Thomas A. Ban Psychopharmacology and the Classification of Functional Psychoses

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Prologue (2020)

Simultaneously, with the availability of psychotropic drugs with demonstrable efficacy for mental illness there was a shift in the understanding of the nature of synaptic transmission in the brain from a purely electrical to a chemically mediated event. By the end of the 1950s seven neurotransmitters had been identified in the central nervous system (Ban 2006).

Recognition of chemical mediation at the site of the synapse, coupled with the introduction of the spectrophotofluorometer, an instrument with a resolution power to detect drug-induced changes in the concentration of neurotransmitter monoamines and their metabolites, led to the development of *neuropharmacology*, a branch of pharmacology that deals with the detection and identification of structures responsible for the psychotropic effects of centrally acting drugs (Bowman, Caulfield and Udenfriend 1955). Previously, research dealing with centrally acting drugs was restricted to behavioral pharmacology and neurophysiological measures, spectrophotofluorometry provided direct access to the detection of the biochemical changes that might be responsible for therapeutic effects. Spectrophotofluorometry also opened the path for the development of *neuropsychopharmacology*, a new discipline that studies the relationship between neuronal and mental processing in the brain with the employment of centrally acting drugs (Ban 2004).

The first neuropharmacological studies with the aid of spectrophotofluorometry revealed: (1) that administration of reserpine, a Rauwolfia alkaloid, produced a decrease in brain serotonin (5HT) and norepinephrine (NE) levels in the brain (Holzbauer and Vogt 1956; Pletscher, Shore and Brodie 1955); (2) that administration of iproniazid, an inhibitor of the monoamine oxidase (MAO) enzyme, responsible for the breakdown of monoamines like 5HT and NE (Zeller, Barsky, Fouts et al. 1952), increased brain serotonin levels (Pletscher 1956); (3) that pre-treatment with iproniazid attenuated reserpine-induced depletion of 5HT and NE, a catecholamine (Besendorf

and Pletscher 1956; Carlsson, Rosengren, Bertler and Nilsson 1957); and (4) that only those Rauwolfia alkaloids like reserpine (Brodie, Pletscher and Shore 1956) and benzoquinolizines (Pletscher 1957) (a group of synthetic substances) that depleted 5HT, had tranquilizing or sedating action.

Instrumental for opening up neuropsychopharmacological research was the postulation of a relationship between mood and cerebral monoamine levels. It was based in part on clinical reports which indicated that treatment with iproniazid induced euphoria, a feeling of well-being in some tubercular patients (Flaherty 1952; Selikoff, Robitzek and Orenstein 1957), whereas treatment with reserpine induced depressed mood or dysphoria in about 15% of hypertensive patients in treatment with the drug (Freis 1954; Mueller, Pryor, Gibbson and Orgain 1955).

The "birth" of neuropsychopharmacology in the mid-1950s was the result of combining these clinical observations with findings in neuropharmacological research (Ban 2008).

The foundation of the new discipline was tenuous. There were reports that isoniazid, the parent substance of iproniazid, had similar effects on mood to those of iproniazid without virtually any effect on MAO activity (Delay, Laine and Buisson 1952: Salzer and Lurie 1953, 1955). There was also a report on the favorable rather than dysphoric effects of reserpine in anxious and depressed patients (Davies and Shepherd 1955; Healy 1997).

One of the first to recognize that neuropsychopharmacology opened a new perspective in the understanding of psychiatric illness was Abraham Wikler, an American psychiatrist (Hollister 1996). In his monograph, *The Relation of Psychiatry to Pharmacology*, he suggested that studying the mode of action of psychotropic drugs with known therapeutic effects might provide information on the biochemical basis of mental disorders (Wikler 1957). By generating information on molecular changes in psychiatric illness, findings in neuropsychopharmacological research were to guide the development of rational drug treatments.

The notion that drugs with known therapeutic effects might provide the key for bridging the gap between neuronal processing and mental pathology has remained the driving force for research in the new field. The target of the drive has been the identification of the missing links: pharmacologically homogeneous populations of mental illness.

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Preface (circa 1985)

Recommended reading

Introduction

Multi-Axial Classifications

Empirical, Experimental and Nosology-derived Classifications

From One to Four Dimensional Classifications

Classification of Mental Disorders and Psychopharmacology

Psychopharmacology

ICD-9 of the World Health Organization

DSM-3 of the American Psychiatric Association

Diagnostic Criteria for Research

St. Louis Criteria

Research Diagnostic Criteria

Vienna Research Criteria

The KDK Budapest

Diagnostic Assessment Scale (DAS)

One- and Two-Dimensional Diagnoses

Conceptual Development

One-Dimensional Diagnosis

Concept of Psychosis

Organic vs Functional Psychosis

Acute Exogenous Predilectional Types

Delirium, Dysmnesia and Dementia

Two-Dimensional Diagnoses

Functional Psychosis: Reactive and Endogenous

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Further Elaboration

Delusional Development

Delusional Psychoses

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Psychopharmacological Implications

Bayle's Unitary Concept of Psychosis

Endogenous Psychoses

From Kahlbaum to Kraepelin

From Kraepelin to Leonhard via Kleist

Nyiro's Structural Psychopathology

Endogenous Psychoses in the KDK

Validity of KDK (Leonhard's) Diagnoses

Affective Psychoses

Manic-Depressive Illness

Melancholia and Depression

Separation of Vital Depression

Empirical and Pragmatic Classifications of Depression

Genetic and Experimental Classifications of Depression

Nosology Derived Classifications of Depression

Psychopharmacology of Depressions

Tricyclic Antidepressants

Monoamine Oxidase Inhibitors

Pharmacotherapy of Depression

Mania and Euphorias

Schizophrenic Psychoses

From Dementia Praecox to Schizophrenia

From Schizophrenia to the Schizophrenias

Non-Systematic and Systematic Schizophrenias

Systematic Schizophrenias

Systematic Catatonias

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Systematic Hebephrenias

Non-Systematic Schizophrenias

Treatment of Schizophrenic Disorders

Pharmacotherapy versus Other Treatments

Pharmacotherapy with Neuroleptic-Antipsychotics

Neuroleptics and Etiological Considerations

Dopamine Excess or Deficiency

Prostaglandin Deficiency or Excess

Endorphins: Excess or Deficiency

Neuroleptics: Long-term Effects

Neuroleptics and Leonhard's Classification

Cycloid Psychoses

Confusion Psychosis

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Anxiety-Happiness Psychosis

Differential Characteristics

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Course of Illness

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Appendix

References

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