on the higher nervous activity of animals. V. The functional mechanism of the active conditioned reflex. *Acta Physiol Hung* 1956; 10:89-100.

Knoll J. Experimental studies on the higher nervous activity of animals. VI. Further studies on active reflexes. *Acta Physiol Hung* 1957; 12:65-92.

Knoll J. The Theory of Active reflexes. An Analysis of Some Fundamental Mechanisms of Higher Nervous Activity. Budapest/NewYork: Publishing House of the Hungarian Academy of Sciences/ Hafner Publishing Company;1969.

Knoll J, Knoll B. Methode zur Untersuchung der spezifisch depressiven Wirkung von “Tranquilizern” auf das Zentralnervensystem I. *Arzneimittel-Forschung* 1958; 8:330-3.

Knoll J, Knoll B. Methode zur Untersuchung der spezifisch depressiven Wirkung von “Tranquilizern” auf das Zentralnervensystem II. *Arzneimittel-Forschung* 1959; 9:633-6.

Pavlov IP. Conditioned reflexes. (Translated from the Russian original into English by GV Anrep). Oxford: Oxford University Press; 1927.

March 20, 2014

Agnosia (Hector Warnes)

Disturbance in the recognition of objects following some brain injury that is not related to loss of vision, memory, language or mental retardation. The term was introduced in 1890 by Heinrich Lissauer in his paper published in *Archiv für Psychiatrie und Nervenkrankheiten*. It was derived by clinical observation.

References:

Lissauer H. Ein Fall von Seelenblindheit nebst einem Beitrage zur Theorie derselben. *Archiv für Psychiatrie und Nervenkrankheiten*.1890; 21:222-70.

February 11, 2016

Anhedonia (Thomas A. Ban)

The term “anhédonie” with the meaning of “insensibility of relating to pleasure alone” was coined by Théodule-Armand Ribot (Shorter 2005). He introduced the term in 1896, in his book, *La Psychologie des Sentiments*, to designate a “specific type of depression that was characterized by a passive lack of joy” and “loss of enjoyment and desire” (Crocq 2015). The term, “anhedonia” first appeared in the English language, one year later, in 1897, in the English translation of Ribot’s book.

References:

Crocq M-A. French perspectives on psychiatric classifications. *Dialogues in Clinical Neuroscience* 2015; 17:51-7.

Ribot T-A. *La Psychologie des Sentiments*. Paris: Felix Alcan; 1896.

Ribot T-A. *The Psychology of the Emotions*. London: W. Scott; 1897.

Shorter E. *A Historical Dictionary of Psychiatry*. New York: Oxford University Press; 2005.

July 9, 2015

Amine oxidase (Joseph Knoll)

In 1937, Blaschko, Richter and Schlossman demonstrated that tyramine oxidase, the enzyme discovered by Hare in 1928, noradrenaline oxidase and aliphatic amine oxidase was the same enzyme. They referred to the enzyme as “amine oxidase.” In the same year, 1937, as Blaschko and his associates demonstrated the presence of “amine oxidase” in the liver, Pugh and Quastel demonstrated the presence of the same enzyme in the brain. One year later, in 1938, after Zeller’s separation of diamine oxidase from “amine oxidase”, the term was replaced by the term “monoamine oxidase” to indicate that its function is restricted to the oxidative deamination of monoamines.

References:

Blaschko H, Richter D, Schlossman H. The inactivation of adrenaline. *J Physiol* 1937; 90:1-17.

Blaschko H, Richter D, Schlossmann H. The oxidation of adrenaline and other amines. *Biochemical Journal* 1937; 31:2187-96.

Hare MLC. Tyramine oxidase I. A new enzyme system in the liver. *Biochemical Journal* 1928; 22:968-79.

Pugh C, Quastel JH. Oxidation of aliphatic amines in brain and other tissues. *Biochemical Journal* 1937; 31:286-91.

Zeller EA. Über den enzymatischen Abbau von Histamin und Diaminen. *Helvetica Chimica Acta* 1938; 21:881-90.

June 26, 2014

Anna Monika Prize (Samuel Gershon)

The Anna Monika Prize is a monetary award that is awarded bi-annually to clinical scientists who have made major contributions to the understanding of the neurobiology of depression and who advanced the pharmacological options for the treatment of affective disorders. The awards are given by the Anna Monika Foundation, a private foundation, founded

by Peter Rehme, an international merchant with the assistance of Professor Florin Laubenthal of Essen by approval of the Minister of Interior of North Rhine-Westphal, in Dusseldorf, Germany, on June 9, 1965. Rehme named the Foundation after his Mother, Anna and his daughter, Monika Rief.

July 24, 2014

Anosognosia (Hector Warnes)

Denial of loss of vision associated with confabulations following an infarction in the occipital lobe. The term was introduced in 1899 by Gabriel Anton in his paper published in *Archiv für Psychiatrie und Nervenkrankheiten*. It was derived by clinical observation.

References:

Anton G. Über die Selbstwahrnehmung der Herderkrankungen des Gehirn durch Kranken bei Rindenblindheit und Rindetaubheit. *Archiv für Psychiatrie und Nervenkrankheiten* 1899; 32:86-122.

February 18, 2016

Apperceptive agnosia (Hector Warnes)

Inability to discriminate visually presented objects despite normal vision following brain injury. The term was introduced, in 1890 by Heinrich Lissauer in his paper published in *Archiv für Psychiatrie und Nervenkrankheiten*. It was derived by clinical observation.

References:

Lissauer H. Ein Fall von Seelenblindheit nebst einem Beitrage zur Theorie derselben. *Archiv für Psychiatrie und Nervenkrankheiten*.1890; 21:222-70.

March 3, 2016

Associative agnosia (Hector Warnes)

Inability to recognize objects, despite their intact representation and normal vision, in the absence of aphasia, following brain injury. The term was introduced, in 1890 by Heinrich Lissauer in his paper published in *Archiv für Psychiatrie und Nervenkrankheiten*. It was derived by clinical observation.

References:

Lissauer H. Ein Fall von Seelenblindheit nebst einem Beitrage zur Theorie derselben. *Archiv für Psychiatrie und Nervenkrankheiten*.1890; 21:222-70.

March 10, 2016

Ataraxics (Carlos R. Hojaij)

The term “ataractic” is derived from the Greek adjective, “ataractos”, that translates into English “without confusion, cool and collected”, and from the Greek noun, “ataraxia”, that translates into “peace of mind” or “freedom from confusion.” In 1955 in a paper published in the *Journal of the American Medical Association*, Howard Fabing and Alister Cameron, a professor of classics, proposed that chlorpromazine and similar drugs which produce “ataraxia,” i.e., absence of emotional upset and a condition of imperturbability, be called, “ataraxics” (Fabing 1955; Berger 1976).

References:

Berger A. History. In: Usdin E, Forrest IS, editors. *Psychotherapeutic Drugs*. Volume One. New York/Basel: Marcel Dekker; 1976, pp. 111-57.

Fabing HD. Designation of tranquilizing agents in neuropharmacology. *JAMA* 1955; 158:1461.

March 27, 2014

Atypical exogenous psychoses (Thomas A. Ban)

Exogenous psychoses which are displayed by other manifestations than delirium, epileptiform reactions, stupor or confused states (Bonhoeffer 1909). The term was introduced

in 1909 by Karl Bonhoeffer in his paper “Zur Frage der exogenen Psychosen.” The most frequently encountered atypical exogenous reactions are: “dysthymic reactions,” “hallucinatory reactions,” “paranoid reactions” and “schizophreniform reactions” (Ban and Ucha Udabe 1995).

References:

Ban TA, Ucha Udabe R. Clasificacion de la Psicosis. Buenos Aires: Editorial Salerno; 1995.

Bonhoeffer K. Zur Frage der exogenen Psychosen. Zentbl Nervenheilk 1909; 32:499-505.

January 14, 2016

Catecholaminergic activity enhancer effect (Joseph Knoll)

“Catecholaminergic activity enhancer effect” refers to an increase of catecholamine synthesis induced by a substance. The term was introduced by Joseph Knoll in 1998 in reference to findings that in rats treated for 21 days with deprenyl (0.01 mg/kg/day), a synthetic β -phenylethylamine derivative, the release of dopamine from the corpus striatum, substantia nigra and tuberculum olfactorium, and norepinephrine from the locus coeruleus was statistically significantly ($p < 0.001$) increased 24 hours after the injection of the last dose (Knoll and Miklya 1994). He also used it in reference to deprenyl-induced enhancement of electrical-stimulation-induced release of tritiated catecholamines from isolated rat brain stem (Knoll et al. 1996).

References:

Knoll J. (-)-Deprenyl (selegiline) a catecholaminergic activity enhancer (CAE) substance acting in the brain. Pharmacology and Toxicology 1998; 82:57-66.

Knoll J, Miklya I. Multiple, small dose administration of (-)-deprenyl enhances catecholaminergic activity and diminishes serotonergic activity in the brain and these effects are unrelated to MAO-B inhibition. Archives internationales Pharmacodynamie de Therapie 1994; 328:1187-209.

April 17, 2014

Chain reflex (Joseph Knoll)

The concept of "chain reflex" refers to a series of consecutive responses in which each response serves as a stimulus that evokes the next response. The term was coined and introduced in 1899 by Jacques Loeb (Loeb 1899, 1900). The concept was based on findings in research in physiology and psychology. In 1906, Charles Sherrington demonstrated that all elementary motor activities of animals are based on "chain reflexes" (Sherrington 1906). The concept of "chain reflex" was extended by Ivan Petrovich Pavlov to include conditional reflex chains (Pavlov 1927).

References:

Loeb J. Einleitung in die vergleichende Hirnphysiologie und vergleichende Psychologie. Leipzig: Barth; 1899.

Loeb J. Comparative physiology of the brain and comparative psychology. New York: Putnam; 1900.

Pavlov IP. Conditional Reflexes (Translated from the Russian original into English by G.V. Anrep). Oxford University Press, Oxford; 1927

Sherrington CS. The Integrative Action of the Nervous System. New Haven: Yale University Press; 1906.

October 22, 2015

CINP (Thomas A. Ban)

The CINP or by its full name, Collegium Internationale Neuro-Psychopharmacologicum, is an international association that provides a forum for interaction between clinicians and basic scientists involved in neuropsychopharmacology. It was founded in Zurich, Switzerland on September 2, 1957. According to its Constitution and By-Laws, the primary objective of the Collegium is "to establish an organization whose members shall meet from time to time," at least once every two years, "to consider and discuss matters related to neuropsychopharmacology and through the organization encourage and promote international scientific study, teaching and application of neuropsychopharmacology." Other objectives of the Collegium include, "consultation for the better evaluation of the biochemistry, pharmacology, safety and therapeutic efficacy of neuropsychiatric drugs" and "advice to

educational institutions, governmental agencies and other such organizations” on matters related to neuropsychopharmacology (Collegium Internationale Neuro-Psychopharmacologicum, 1977-1978; Ban and Hippus 1988).

References:

Ban TA, Hippus H, editors. Thirty Years CINP. Berlin: Springer; 1988.

Collegium Internationale Neuro-Psychopharmacologicum. Roster of Members. Brussels: CINP; 1977-1978, pp. 30-1.

March 19, 2015

CODE (Thomas A. Ban)

CODE is an acronym for Composite Diagnostic Evaluation. It is a poly-diagnostic method comprised of a set of symptoms (“codes”) which can provide diagnoses in all its component diagnostic systems; a semi-structured interview for the elicitation of all the symptoms in terms of present or absent; and diagnostic decision trees which, by specially devised algorithms organize the symptoms into distinct psychiatric disorders in the component diagnostic systems. The term was introduced in 1989 by Thomas A. Ban in his book, *CODE-DD Composite Diagnostic Evaluation of Depressive Disorders*. The methodology was developed for uncovering idiosyncratic diagnoses affected by psychotropic drugs in clinical trials in which patients are included on the basis of their “consensus-based diagnoses” (Ban 1989, 1991).

References:

Ban TA. CODE-DD Composite Diagnostic Evaluation of Depressive Disorders. Brentwood: JM Productions, Inc; 1989.

Ban TA. CODE System: Theory and practice. In: Aguglia E, Ban TA, editors. International Symposium on Functional Psychoses Today. Rome: John Libbey; 1991, pp. 279-95.

June 11, 2015

Collegium Internationale Neuro-Psychopharmacologicum (Thomas A. Ban)

The Collegium Internationale Neuro-Psychopharmacologicum, or CINP in brief, is an international association that provides a forum for interaction between clinicians and basic scientists involved in neuropsychopharmacology. It was founded in Zurich, Switzerland, on September 2, 1957. According to its Constitution and By-Laws, the primary objective of the Collegium is “to establish an organization whose members shall meet from time to time” at least once every two years, ”to consider and discuss matters related to neuropsychopharmacology and through the organization encourage and promote international scientific study, teaching and application of neuropsychopharmacology.” Other objectives of the Collegium include “consultation for the better evaluation of the biochemistry, pharmacology, safety and therapeutic efficacy of neuropsychiatric drugs,” and “advice to educational institutions, governmental agencies, and other such organizations” on matters related to neuropsychopharmacology (Collegium Internationale Neuro-Psychopharmacologicum, 1977-1978; Ban and Hippus 1988).

References:

Ban TA, Hippus H, editors. Thirty Years CINP. Berlin: Springer; 1988.

Collegium Internationale Neuro-Psychopharmacologicum. Roster of Members. Brussels: CINP; 1977-1978, pp. 30-1.

March 5, 2015

Component-specific clinical trial (Martin M. Katz)

The term, “component-specific clinical trial” (CSCT), first appeared in a paper by Martin Katz, Charles Bowden and Alan Frazer, published in 2010. It was more completely defined three years later, in 2013 by Katz, as a trial in which the method for measuring outcome is profiling the specific drug effects on the principal behavioral, mood and cognitive components of a disorder instead of focusing exclusively on changes in the overall severity of that disorder. The CSCT was employed in a series of clinical trials in the study of drug effects in depression in the early years of the 21st century, the findings of which were reviewed in

Katz's monograph, *Depression and Drugs The Neurobehavioral Structure of a Psychological Storm*, published in 2013.

References:

Katz MM. *Depression and Drugs The Neurobehavioral Structure of Psychological Storm*/ Berlin: Springer; 2013, pp. 61-71.

Katz MM, Bowden CL, Frazer A. Rethinking depression and the actions of antidepressants: Uncovering the links between the neural and behavioral elements. *J Affective Disorders* 2010; 120:16-23.

April 3, 2014

Composite Diagnostic Evaluation (Thomas A. Ban)

Composite Diagnostic Evaluation (CODE) is a poly-diagnostic method comprised of a set of symptoms ("codes") which can provide diagnoses in all its component diagnostic systems; a semi-structured interview for the elicitation of all the symptoms in terms of present or absent; and diagnostic decision trees which, by specially devised algorithms organize the symptoms into distinct psychiatric disorders in the component diagnostic systems. The term was introduced in 1989 by Thomas A. Ban in his book, *CODE-DD Composite Diagnostic Evaluation of Depressive Disorders*. The methodology was developed for uncovering idiosyncratic diagnoses affected by psychotropic drugs in clinical trials in which patients are included on the basis of their "consensus-based diagnoses" (Ban 1989, 1991).

References:

Ban TA. *CODE-DD Composite Diagnostic Evaluation of Depressive Disorders*. Brentwood: JM Productions, Inc; 1989.

Ban TA. CODE System: Theory and practice. In: Aguglia E, Ban TA, editors. *International Symposium on Functional Psychoses Today*. Rome: John Libbey; 1991, pp. 279-95.

May 28, 2015

Cycloid psychoses (Thomas A. Ban)

A group of non-deteriorating, recurrent psychoses with full remission between episodes, which circle between two “poles,” as “manic-depressive psychosis” but in which the dominant psychopathology is not “elated” and “melancholic” mood, as in “manic-depressive psychosis,” but in another area of mental pathology. The term without any qualifier was introduced, in 1928, by Karl Kleist. Prior to it, he referred to the same group of psychoses by various terms including “autochthonous constitutional psychoses”, “marginal degeneration psychoses,” “cycloid degeneration psychoses” (Kleist 1928; Shorter 2005; Teichmann 1990).

References:

Kleist K. Über zyklische, paranoide und epileptoide Psychosen und über die Frage der Degenerationpsychosen. *Schweiz Arch Neurol Neurochir Psychiat* 1928; 23:3-37

Shorter E, *A Historical Dictionary of Psychiatry*. Oxford/New York: Oxford University Press; 2005, pp. 300-3.

Teichmann G. The influence of Karl Kleist on the nosology of Karl Leonhard. *Psychopathology* 1990; 23:267-76.

February 25, 2016

Dahlem Conference (Jules Angst)

The Dahlem Conferences, named after the area of Berlin in which they were held, were inaugurated in 1974 under the joint sponsorship of the German Science Foundation (Deutsche Forschungsgemeinschaft) and the Association for the Promotion of Science and the Humanities in Germany (Stifterverband für die Deutsche Wissenschaft). Ever since, they have provided an innovative format for expert scientific exchange on a wide range of topics, in the form of one-week workshops (more than 100 to date), consisting of short presentations and intensive discussions. In the 1980s three Dahlem conferences were devoted to psychiatric topics: 1982 "The Origins of Depression: Current Concepts and Approaches," organized by J. Angst; 1986 "Biological Perspectives of Schizophrenia," by H. Helmchen and F.A. Henn; and 1987 "Etiology of Dementia of Alzheimer's Type" by A. S. Henderson and J. H. Henderson.

References:

Angst J, editor, *The Origins of Depression: Current Concepts and Approaches*. Berlin: Springer; 1983.

Helmchen H, Henn F, editors. *Biological Perspectives in Schizophrenia*. Chichester: Wiley; 1987.

Henderson AS, Henderson JH, editors. *Etiology of Dementia of Alzheimer's Type*. Chichester: Wiley; 1989.

October 2, 2014

Dahlem Conference (Carlos R. Hojaij)

In 1957, in the “psychopharmacology symposium” at the Second World Congress of Psychiatry, organized by the World Psychiatric Association (WPA) in Zurich (Switzerland), Jean Delay (1959a) proposed to classify “psychiatric medications” into three groups: “psycholeptics,” “psychoanaleptics” and “psychodysleptics.” In the same presentation, he defined “psycholeptics” as substances that produced relaxation and depressed mental activity; “psychoanaleptics” as substances that simulated mental activity; and “psychodysleptics” as substances that disturbed mental activity. He further divided “psycholeptics” into “depressors of vigilance” (hypnotics) and depressors of affect (tranquilizers) and “psychoanaleptics” into “stimulants of vigilance” (psyhostimulants) and stimulants of affect” (antidepressants). Delay (1959b) repeated the same proposition, in 1958, at the 1st Congress of the Collegium Internationale Neuro-Psychopharmacologicum (CINP), in Rome (Italy).

References:

Delay J. Intervention à propos de la terminologie et la classification des médicaments psychiatriques. In: Kline NS, editor. *Neuropsychopharmacology Frontiers*. New York: Little Brown; 1959a, pp. 426-9.

Delay J. Discussion: Fourth Symposium. In: Bradley PB, Deniker P, Radouco-Thomas C. *Neuropsychopharmacology. Proceedings of the First International Congress of Neuro-Pharmacology*. Amsterdam: Elsevier; 1959b, pp. 167-72.

August 7, 2014

Depressors of affect (Carlos R. Hojaij)

“Depressors of affect” were defined as substances which regulate the oscillation of “emotional tone” between an “apathetic,” or under responsive, and “pathetic,” or over-responsive pole, and by their action replace a “pathetic” by an “apathetic one.” They are one of the two groups of, ”psycholeptics” in Delay’s (1959a,b) classification of “psychiatric drugs,” presented in Delay and Deniker’s monograph published in 1961. “Depressors of affect” include the ”minor tranquilizers,” also referred to as “anxiolytics,” and the “neuroleptics,” also referred to as “major tranquilizers” and “antipsychotics” (Ban 1969).

References:

Ban TA. Psychopharmacology. Baltimore: William & Wilkins; 1969, p. 368.

Delay J. Intervention à propos de la terminologie et la classification des médicaments psychiatriques. In: Kline NS, editor. Neuropsychopharmacology Frontiers. New York: Little Brown; 1959a, pp. 426-9.

Delay J. Discussion: Fourth Symposium. In: Bradley PB, Deniker P, Radouco-Thomas C. Neuropsychopharmacology. Proceedings of the First International Congress of Neuro-Pharmacology. Amsterdam: Elsevier; 1959b, pp. 167-72.

Delay J, Deniker P. Méthodes Chimiothérapeutiques en Psychiatrie. Paris; Masson et Cie; 1961.

January 30, 2014

Depressors of vigilance (Carlos R. Hojaij)

“Depressors of vigilance” were defined as substances which depress the level of consciousness, lower noetic (intellectual) activity, produce a “hypnoid” state, and induce clinical and electroencephalographic sleep. They are one of the two groups of ”psycholeptics” in Delay’s (1959a,b) classification of “psychiatric drugs,” presented in Delay and Deniker monograph published in 1961. “Depressors of vigilance” include the “hypnotics” (Ban 1969).

References:

Ban TA. Psychopharmacology. Baltimore: William & Wilkins; 1969, p. 368.

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January 23, 2014

Electroencephalogram (Antonio E. Nardi)

The electroencephalogram (EEG) is the record of brain electrical activity obtained by means of an electroencephalograph (Stedman 1990). The term was introduced, in 1929, by Hans Berger in the title of his paper (Über das Elektrenzenkephalogram des Menschen) published in the *Archiv für Psychiatrie und Nervenkrankheiten*. It was the first of a series of papers in which Berger reported on his research that dealt with the recording of electric currents (action potentials) of the brain in man. Recognition that electrical activity is a natural property of the living brain dates back to detection of electric currents from the peripheral nerves of frogs by a galvanometer reported by Emil du Bois Raymond, in 1848. His discovery that the living brain generates electricity was substantiated independently, in the mid-1870s by Richard Caton (1875) and Vasilij Jakovlevich Danilevsky (1875) who recorded electrical currents and the fluctuations of these currents from the cerebral hemispheres of rabbits, monkeys and dogs (Ban 2011). Yet, it was Berger, who first succeeded with the recording of spontaneous electrical activity of the brain of man, in 1924, using electrodes attached to the intact skull. By the early 1930s, he introduced electroencephalography, a technique for recording electrical activity of the brain and showed that the spontaneous waking EEG was “sensitive to” hypoxia, hypocapnia, barbiturates, bromides, caffeine, cocaine, chloroform, morphine, scopolamine and insulin coma (Berger 1929, 1938; Gloor 1969; Fink 1978).

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July 10, 2014

Elementary symptom (Thomas A. Ban)

An “elementary symptom” (Elementarsymptom) is a psychopathological symptom, from which the other symptoms of a mental syndrome are derived (Krahl 2000). The concept was first presented by Carl Wernicke on July 19, 1892, in Breslau, Germany (now Wrocklaw, Poland), in a discussion at the 59th meeting of the East German neurologists (Nervenärzte), published, in 1893, in the *Allgemeine Zeitschrift für Psychiatrie und psychisch-gerichtlich Medizin* (Wernicke 1893). The term, itself, was introduced about a year later, in 1894, at the 61st meeting of the same group (Wernicke 1895). The origin of the concept of “elementary symptom” is in conceptualization of clinical observations. The use of “elementary symptoms,” as a “nosological principle” for the identification of mental diseases dates back to Wernicke's description and separation of Anxiety-Psychosis from other psychoses, in 1894 (Wernicke 1895).

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September 17, 2015

Endogenous enhancer regulation (Joseph Knoll)

The term endogenous enhancer regulation (EER) refers to the existence of enhancer-sensitive neurons in the brain, which have the potential to increase in a split second their activity in response to a specific endogenous enhancer substance, such as β -phenylethylamine and return equally rapidly to their original activity level in the absence of the enhancer substance. The term was coined by Joseph Knoll in his monograph *The Brain and Its Self. A Neurochemical Concept of the Innate and Acquired Drives*, published in 2005. The concept of EER is based on the finding that the electrical stimulation induced increase in norepinephrine and dopamine levels in the brain stem was significantly greater in animals after PEA administration (Knoll, Miklya, Knoll et al 1996).

References:

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October 23, 2014

Endogenous enhancer substance (Joseph Knoll)

The term “endogenous enhancer substance” is a synonym of “natural enhancer substance.” It refers to brain constituents which increase the activity of special neurons which

are sensitive to them, as for example, β -phenylethylamine (PEA) increases the activity of catecholamine producing neurons. The term was coined by Joseph Knoll in his monograph *The Brain and Its Self* published in 2005. The recognition that PEA is an “enhancer substance” and the introduction of the concept of “endogenous enhancer substance” was based on the finding that on a perfused rabbit central ear artery a low concentration of PEA did not affect smooth muscle resting tone but increased (enhanced) in a dose-dependent manner the muscle contractions in response to electrical stimulation (Knoll, Miklya, Knoll et al. 1996).

References:

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November 13, 2014

February 19, 2015

Enhancer Substance (Joseph Knoll)

The term “enhancer substance” refers to chemicals which increase the activity of special neurons which are sensitive to them, as for example, selegiline increases the activity of catecholamine producing neurons. The term was coined by Joseph Knoll in his monograph *The Brain and Its Self* published in 2005. It was based on Knoll and Miklya’s findings reported in 1994 that subcutaneous administration of selegiline in the daily dose range from 0.01 to 0.1 mg/kg to rats for 21 days significantly increased catecholamine levels in the striatum, substantia nigra, tuberculum olfactorium (dopamine) and locus coeruleus (norepinephrine).

References:

Knoll J. *The Brain and Its Self. A Neurochemical Concept of the Innate and Acquired Drives.* Springer, Berlin/Heidelberg/New York, 2005. pp. 25-94.

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October 16, 2014

Exogenous Psychoses (Thomas A. Ban)

Exogenous psychoses are nonspecific psychoses which follow such physical conditions as acute infectious disease, infectious chorea, acute exhaustion as in cachexia or anemia, autointoxication as in dropsy, uremia, jaundice, diabetes and Basedow's disease (Bonhoeffer 1909). In typical cases, they become manifest in delirium, epileptiform reactions, stupor or confused states. The term was introduced in 1909 by Karl Bonhoeffer in the title of his paper "Zur Frage der exogenen Psychosen." The diagnostic concept of "exogenous psychoses" is based on clinical observations.

References:

Bonhoeffer K. Zur Frage der exogenen Psychosen. Zentbl Nervenheilk 1909; 32:499-505.

January 7, 2015

Feeling of loss of feeling (Thomas A. Ban)

The term "feeling of loss of feeling" (das Gefühl man habe keine keine Gefühle mehr) was introduced in 1913, in the first edition of Karl Jaspers' *General Psychopathology*, in reference to a "distressful feeling of not having any feeling" (Jaspers 1963; Shorter 2005). The term was adopted by the AMDP System, in which, in the 1982 English edition and translation, it was defined as "Feeling that one has lost the ability for emotional resonance: loss or absence of feeling, feeling of emotional emptiness, feeling that one's emotions are 'dead'" (Guy and Ban 1982).

References:

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July 16, 2015

Glass-cylinder seeking drive (Joseph Knoll)

The term “glass-cylinder seeking drive” (GCSD) was coined by Joseph Knoll, in 1969, in his monograph entitled *The Theory of Active Reflexes*. The glass-cylinder is a 30 cm high, 16 cm (bottom) to 12 cm (top) wide cylinder-shaped open box, with a metal plate on the bottom and a side opening, through which a rat of up to 350 to 400 g body weight can enter (Knoll 1956; Knoll and Knoll 1958). The GCSD is based on a conditioned motor (avoidance) reflex in which rats are conditioned to jump to the upper rim of a glass cylinder to an auditory (sound of a bell) conditional stimulus (CS) to “escape” burning heat (60 degree Celsius), the unconditional stimulus (US) delivered via the metal plate at the bottom of the cylinder. Rats acquired the GCSD, jump to the upper rim of the cylinder as soon as placed into the cylinder, even without the sound of a bell (CS) by developing a second order visual conditional (chain) reflex to the glass cylinder itself. The GCSD is so strong, that even if there is a receptive female and/or food at the bottom of the cylinder, rats ushered into the cylinder jump to the ceiling of the cylinder. In some rats, the GCSD qualifies for an “in-extinguishable active reflex” that is retained for lifetime (Knoll 2014). Knoll (1969, 2005) perceives GCSD as a specific acquired drive, an unnatural urge that overrides innate drives, such as hunger or sexual drives. GCSD was initially employed in a series of behavioral pharmacological studies conducted with centrally acting drugs by Knoll (1968) in the late 1950s and 1960s. After the demonstration by Berta Knoll, in 1961, that GCSD cannot be acquired in the mouse the study of GCSD became central to Knoll’s research in the evolution of homo sapiens. The findings of this research and the conceptualization of these findings were presented by Knoll (2005) in his monograph, *The Brain and Its Self* (Knoll 2014).

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April 10, 2014

IGSAD (International Group for the Study of Affective Disorders) (Jules Angst)

The International Group for the Study of Affective Disorders (IGSAD) was founded in 1970 by Jules Angst, Jan-Otto Ottosson, Carlo Perris and George Winokur. IGSAD's inaugural meeting was organized by Pierre Pichot in Paris in 1970. The meetings of IGSAD provided a valuable forum for leading mood researchers to meet with some regularity in order to exchange and discuss their findings. The first meetings dealt with the classification and long-term course of mood disorders, and with the prophylactic efficacy of lithium. The last meeting of the IGSAD took place in 1990.

September 18, 2014

Leonhard's phasic psychosis (Thomas A. Ban)

The term "phasic psychoses" coined by Edna Neele, in 1949, was adopted by Karl Leonhard, in 1957, in his *Classification of Endogenous Psychoses* in which he used the term

to indicate a class of psychoses characterized by episodic course with full remission between episodes. The scope of “Leonhard’s phasic psychoses” is narrower than Neele’s. It is restricted to manic-depressive disease, pure melancholia, pure mania, pure depressions and pure euphorias.

References:

Leonhard K. Aufteilung der endogenen Psychosen. Berlin: Akademie-Verlag; 1957.

Neele E. Die phasischen Psychosen mit Ihrem Erscheinungs – und Erbbild. Leipzig: J.A. Barth; 1949.

January 2, 2015

Möbius' endogeny theory (Thomas A. Ban)

The notion that for developing some mental illness the only essential prerequisite is an inborn predisposition. It was first presented, in 1997 by Paul Julius Möbius in his “Outline of the Doctrine of Nervous Diseases” (Ban 2005; Shorter 2005).

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Ban TA. Neuropsychopharmacology the interface between genes and psychiatric nosology. In Lerer B, editor. Pharmacogenetics of Psychotropic Drugs. Cambridge: Cambridge University Press; 2005, pp. 36-56.

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Shorter E. A Historical Dictionary of Psychiatry. Oxford: Oxford University Press; 2005.

February 18, 2016

Monoamine Oxidase (Joseph Knoll)

Monoamine oxidase is the enzyme that metabolizes monoamines by oxidative deamination in the body. The generic name, “monoamine oxidase,” was given to the enzyme by Albert Zeller, in 1938, in order to differentiate within “amine oxidase” -- shown to be present

in 1937 in the liver by Blaschko, Richter and Schlosberg, and in the brain by Pugh and Quastel - the enzyme that metabolizes monoamines from the enzyme that metabolizes diamines in the body. The enzyme is also referred to as “mitochondrial monoamine oxidase” because it is located intracellularly on the outer membrane of mitochondria.

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Blaschko H, Richter D, Schlossmann H. The oxidation of adrenaline and other amines. *Biochemical Journal* 1937; 31:2187-96.

Pugh C, Quastel JH. Oxidation of aliphatic amines in brain and other tissues. *Biochemical Journal* 1937; 31:286-91.

Zeller E.A. Über den enzymatischen Abbau von Histamin und Diaminen. 2. Mitteilung. *Helvetica Chimica Acta* 1938; 21:880-90.

May 22, 2014

Type-A monoamine oxidase (Joseph Knoll)

Type-A monoamine oxidase (MAO-A) is the form of monoamine oxidase (MAO) that is sensitive to clorgyline. Clorgyline, 3-(2, 4-dichlorophenoxy)-N-methyl-N-2-ynylpropan-1-amine, is an irreversible MAO inhibitor substance, structurally related to pargyline. This term was coined and introduced, in 1968, by Johnston, to distinguish between clorgyline-sensitive and insensitive forms of monoamine oxidase (MAO) enzymes that he referred to as Type-A monoamine oxidase and Type-B monoamine oxidase, respectively. MAO-A was found to be present in the neurons, astroglia, gastrointestinal tract, liver and placenta (Neff and Gorodis 1972). By the early 1970, it was recognized that MAO-A is primarily responsible for the oxidative deamination of the monoamines serotonin, melatonin, noradrenaline (norepinephrine) and adrenaline (epinephrine), and not only of serotonin, as originally proposed (Costa and Sandler 1972).

References:

Costa E, Sandler M (editors). *Monoamine Oxidases – New Vistas. Advances in Biochemical Psychopharmacology. Volume 5.* New York: Raven Press; 1972.

Johnston JP. Some observations upon a new inhibitor of monoamine oxidase in human brain. *Biochemical Pharmacology* 1968; 17:1285-97.

Neff NH, Gorodis C. Neuronal monoamine oxidase: specific types and their rates of formation. *Advances Biochemical Psychopharmacology* 1972; 5:2017-23.

August 14, 2014

Type-B monoamine oxidase (Joseph Knoll)

Type-B monoamine oxidase (MAO-B) is the form of monoamine oxidase (MAO) that is insensitive to clorgyline. Clorgyline, 3-(2, 4-dichlorophenoxy)-N-methyl-N-2-ynylpropan-1-amine, is an irreversible MAO-inhibitor substance, structurally related to pargyline. The term was coined and introduced, in 1968, by Johnston, to distinguish between clorgyline-sensitive and insensitive forms of monoamine oxidase (MAO) enzymes, referred to as Type-A monoamine oxidase and Type-B monoamine oxidase, respectively. MAO-B was found to be present in the neurons, astroglia and platelets (Neff and Gorodis 1972) and was primarily responsible for the oxidative deamination of beta-phenylethylamine and benzylamine (Costa and Sandler 1972). In 1971, it was shown that MAO activity progressively increased in the aging brain (Robinson, Davis, Nies et al. 1971) and, by 1980, it was also recognized that this was due entirely to the increase in MAO-B concentrations in brain tissue (Fowler, Wiberg, Oreland et al. 1980). The first selective MAO-B inhibitor, (-)-deprenyl/selegiline, an (R) –N-methyl-N-(1-phenylpropan-2-yl) prop-2-yn-1-amine, was identified, in 1972, by Knoll and Magyar.

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August 28, 2014

Morel's degeneration theory (Thomas A. Ban)

The notion that mental illness is the result of an innate biological defect, the result of corruption of germ plasm that becomes manifest in increasingly severe mental syndromes from generation to generation. It was first presented in 1857 by Bénédict Augustin Morel in his "treatise" on "degeneration" (Ban 2002; Shorter 1997, 2005).

References:

Ban TA. Neuropsychopharmacology the interface between genes and psychiatric nosology. In Lerer B, editor. Pharmacogenetics of Psychotropic Drugs. Cambridge: Cambridge University Press; 2005, pp. 36-56.

Morel B-A. Traité des dégénérecences physiques, intellectuelles et morales de l'éspece humaine. Paris: Bailliere; 1857.

Shorter E. A History of Psychiatry. New York: John Wiley; 1997.

Shorter E. A Historical Dictionary of Psychiatry. Oxford: Oxford University Press; 2005.

February 4, 2016

Multivantaged Assessment Method (Martin M. Katz)

The term "multivantaged assessment method" (MVAM) was introduced, in 1984 by Martin M. Katz and co-investigators in their report of the US National Institute of Mental Health (NIMH) Collaborative Study of the Psychobiology of Depression. It is based on a dimensional conceptualization of mental disorders, and the assumption that mental disorders are structured by interaction between their measurable emotional and behavioral components. Because of the many ways these components can be manifested, in a multivantaged assessment, methods of assessment from several "vantage" points are combined. The prototype multivantaged assessment includes quantified observational methods, such as ratings scales by experts, subjects' judgment on current state, and measurement of cognitive and psychomotor

performances. The multivantaged assessment method was employed in a series of studies in depression, in the Departments of Psychiatry and Pharmacology in the University of Texas Health Science Center at San Antonio, by Katz and his associates, and the term reappeared in 2004, 20 years after its introduction, in a report of these studies on the “onset and sequence of clinical actions” of antidepressants, published in the *International Journal of Neuropsychopharmacology*. Information on the development and definition of the concept of MVAM was presented by Katz in 2013, in his monograph, *Depression and Drugs - The Neurobehavioral Structure of a Psychological Storm*.

References:

Katz MM. *Depression and Drugs - The Neurobehavioral Structure of a Psychological Storm*. New York; Springer: 2013, pp. 21-34.

Katz MM, Houston JP, Brannan S, Bowden CL, Berman N, Swann A, Frazer A. A multivantaged behavioral method for measuring onset and sequence of the clinical actions of antidepressants. *International J of Neuropsychopharmacology* 2004; 7:471-9.

Katz MM, Koslow SH, Berman N, Secunda S, Maas JW, Casper R, Kocsis J, Stokes P. A multivantaged approach to the measurement of behavioral and affect states for clinical and psychobiological research. *Psychological Reports Monograph* 1984; 55:619-73

May 1, 2014

National Advisory Committee on Psychopharmacology (Martin M. Katz)

The National Advisory Committee on Psychopharmacology was established in 1956 by the National Institutes of Health (NIH) to guide a new program of the National Institute of Mental Health (NIMH) that would stimulate research in the new science of psychopharmacology. The new program was implemented with the establishment of the Psychopharmacology Service Center (PSC) from the \$2 million allocated in 1956 by the US Congress to the NIH in response to the discovery of new drugs for the treatment of mental disorders. The Committee consisted of expert psychiatrists, pharmacologists, psychologists and statisticians. Its members included Louis Goodman (Pharmacology), Seymour Kety (Biological Science), Nathan Kline (Psychiatry), Morton Kramer (Biostatistics) and Joseph Zubin (Psychology). The appointed Chairman of the Committee was Ralph Gerard; the Executive Secretary, Martin Katz (Katz 2011). The role of the Committee was to both guide

the activities of the PSC, its leader, Jonathon Cole and staff, in implementing the program initiatives, and to review applications for research grants from outside investigators in the field (Cole 2011). In the early 1960's, most of the Committee's research grant review function was transferred from the NIMH to the NIH. Its prime function, following the PSC becoming the Psychopharmacology Research Branch in 1965, was to advise on ongoing and planned clinical research goals of the psychopharmacology program.

References:

Cole JO interviewed by Ban TA. In: Ban A, editor An Oral History of Neuropsychopharmacology - The First Fifty Years Katz MM, volume editor. Peer Interviews, Volume 10 - "History of the ACNP." Nashville: American College of Neuropsychopharmacology; 2011. p. 45-53.

Katz MM interviewed by Ban TA. In: Ban A, editor An Oral History of Neuropsychopharmacology - The First Fifty Years Katz MM, volume editor. Peer Interviews, Volume 10 - "History of the ACNP." Nashville: American College of Neuropsychopharmacology; 2011. p. 77-81.

November 6, 2014

National Institute of Mental Health Collaborative Studies in Psychopharmacology (Martin M. Katz)

The National Institute of Mental Health Collaborative Study refers to the study the Psychopharmacology Service Center (PSC) of the National Institute of Mental Health (NIMH) was charged with to carry out under the guidance of the National Advisory Committee on Psychopharmacology (Katz 2011). It was a nationwide controlled study of phenothiazine treatment in acute schizophrenia that was led by principal investigators Jonathon O. Cole, Gerald L. Klerman and Salomon Goldberg and carried out in disparate public, private and university hospitals (National Institute of Mental Health, Psychopharmacology Service Center Collaborative Study Group 1964; National Institute of Mental Health, Psychopharmacology Research Branch Collaborative Study Group 1967).

References:

Katz MM interviewed by Ban TA. In: Ban A, editor An Oral History of Neuropsychopharmacology - The First Fifty Years Katz MM, volume editor. Peer Interviews,

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National Institute of Mental Health Psychopharmacology Service Center Collaborative Study Group. Phenothiazine treatment of acute schizophrenia: Effectiveness. Arch G Psychiat 1964; 10:246-61.

National Institute of Mental Health Psychopharmacology Research Branch Collaborative Study Group. Differences in clinical effects in three phenothiazines in acute schizophrenia, Dis Nerv Syst 1967; 28:369 -83.

October 16, 2014

Natural enhancer substance (Joseph Knoll)

The term "natural enhancer substance" is a synonym of "endogenous enhancer substance." It refers to brain constituents which increase the activity of special neurons which are sensitive to them, as for example, β -phenylethylamine (PEA) increases the activity of catecholamine producing neurons. The term was coined by Joseph Knoll in his monograph *The Brain and Its Self* published in 2005. The recognition that PEA is an "enhancer substance" and the introduction of the concept of "natural enhancer substance" was based on the finding that on a perfused rabbit central ear artery a low concentration of PEA did not affect smooth muscle resting tone but increased (enhanced) in a dose-dependent manner the muscle contractions in response to electrical stimulation (Knoll, Miklya, Knoll et al. 1996).

References:

Knoll J. *The Brain and Its Self. A Neurochemical Concept of the Innate and Acquired Drives.* Springer, Berlin/Heidelberg/New York, 2005. pp. 25-94.

Knoll J, Miklya I, Knoll B, Markó R, Rácz D. Phenylethylamine and tyramine are mixed acting sympathomimetic amines in the brain. *Life Sciences* 1996; 58:2101-14.

February 19, 2015

Neuroleptics (Thomas A. Ban)

The term “neuroleptic” first appeared in 1955 in the title of Jean Delay and Pierre Deniker’s paper, “Hibernothérapies et cures neuroleptiques en psychiatrie”, published in the *Bulletin of the National Academy of Medicine* (Paris) for the designation of a new class of drugs. By introducing the term, Delay and Deniker linked the specific therapeutic activity of this new class of drugs to particular neurological effects. The term reappeared in the title of the International Colloquium on Chlorpromazine and Neuroleptic Drugs in Psychiatric Treatment, held in Paris from October 20 to 22 in the same year. At the First International Symposium on Psychotropic Drugs held in May 1957 in Milan, “neuroleptics” were defined by Delay and Deniker as drugs which (i) induce a “psycholeptic state without hypnotic effect (i.e., indifference, affective and emotional neutrality), and decrease initiative and motor activity without gross alteration of vigilance and cognitive functions; (ii) control (treat) excitation, aggressiveness and agitation in manic and psychotic patients; (iii) improve (decrease) acute and chronic psychotic symptoms (hallucinations, delusions), ameliorate deficit symptoms of schizophrenia and control the symptoms induced by psychodysleptics; iv. induce neurovegetative and neurological side effects; and (v) exert their action at sub-cortical level (brain stem reticular formation, diencephalon) (Crocq and Macher 2006). The definition includes their description of the effects of chlorpromazine published in 1952 in a paper coauthored by Harl (Delay, Deniker and Harl 1952). The criteria were simplified in 1961 in their monograph, *Méthodes Chimiothérapeutiques en Psychiatrie*, in which to qualify for a neuroleptic, therapeutic effects in psychoses associated with neurological signs sufficed (Delay and Deniker 1961). It was this simple definition of neuroleptics that was adopted in 1967 in Number 371 of the Technical Report Series of the World Health Organization (WHO).

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World Health Organization. Research in Psychopharmacology. Technical Report Series 371. Geneva: WHO; 1967.

January 16, 2014.

Nihilistic delusions (Thomas A. Ban)

The denial of the existence of anything to which the patient's attention is directed (Shorter 2005). Patients with nihilistic delusions deny their own existence and the existence of the world (Cotard 1974). The term "du délire négations" was introduced in 1880 by Julius Cotard in his presentation to the Société médico-psychologique in Paris. It was first published in 1882 with the title *Du délire négations*, in *Archives de Neurologie*. The syndrome dominated by "nihilistic delusions" is referred to as Cotard's syndrome (Shorter 2005).

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Shorter E. *A Historical Dictionary of Psychiatry*. Oxford: Oxford University Press; 2005, pp. 63-64.

March 17, 2016

NIMH Collaborative Studies in Psychopharmacology (Martin M. Katz)

The National Institute of Mental Health Collaborative Study refers to the study the Psychopharmacology Service Center (PSC) of the National Institute of Mental Health (NIMH) was charged with to carry out under the guidance of the National Advisory Committee on Psychopharmacology (Katz 2011). It was a nationwide controlled study of phenothiazine treatment in acute schizophrenia that was led by principal investigators Jonathon O. Cole, Gerald L. Klerman and Salomon Goldberg and carried out in disparate public, private and university hospitals (National Institute of Mental Health, Psychopharmacology Service Center Collaborative Study Group 1964; National Institute of Mental Health, Psychopharmacology Research Branch Collaborative Study Group 1967).

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Katz MM interviewed by Ban TA. In: Ban A, editor An Oral History of Neuropsychopharmacology - The First Fifty Years Katz MM, volume editor. Peer Interviews, Volume 10 - "History of the ACNP." Nashville: American College of Neuropsychopharmacology; 2011. p. 77-81.

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National Institute of Mental Health Psychopharmacology Research Branch Collaborative Study Group. Differences in clinical effects in three phenothiazines in acute schizophrenia, Dis Nerv Syst 1967; 28:369-83.

October 16, 2014

Nosologic homotypes (Thomas A. Ban)

Nosologic homotypes are psychiatric populations identical in psychopathological symptoms and assigned to the same position in a “nosologic matrix” based on form of onset, course, outcome, polarity and totality of clinical manifestations. The term was coined by Thomas A. Ban, while searching for psychiatric populations more suitable for neuropsychopharmacological and molecular genetic research than those identified by psychiatric diagnoses. It was introduced, in 2002, in his chapter on

“Neuropsychopharmacology: the interface between genes and psychiatric nosology”, published in Bernard Lerer’s *Pharmacogenetics of Psychotropic Drugs* (Ban 2002, 2007).

References:

Ban TA. Neuropsychopharmacology: the interface between genes and psychiatric nosology. In: Lerer B, editor. *Pharmacogenetics of Psychotropic Drugs*. United Kingdom: Cambridge University Press; 2002, pp. 36-56.

Ban TA. Towards a clinical methodology for neuropsychopharmacological research. *Neuropsychopharmacologia Hungarica* 2007; 9:81-90.

May 21, 2015

Nosologic homotyping (Thomas A. Ban)

Nosologic homotyping is a methodology for identifying “nosologic homotypes” (see Dictionary), identical in their psychopathological symptoms and assigned the same position in the “nosologic matrix.” The term was introduced in 2007 by Thomas A. Ban, in his paper “Towards a clinical methodology for neuropsychopharmacology.”

Reference:

Ban TA. Towards a clinical methodology for neuropsychopharmacology. *Neuropsychopharmacologia Hungarica* 1007; 9:81-9.

June 4, 2015

Nosological postulate (Thomas A. Ban)

The notion that in a “nosological entity” there is a close correspondence between etiology, brain pathology, symptom pattern and outcome picture. It was put forward by Karl Kahlbaum in the mid-1870’s (Jablensky 1981; Kahlbaum 1874)).

References:

Jablensky A. Symptoms of course and predictions of outcome in the functional psychoses. Some nosological indications. In: Tognomi G, Bellantonio C, Lader M, editors. *Epidemiological Impact of Psychotropic Drugs*. Amsterdam: Elsevier; 1981.

Kahlbaum K. *Die Katatonie oder des Spannungs irresein*. Berlin: Hirschwald; 1874.

January 28, 2016

Nosology (Thomas A. Ban)

Nosology is the discipline that deals with “theory” that provides organizing principles for identifying and classifying disease ((Ban 2000; Jaspers 1913; Shorter 2005). The term first appeared, in 1743, in Robert James’ *Medical Dictionary* (James 1743) and was adopted, in 1763, in the title of Francois Boissier de Sauvages’ *Nosologia Methodica*. The use of the term was conceptually derived and is based on Sauvages’ stipulation that a disease should be defined by the enumeration of symptoms that suffice to recognize it and distinguish it from other diseases, and a classification should be devised in a manner that it should allow the attribution of each patient to one and only one class (Sauvages 1763).

References:

Ban TA. Nosology in the teaching of psychiatry. *Jornal Brasileiro de Psiquiatria*. 2000; 49:39-49.

De Sauvages FB. *Nosologia methodica sistens morborum classes, genera et species, juxtà sydenhami mentem & botanicorum ordinem. sumptibus Fratrum de Tournefort, 1763, p. 552.*

James R. *Medical Dictionary*. London: J. Osborne; 1743.

Jaspers K. *Allgemeine Psychopathologie*. Elin: Springer; 1913.

Sydenhami mentem et botanicorum ordinem. Amsterdam: Frat des Tournefort; 1763-8.

Shorter E. *A Historical Dictionary of Psychiatry*. Oxford/New York: Oxford University Press; 2005.

September 24, 2015

Papez circuit (Thomas A. Ban)

Papez circuit has been called, “a reverberating circuit in the brain which consist of the cingulate gyrus, hippocampus, amygdala, mammillary bodies, hypothalamus and anterior thalamus” (Kaplan and Saddock 1988). It was named after James Papez who, in 1939, suggested that emotions are processed through these structures in the brain before becoming subjectively experienced feelings in the cerebral cortex (Ban 2011; Papez 1939). The Papez circuit is also referred to as “limbic lobe”, because its site corresponds with Paul Broca’s “great limbic lobe” (Broca 1878), and “visceral brain, because of its numerous connections with the autonomic nervous system (Maclean 1942).

References:

Ban TA. Preface. In Ban TA, editor. An Oral History of Neuropsychopharmacology. Volume 2. Fink M, volume editor. Neurophysiology. Brentwood: American College of Neuropsychopharmacology; 2011, pp. IX-XXII.

Broca P. Anatomie compare des corconvolutions cérébralés; le grand lobe limbique. Rev Anthrool 1878 1: 385-498.

Kaplan HI, Saddock BJ, editors. Synopsis of Psychiatry, Behavioral Sciences, Clinical Psychiatry. Fifth edition. Baltimore: Williams and Wilkins; 1988.

MacLean PD. Psychosomatic disease and the visceral brain. Recent developments bearing on the Papez theory of emotions. Psychosom Med 1949; 11: 338-53.

Papez W. A proposed mechanism of emotions. Arch Neurol Psychiatry 1937; 38:725-43.

December 17, 2015

Pharmacogenetics (Thomas A. Ban)

The discipline that studies genetically determined inherited inter-individual differences (variability) in therapeutic response to drugs and susceptibility to adverse effects (Lerer 2002). The term pharmacogenetics was coined and introduced in 1959 by Friedrich Vogel in his article on “Moderne probleme der humangenetik” published in the journal, *Ergebnisse Inneren Medizin und Kinderheilkunde* (Arranz and de Leon 2007; Vogel 2007).

References:

Arranz MJ, de Leon J. Pharmacogenetics and pharmacogenomics of schizophrenia: a review of last decade of research. *Mol Psychiatry*. 2007 Aug; 12 (8): 707-47.

Lerer B. Introduction. In Lerer B, editor. *Pharmacogenetics of Psychotropic Drugs*. Cambridge: Cambridge University Press; 2002, 3-17.

Vogel F. Moderne probleme der humangenetik” *Ergebnisse Inneren Medizin und Kinderheilkunde*. 1959; 12: 52-125.

March 10, 2016

Pharmacopsychology (Thomas A. Ban)

The term pharmacopsychology was introduced in Emil Kraepelin’s Thesis for his “habilitation,” the German equivalent for a PhD, published in 1892 with the title *Über die Beeinflussung einfacher psychischer Vorgänge durch einige Arzneimittel* (On the Modulation of Simple Psychological Processes by Some Medicines). It defined an area of pharmacological research that studied the effects of nervina (centrally acting drugs), on mental processes, such as attention, memory, language, etc., with the employment of psychometric performance tests in normal subjects (Muller, Fletcher and Holger 2006). Kraepelin (1881, 1882a, b, 1883), began with his investigations that led to the concept of pharmacopsychology in Wilhelm Wundt’s (1910) laboratories of experimental psychology in the Department of Philosophy, at the University of Leipzig, in Germany, in 1881; he continued his research in Dorpat (now Tartu, Estonia), and completed it in Heidelberg in 1892 (Steinberg 2001; Steinberg and Angermeyer 2001). Included among the substances he studied were common recreational “drugs,” such as alcohol, coffee, and tea, and medicinal products, such as amyl nitrite, chloral hydrate, chloroform, morphine and paraldehyde. It was in the course of this research that Kraepelin (1882b) had shown that increasing the amount of alcohol in the blood by having more drinks, led to a measurable lengthening of reaction time and proposed the use of dose-response comparisons in determining the clinical effects of a drug (Bech 2012). In the 8th edition of his textbook, published from 1909 to 1913, Kraepelin extended the scope of pharmacopsychology to the study of the psychotherapeutic effect of some drugs, such as chloral hydrate, morphine, and phenemal in psychiatric disorders. In 1920, the term, psychopharmacology, a synonym for pharmacopsychology was introduced by David Macht and in the years that followed virtually replaced the use of Kraepelin’s (1892) term.

References:

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- Müller U, Fletscher PC, Steinberg H. The origin of pharmacopsychology: Emil Kraepelin's experiments in Leipzig, Dorpat and Heidelberg (1882-1892). *Psychopharmacology*, 2006; 184:131-8.
- Steinberg H. Kraepelin in Leipzig. Eine Begegnung von Psychiatrie und Psychologie. Bonn: PsychiatrieVerlag; 2001.
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- Wundt W. Die Institut für experimentelle Psychologie zu Leipzig. *Psychologish Studien* 1910; 5:279-93.

February 13, 2014

Phasic psychoses (Thomas A. Ban)

The term “phasic psychoses” refers to a class of psychoses that is characterized by episodic course with full remission between episodes. The concept was introduced in 1949 by Edda Neele in the title of her monograph *Die phasischen Psychosen nach Ihrem Erscheinungs – und Erbbild* (“The Phasic Psychoses Based on Their Presentation and Family History”). In the same monograph (based on her thesis written for “habilitation”), she divided the “phasic psychoses” into “manic-depressive illness of affect,” “hyperkinetic-akinetic motility psychosis,” “excited-stuporous confusion psychosis” and “anxious-ecstatic delusional psychosis.” The concept of “phasic psychoses” was based on an evaluation of all phasic

sicknesses diagnosed at Karl Kleist's Neuropsychiatric Clinic at Goethe University, in Frankfurt, Germany between 1938 and 1942 (Teichman 1990). (See also Leonhard's phasic psychoses).

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Neele E. Die phasischen Psychosen mit Ihrem Erscheinungs – und Erbbild. Leipzig: J.A. Barth; 1949.

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June 25, 2014

PSC (Martin M. Katz)

The Psychopharmacology Service Center (PSC) was a program of the National Institute of Mental Health (NIMH). It was created by the National Institutes of Health (NIH) from the \$2 million appropriated by the US Congress in 1956 to initiate a grants program and national effort to stimulate research and treatment in the application of new psychotropic drugs. Jonathon Cole, a young psychiatrist, was appointed to lead the Center with the guidance of a National Advisory Committee, chaired by Ralph Gerard (Cole 2011; Katz 2011). The Center initiated a basic research grants program, conducted a nationwide Collaborative Project to evaluate the new drugs (NIMH Collaborative Studies in Psychopharmacology), created the Early Clinical Drug Evaluation Unit (ECDEU) network to develop new drugs, and published a new periodical, the *Psychopharmacology Bulletin*. The name of the Center was changed in 1965 and established at the NIMH as the Psychopharmacology Research Branch.

References:

Cole JO interviewed by Ban TA. In: Ban TA, editor. *An Oral History of Neuropsychopharmacology - The First Fifty Years: Peer Interviews, Volume 12- "History of the CINP"* (Martin M. Katz, volume editor). Nashville: American College of Neuropsychopharmacology; 2011. p. 45-53.

Katz MM interviewed by Ban TA. In: Ban TA, editor. *An Oral History of Neuropsychopharmacology - The First Fifty Years: Peer Interviews, Volume 12- "History of the CINP"* (Martin M. Katz, volume editor). Nashville: American College of Neuropsychopharmacology; 2011 p. 77-81.

October 30, 2014

Psychoanaleptics (Carlos R. Hojaij)

“Psychoanaleptics” are substances which stimulate mental activity. They are one of the three groups of drugs in the classification of “psychiatric drugs” proposed by Jean Delay (1959 a, b) first in 1957 at the Second World Congress of Psychiatry and subsequently in 1958 at the First Congress of the Collegium Internationale Neuro-Psychopharmacologicum. The term was adopted in Delay and Deniker’s (1961) monograph, *Méthodes Chimiothérapiques en Psychiatrie*, in which, “psychoanaleptics” were divided into “stimulants of vigilance” and “stimulants of affect” (Ban 1969).

References:

Ban TA. Psychopharmacology. Baltimore: William & Wilkins; 1969, p. 368.

Delay J. Intervention à propos de la terminologie et la classification des médicaments psychiatriques. In: Kline NS, editor. Neuropsychopharmacology Frontiers. New York: Little Brown; 1959a, pp. 426-9.

Delay J. Discussion: Fourth Symposium. In: Bradley PB, Deniker P, Radouco-Thomas C. Neuropsychopharmacology. Proceedings of the First International Congress of Neuro-Pharmacology. Amsterdam: Elsevier; 1959b, pp. 167-72

Delay J, Deniker P. Méthodes Chimiothérapiques en Psychiatrie. Paris; Masson et Cie; 1961, pp. 14-7.

February 6, 2014

Psychdysleptics (Carlos R.Hojaij)

“Psychdysleptics” are substances which disturb mental activity. They are one of the three groups of drugs in the classification of “psychiatric drugs” proposed by Jean Delay (1959a,b) first in 1957 at the Second World Congress of Psychiatry and subsequently in 1958 at the First Congress of the Collegium Internationale Neuro-Psychopharmacologicum. The term was adopted in Delay and Deniker’s (1961) monograph, *Méthodes Chimiothérapiques en*

Psychiatrie, in which, “psychoanaleptics” was defined as substances which disturb mental activity by their action that can be antagonized by various “psycholeptics” (Ban 1969). The term, was also adopted by the World Health Organization (WHO) Study Group in Research in Psychopharmacology in 1967, and in the WHO Technical Report Series Number 371, “psychodysleptics” were redefined as substances which produce abnormal mental phenomena, particularly in the “cognitive” and “perceptual spheres.”

References:

Ban TA. Psychopharmacology. Baltimore: William & Wilkins; 1969, p. 368.

Delay J. Intervention à propos de la terminologie et la classification des médicaments psychiatriques. In: Kline NS, editor. Neuropsychopharmacology Frontiers. New York: Little Brown; 1959a, pp. 426-9.

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Delay J, Deniker P. Méthodes Chimiothérapiques en Psychiatrie. Paris; Masson et Cie; 1961.

World Health Organization Study Group on Psychopharmacology. Technical Report Series No. 371. Geneva: World Health Organization; 1967.

March 6, 2014

Psycholeptic (Carlos C. Hojaij)

The term “psycholeptic” was coined by Pierre Janet (1906), who used it for “mental troubles which develop stormily reaching climax sufficiently quickly to constitute a veritable crisis.” Jean Delay (1959a,b) adopted the term for one of the three groups of substances he proposed to classify “psychiatric (psychotropic) drugs” first in 1957, at the Second World Congress of Psychiatry (WPA), and then in 1958, at the First Congress of the Collegium Internationale Neuro-Psychopharmacologicum (CINP). In their monograph, *Méthodes Chimiothérapiques en Psychiatrie*, published in 1961, Delay and Deniker defined “psycholeptics” as substances which produce relaxation and depress mental activity; and divided “psycholeptics” into “depressors of vigilance” and “depressors of affect” (Ban 1969).

References:

Ban TA. Psychopharmacology. Baltimore: William & Wilkins; 1969, p. 368.

Delay J. Intervention à propos de la terminologie et la classification des médicaments psychiatriques. In: Kline NS, editor. *Neuropsychopharmacology Frontiers*. New York: Little Brown; 1959a, pp. 426-9.

Delay J. Discussion: Fourth Symposium. In: Bradley PB, Deniker P, Radouco-Thomas C. *Neuropsychopharmacology. Proceedings of the First International Congress of Neuro-Pharmacology*. Amsterdam: Elsevier; 1959b, pp. 167-72.

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February 27, 2014

Psychopharmacology (Thomas A. Ban)

The term psychopharmacology was introduced in 1920 by David Macht, an American pharmacologist at Johns Hopkins University, in the title of his paper (“Contributions to psychopharmacology”), in which he studied the effects of ethanol, caffeine, bromine, opium alkaloids, and antipyretic analgesics on the “tapping speed test” (Berger 1976). Macht used the term as a synonym for pharmacopsychology, a term introduced by Kraepelin in 1892. Subsequently, the term was first used in psychiatry in 1935 by W.M. Thorner in the title of his paper “The psychopharmacology of sodium amytal,” published in the *Journal of Nervous and Mental Diseases*. The scope of psychopharmacology was gradually extended, first to research with psychomimetics (1940s), then to clinical investigations on the effects of psychotherapeutic drugs (end of the 1950s). In 1969, in Ban’s *Psychopharmacology*, it was defined as “a new scientific discipline which encompasses all the aspects and interactions between psychoactive drugs and biological systems”. In the years that followed, the all-embracing concept of psychopharmacology was deconstructed. In *An Oral History of Neuropsychopharmacology*, a series on The First Fifty Years in the history of the field, based on Peer Interviews, psychopharmacology is separated from behavioral pharmacology, neuropharmacology and neuropsychopharmacology, and restricted (in Volume Four - *Psychopharmacology*) to the discipline that studies the effects of centrally acting drugs on psychopathology and psychiatric diagnoses (Ban 2011a, b).

References:

Ban TA. Psychopharmacology. Baltimore: Williams & Wilkins; 1969, p. VII.

Ban TA, An overview of the series. In: Ban TA, editor. An Oral History of Neuropsychopharmacology. The First Fifty Years. Peer Interviews. Volume One (Starting Up, edited by Edward Shorter). Brentwood: American College of Neuropsychopharmacology; 2011, p. IX.

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Kraepelin E. Über die Beeinflussung einfacher psychischer Vorgänge durch einige Arzneimittel. Jena: Fischer; 1892.

Macht DL. Contributions to psychopharmacology. Bull Johns Hopkins Hospital 1920; 31:167-73.

Thorner MW. The psycho-pharmacology of sodium amytal. Journal of Nervous and Mental Diseases 1935; 81:161-7.

December 12, 2013

Psychopharmacology Service Center (Martin M. Katz)

The Psychopharmacology Service Center (PSC) was a program of the National Institute of Mental Health (NIMH). It was created by the National Institutes of Health (NIH) from the 2 million dollars appropriated by the US Congress in 1956 to initiate a grants program and national effort to stimulate research and treatment in the application of new psychotropic drugs. Jonathon Cole, a young psychiatrist, was appointed to lead the Center with the guidance of a National Advisory Committee, chaired by Ralph Gerard (Cole 2011; Katz 2011). The Center initiated a basic research grants program, conducted a nationwide Collaborative Project to evaluate the new drugs (NIMH Collaborative Studies in Psychopharmacology), created the Early Clinical Drug Evaluation Unit (ECDEU) network to develop new drugs, and published a new periodical, the *Psychopharmacology Bulletin*. The name of the Center was changed in 1965 and established at the NIMH as the Psychopharmacology Research Branch.

References:

Cole JO interviewed by Ban TA. In: Ban TA, editor. An Oral History of Neuropsychopharmacology - The First Fifty Years: Peer Interviews, Volume 12- "History of

the CINP” (Martin M. Katz, volume editor). Nashville: American College of Neuropsychopharmacology; 2011. p. 45-53.

Katz MM interviewed by Ban TA. In: Ban TA, editor. An Oral History of Neuropsychopharmacology - The First Fifty Years: Peer Interviews, Volume 12- "History of the CINP” (Martin M. Katz, volume editor). Nashville: American College of Neuropsychopharmacology; 2011. p. 77-81.

October 30, 2014

Psychotropic drugs (Thomas A. Ban)

The term “psychotropic drugs” first appeared in 1957, in Ralph Gerard’s paper, “Dugs for the soul; the rise of psychopharmacology,” published in *Science*, and in the title of the First International Symposium on Psychotropic Drugs, held in Milan in May 1957 (Garattini and Ghetti 1957). In his paper, Gerard defined psychotropic drugs as substances which possess “psychic tropism” and are capable of modifying mental activity (Ban 1969). The term was adopted into French by Jean Delay (1959), who first referred to psychiatric drugs, as “drogues psychotropes,” in his discussion of the fourth symposium (“Comparison of Drug Induced and Endogenous Psychoses”) at the First Congress of The Collegium Internationale Neuro-Psychopharmacologicum (CINP), held in 1958 in Rome (Italy).

References:

Ban TA. Psychopharmacology. Baltimore: Williams & Wilkins; 1969, p. 365.

Delay J. Discussion (Fourth Symposium). In: Bradley PB, Deniker P, Radouco-Thomas C. Neuropsychopharmacology. Proceedings of the First Congress of Neuro-Pharmacology. Amsterdam: Elsevier; 1959, pp. 167-72.

Garattini S, Ghetti V, editors. Psychotropic Drugs. Amsterdam: Elsevier; 1957.

Gerard R. Drugs for the soul; the rise of psychopharmacology. *Science* 1957; 125: 201-3.

February 20, 2014

[Comment by Carlos R. Hojaij]

The years succeeding 1952 (chlorpromazine) set the stage for “a rapid multiplication of drugs and psychic medications,” with different actions. This observation by Jean Delay and his concern that certain degree of confusion could have a negative impact on research and clinic, led him to search for common terminology and classification. It was then, in 1957, during a Symposium of Psychopharmacology for the occasion of the 2nd International Congress of Psychiatry in Zurich and not in the 1st Congress of the Collegium Internationale Neuro-Psychopharmacologicum, held in 1958, in Rome -- as Tom Ban suggests -- that Jean Delay first used and adopted the term psychotropic and proposed a classification of these drugs based on their main effect in “human clinic.” For Jean Delay (1957) the term psychotropic was valid since it was a general term to include all chemical substances, being natural or pharmaceutically made, which have a “psychological tropism” capable of interfering in mental activity, not considering a priori the kind of modification to be promoted.

Reference:

Delay J. Intervention à propos de la terminologie et la classification des médicaments psychiatriques. In: Kline NS, editor. Neuropsychopharmacology Frontiers. New York: Little Brown; 1959, pp. 426-9.

March 13, 2014

QPRA (Thomas A. Ban)

The QPRA, or Quebec Psychopharmacological Research Association, was founded on the initiative of Thomas A. Ban in October 1963 by a group of about 20 people involved in psychopharmacological research in the Province of Quebec, Canada. At the same meeting, held in the Medical Library of the Verdun Protestant Hospital (Douglas Hospital), in Verdun, Heinz E. Lehmann was elected president and Thomas A. Ban, executive secretary of the Association. The primary objective of the Association was the improvement of standards in clinical psychopharmacological research. Another objective was the facilitation of communication of research findings by the organization of symposia and colloquia. In the 15 years that followed, the Association held meetings with some regularity. The proceedings of six of these meetings were published in books (Lehmann and Ban 1964, 1965, 1968; Lehmann, Berthiaume and Ban 1964; Villeneuve 1976; Villeneuve and Bordeleau 1973). The Association was dissolved at the

end of 1980 with its remaining funds, approximately \$2,000, transferred to the Canadian College of Neuropsychopharmacology (Ban 2004).

References:

Ban TA. The history of the Quebec Psychopharmacological Research Association. Budapest: Animula; 2004, pp.621-3.

Lehmann HE, Ban TA, editors. The Butyrophenones in Psychiatry. Montreal: Quebec Psychopharmacological Research Association; 1964.

Lehmann HE, Ban TA, editors. Toxicity and Adverse Reaction Studies with Neuroleptics and Antidepressants. Montreal: Quebec Psychopharmacological Research Association; 1965.

Lehmann HE, Ban TA. The Thioxanthenes. Basel: Karger; 1968.

Lehmann HE, Berthiaume M, Ban TA, editors. Trimipramins, A New Antidepressant. Montreal: Quebec Psychopharmacological Research Association; 1964.

Villeneuve A. Proceedings of the First Canadian International Symposium on Lithium. Quebec: Les Presses de l'Université Laval; 1976.

Villeneuve A, Bordeleau J-M, editors. The Diphenylbutylpiperidines. Quebec: Les Presses de l'Université Laval; 1973.

April 9, 2015

Quebec Psychopharmacological Research Association (Thomas A. Ban)

The Quebec Psychopharmacological Research Association, or QPRA in brief, was founded on the initiative of Thomas A. Ban in October 1963 by a group of about 20 people involved in psychopharmacological research in the Province of Quebec, Canada. At the same meeting, held in the Medical Library of the Verdun Protestant Hospital (Douglas Hospital), in Verdun, Heinz E. Lehmann was elected president and Thomas A. Ban, executive secretary of the Association. The primary objective of the Association was the improvement of standards in clinical psychopharmacological research. Another objective was the facilitation of communication of research findings by the organization of symposia and colloquia. In the 15 years that followed, the Association held meetings with some regularity. The proceedings of six of these meetings were published in books (Lehmann and Ban 1964, 1965, 1968; Lehmann, Berthiaume and Ban 1964; Villeneuve 1976; Villeneuve and Bordeleau 1973). The Association

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Ban TA. The history of the Quebec Psychopharmacological Research Association. Budapest: Animula; 2004, pp. 621-3.

Lehmann HE, Ban TA, editors. The Butyrophenones in Psychiatry. Montreal: Quebec Psychopharmacological Research Association; 1964.

Lehmann HE, Ban TA, editors. Toxicity and Adverse Reaction Studies with Neuroleptics and Antidepressants. Montreal: Quebec Psychopharmacological Research Association; 1965.

Lehmann HE, Ban TA. The Thioxanthenes. Basel: Karger; 1968.

Lehmann HE, Berthiaume M, Ban TA, editors. Trimipramins, A New Antidepressant. Montreal: Quebec Psychopharmacological Research Association; 1964.

Villeneuve A. Proceedings of the First Canadian International Symposium on Lithium. Quebec: Les Presses de l'Université Laval; 1976.

Villeneuve A, Bordeleau J-M, editors. The Diphenylbutylpiperidines. Quebec: Les Presses de l'Université Laval; 1973.

March 12, 2015

Samuel Gershon Award for Young Investigators (Samuel Gershon)

These awards were initiated in 2006 and given to four candidates by an international competition of submissions each year. They are funded by the International Society of Bipolar Disorders.

October 15, 2015

Samuel Gershon Medal (Samuel Gershon)

The Samuel Gershon Medal for Lifetime Achievement in Translational Neuroscience was created in honor of Samuel Gershon by the Mind and Body Theme (Head: Julio Licinio), South Australian Health and Medical Research Institute (SAHMRI), to celebrate lifetime

achievement in translational neuroscience. The Medal was first presented on June 27, 2014, during the Translational Neuroscience Day, hosted by SAHMRI's Mind and Body Theme, at the Institute, in Adelaide, Australia.

June 4, 2015

Sejuncton hypothesis (Marcelo Cetkovich)

“Sejuncton hypothesis” (Franzek 1990; Pichot 1983; Shorter2005), also referred to as “sejuncton theory” (Pichot 1983; Shorter 2005), postulates that psychopathological symptoms result from interruption (“sejunction”) of associative connections in the brain. It was put forward by Carl Wernicke, in 1900, in the 12th lecture of his Textbook of Clinical Lectures in Psychiatry (Wernicke 1900). The “hypothesis” is conceptually derived. It is built on Wernicke’s adoption of Griesinger’s “psychic reflex” as the basis of mental activity and his notion that the nature of psychopathology is determined by the site of an assumed severance in the path of the “psychic reflex” (Griesinger 1843; Wernicke 1906).

References:

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- Griesinger W. Ueber psychische Reflexactionen. *Archiv fuer Physiologische Heilkunde*. 1843; 2:76-112.
- Pichot P. *A Century of Psychiatry*. Paris: Roger de Costa; 1893, p. 56.
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October 1, 2015

[Comment by Hector Warnes]

Congratulation to Marcelo Cetkovich for his concise entry of Wernicke's "sejunction theory/hypothesis" into INHN's Dictionary. It might be of interest that Otto Hans Adolf Gross in his paper *Dementia Sejunctiva*, published in the early years of the 20th century, just a few years after the "theory" was introduced, referred to "sejunction" as a "closed circuit" of associative ties characterized by a loss of certain associations assumedly caused by an interruption of neuronal pathways. He went even further by suggesting that "sejunction" could explain fragmentation of the thinking process with collapse of several functionally separate series of associations and a break in the continuity of temporal memory (Gross 1904).

It might be also of interest that Karl Jaspers understood "sejunction" as the underlying pathophysiology of a variety of "psychic disturbances." In his *General Psychopathology*, he wrote: "The basis of the majority of psychic disturbances lies primarily in the parting of the association-links or sejunction. Where there are false ideas or judgements in an individual or they are in conflict with each other or with reality this is thought to be due to a 'loosening up' in the firm network of associations. By severing the continuity tracks, by an absence of certain associative performances a number of different personalities may simultaneously arise in the same individual and a 'break up' of individuality occur. Sejunction can also explain a large number of hallucinations... if association is interrupted, excitation processes are dammed up and thus a progressively increasing stimulus is established which brings the hallucinations about. Similarly 'autochthonous ideas' (the so called 'made thoughts') are due to a process of irritation when continuity is interrupted whereas compulsive thinking is explained by a process of irritation while continuity is preserved. Abnormal movements (parakinesis) are also due to these sejunctions. Because hallucinations are due to sejunction, Wernicke finds it quite feasible that they are without any counter-image and therefore there is no criticism of them; also that they so often have contents of an imperative character....." (Jaspers 1963).

References:

Gross OHA. *Dementia Sejunctiva*. *Neurologisches Zentralblatt* 1904; 23:1144-6.

Jaspers K. *General Psychopathology*- (translated by J. Hoenig and Mariam W. Hamilton). Manchester: Manchester University Press; 1963.

January 21, 2016

Sejuncton theory (Marcelo Cetkovich)

“Sejuncton theory” (Pichot 1983; Shorter2005), also referred to as “sejuncton hypothesis” (Franzek 1990), postulates that psychopathological symptoms result from interruption (“sejunction”) of associative connections in the brain. It was put forward by Carl Wernicke, in 1900, in the 12th lecture of his *Textbook of Clinical Lectures in Psychiatry* (Wernicke1900). The “theory ” is conceptually derived. It is built on Wernicke’s adoption of Griesinger’s “psychic reflex” as the basis of mental activity and his notion that the nature of psychopathology is determined by the site of an assumed severance in the path of the “psychic reflex” (Griesinger 1843; Wernicke 1906).

References:

- Franzek E. Influence of Carl Wernicke on Karl Leonhard’s nosology. *Psychopathology* 1990; 23:277-81
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- Wernicke C. *Grundriss der Psychiatrie in klinische Vorlesungen*. 2 Auflage. Leipzig: Thieme; 1900, p. 109.

September 17, 2015

Stereotypy (Carlos A. Morra)

Persistent repetition of one invariable form of gesture, act or verbal expression without an identifiable objective. The term was introduced in 1864 by Jean-Pierre Falret in his textbook

Les Maladies Mentales et des asiles d'aliénés. It was derived by clinical observation (Guiraud 1936).

References:

Falret JP. *Les Maladies Mentales et des asiles d'aliénés.* Paris: J.B. Ballière et Fills; 1864, pp. 193-4.

Guiraud P. Analyse du symptôme stéréotypie, avec trois planches hors-texte. *Encéphale*, 1936; 31:229-70.

October 19, 2017

Stimulants of affect (Carlos R. Hojaij)

“Stimulants of affect” are one of the two groups of “psychanaleptics” in Delay’s (1959a,b) classification of “psychiatric drugs.” They were defined in Delay and Deniker’s monograph published in 1961 as substances which regulate the oscillation of “mental tone” between “apathetic,” or under-responsive, and “pathetic” or over-responsive, with the potential by their action to replace an “apathetic tone” by a “pathetic” one. Included among the “stimulants of affect” are all antidepressants (Ban 1969).

References:

Ban TA. *Psychopharmacology.* Baltimore: William & Wilkins; 1969, p. 368.

Delay J. Intervention à propos de la terminologie et la classification des médicaments psychiatriques. In: Kline NS, editor. *Neuropsychopharmacology Frontiers.* New York: Little Brown; 1959a, pp. 426-9.

Delay J. Discussion: Fourth Symposium. In: Bradley PB, Deniker P, Radouco-Thomas C. *Neuropsychopharmacology. Proceedings of the First International Congress of Neuro-Pharmacology.* Amsterdam: Elsevier; 1959b, pp. 167-72.

Delay J, Deniker P. *Méthodes Chimiothérapeutiques en Psychiatrie.* Paris: Masson et Cie; 1961, p.15.

January 9, 2014

Stimulants of vigilance (Carlos R. Hojaij)

“Stimulants of vigilance” are one of the two groups of “psychoanaleptics” in Delay’s (1959a,b) classification of “psychiatric drugs.” They are defined in Delay and Deniker’s monograph, *Méthodes Chimiothérapiques en Psychiatrie*, published in 1961, as substances which increase alertness, intellect and noetic activity by stimulating arousal. Included among these substances are the “cortical stimulants,” as caffeine, and the “adrenergic activators,” as methylphenidate (Ban 1980).

References:

Ban TA. Psychopharmacology for the Aged. Basel: Karger; 1980, pp. 42-7.

Delay J. Intervention à propos de la terminologie et la classification des médicaments psychiatriques. In: Kline NS, editor. *Neuropsychopharmacology Frontiers*. New York: Little Brown; 1959a, pp. 426-9.

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Delay J, Deniker P. *Méthodes Chimiothérapiques en Psychiatrie*. Paris; Masson et Cie; 1961, p. 15.

January 2, 2014

Symposia Medica Hoechst (Jules Angst)

The Symposia Medica Hoechst were sponsored by the Medical Department of Hoechst Company, which also published the symposia proceedings. The meetings were organized by the chairmen of the symposia, usually a leading authority in a medical specialty, together with Dr Elke Lindenlaub from Hoechst. The venue was the Castle of Reinhartshausen on the Rhine. The first Hoechst Symposium, on Causal Factors of Myocardial Infarction, was held in 1968 and the report published 1969 (Schettler 1969). The 8th symposium (1973) was devoted to a psychiatric topic: Classification and Prediction of Outcome in Depression. It was organized by Jules Angst and attended by 33 experts from America, Europe and Australia. The presentations and discussions of this symposium were published in 1974 (Angst 1974). The last Hoechst

Symposium, on the Biology of Memory, was held in 1988 and its report was published in 1990 (Squire and Lindenlaub 1990).

References:

Angst J, editor. Classification and Prediction of Outcome of Depression. Stuttgart/New York: Schattauer; 1974.

Schettler G, editor. Causal Factors of Myocardial Infarction. Stuttgart/New York: Schattauer; 1969.

Squire LR, Lindenlaub E, editors. The Biology of Memory. Stuttgart/New York: Schattauer; 1990.

October 9, 2014

Synapse (Thomas A. Ban)

Structure that permits a neuron (nerve cell) to pass an electrical or chemical signal to another neuron. The term, “synapse” -- derived from the Greek term, “synapsis” with the meaning “conjunction” -- was introduced by Michael Foster, in 1897, in the 3rd volume of the 7th edition of his *Textbook of Physiology* that was revised by Charles Scott Sherrington (Foster and Sherrington 1897; Shorter 2005).

References:

Foster M, Sherrington CS. Textbook of Physiology. Volume 3, 7th edition. London: Macmillan; 1897, p. 929.

Shorter E. Historical Dictionary of Psychiatry. Oxford/New York: Oxford University Press; 2005, p. 288.

March 3, 2016

Synthetic enhancer substance (Joseph Knoll)

The term, “synthetic enhancer substance” refers to a manufactured copy of “an endogenous enhancer substance,” which increases the activity of special neurons which are

sensitive to the natural substance. The term was coined by Joseph Knoll in his monograph *The Brain and Its Self* published in 2005. The prototype of “synthetic enhancer substances” is deprenyl, a manufactured copy of the “natural enhancer substance,” β -phenylethylamine (PEA) which, similar to PEA, increases the activity of catecholamine producing neurons. Introduction of the concept of “synthetic enhancer substance” was based on Knoll and Miklya’s findings in 1994 that subcutaneous administration of deprenyl in a dose range of 0.01- 0.1 mg/kg daily for 21 days, increased statistically significantly ($P < 0.001$) both dopamine and norepinephrine levels in the striatum, substantia nigra and tuberculum olfactorium and locus coeruleus, respectively (Knoll and Miklya 1994).

References:

Knoll J. *The Brain and Its Self. A Neurochemical Concept of the Innate and Acquired Drives.* Springer, Berlin/Heidelberg/New York, 2005. pp. 25-94

Knoll J, Miklya I. Multiple small dose administration of (-)-deprenyl enhances catecholaminergic activity and diminishes serotonergic activity in the brain and these effects are unrelated to MAO-B inhibition. *Archives internationales Pharmacodynamie de Therapie.* 1994; 328: 1-15

January 15, 2015

Tyramine oxidase (Joseph Knoll)

Tyramine oxidase is the enzyme responsible for the oxidative deamination of tyramine. It was the first enzyme for oxidative deamination that was found to be present in the body. Research for the detection of the enzyme responsible for oxidative deamination began, in 1877, with Oswald Schmiedeberg’s findings that orally administered benzylamine, a monoamine, to dogs, was deaminated and excreted in the urine as benzoylglycin (hippuric acid). It continued in 1910 by Ewins and Laidlaw’s demonstration that both endogenous monoamines, tyramine, a phenylalkylamine, and tryptamine, and indoleamine, were deaminated and excreted in the urine as p-hydroxyphenylacetic acid and indoleacetic acid, respectively. However, it was only 28 years later, in 1928, that Mary Hare had shown the presence of “tyramine oxidase,” the enzyme responsible for oxidative deamination of the monoamine, tyramine, in the liver. Today, the term “tyramine oxidase” is of history. In 1937, Blaschko, Richter and Schlossman discovered that tyramine oxidase, noradrenaline oxidase and aliphatic amine oxidase are the

same enzyme, and in 1938, by Zeller's separation of diamine oxidase from "amine oxidase," tyramine oxidase became part of the monoamine oxidase enzyme system.

References:

Blaschko H, Richter D, Schlossmann H. The oxidation of adrenaline and other amines. *Biochemical Journal* 1937; 31: 2187-96

Ewins AJ, Laidlaw PP. The fate of parahydroxyphenylethylamine in the organism. *Journal Physiology* 1910; 41:78-87.

Hare MLC. Tyramine oxidase I. A new enzyme system in the liver. *Biochemical Journal* 1928; 22:968-79.

Schmiedeberg O. Über das Verhältniss des Ammoniaks und der primären Monoaminbasen zur Harnstoffbildung im Thierkörper. *Archiv für experimentelle Pathologie und Pharmakologie* 1877; 8:1-14.

Zeller EA. Über den enzymatischen Abbau von Histamin und Diaminen. *Helvetica Chimica Acta* 1938; 21:881-90

June 5, 2014

Wimmer's psychogenic psychoses (Thomas A. Ban)

An independent group of psychoses caused by mental trauma on a predisposed foundation, which determine from the start the fluctuations in clinical manifestations and often their cessation. The diagnostic concept was introduced in 1916 by August Wimmer.

Reference:

Wimmer A. *Psykogene Sindssygdomsformer*. Copenhagen: Jacob Lund; 1916.

February 11, 2016

July 16, 2020