The Biopsychiatric Model of "Mental Illness"

A Critical Bibliography

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Presented below is an annotated bibliography addressing today's widely held belief system about the causes and treatment of disturbed and disturbing behavior usually labeled as some form of serious mental illness. As "schizophrenia" is psychiatry's most vexing and perplexing "disorder" and viewed as the most serious of the "mental illnesses" it is the primary focus of this list. It excludes children. It is not exhaustive, but is representative.

Conclusions: Today's dominant theory of serious"mental illnesses" posits them to be genetically determined (i.e., inherited), biochemically mediated (via "chemical imbalances"), life-long"brain diseases"(with associated specific neuropathologic changes) whose cause(s) and course is more or less independent of environmental factors ***is not supported by existing evidence***. A critical review of the scientific available evidence reveals no clear indication of hereditary factors, no specific biochemical abnormalities, and no associated causal neurologic lesion(s). However, a number of environmental factors have been found to be related to their cause(s) and course(bibliography in preparation). It is also generally held that the anti-psychotic drugs are the mainstay of treatment and should, in most cases, be taken for a lifetime. In fact, the data indicate that neuroleptic drug treatment is not usually necessary (especially in persons newly identified as psychotic) if a proper interpersonal environment and social context is provided in alternatives to hospital care. It also appears that has drug treatment has resulted in less favorable long-term outcomes than was the case before anti-psychotic drugs were introduced. Furthermore, anti-psychotic drug treatment is associated with the induction of irreversible brain pathology (resulting in reduced intellectual and abnormal motor functioning) and shortened life expectancy. Pre-neuroleptic drug era long-term follow-up studies indicate that ***recovery can not only occur, but is to be expected in the majority of cases***. Ergo, so called "chronicity" in "mental illness" is likely the result of its medicalization, institutionalization with its social network disruption, marginalization, discrimination and the less specific social consequences (e.g. poverty) that accompany these processes.

General:

Harding, C. M. and Zahniser, J.M. (1994) Empirical Correction of Seven Myths about Schizophrenia with Implications for Treatment. *Acta Psychiatrica Scandinavica*. **90**(suppl.384): 140-146.

Colin Ross & Alvin Pam. (1995). *Pseudo-science in biological psychiatry: Blaming the body*. NY, John Wiley

Van Praag, Herman. (1993). *"Make-believes" in psychiatry, or the perils of progress*. Clinical and Experimental Psychiatry Monograph No. 7. New York: Brunner/Mazel.

Siebert, A. (1999) Brain Disease Hypothesis Disconfirmed by All Evidence. *J. of Ethical Human Sciences and Services*. **1**(2) 179-199.

Valenstein, E (1998) *Blaming the Brain: the truth about drugs and mental illness*. NY, Free Press.

Genetics:

Barondes, S. et al (1999) An Agenda for Psychiatric Genetics. *Arch. Gen. Psych*. **56**: 549-552. ("genetically influenced psychiatric disorders have so far been resistant to analysis")

Joseph, J. (1998). The equal environment assumption of the classical twin method: A critical analysis. *Journal of Mind and Behavior*, **19**, 325-358. (Joseph points out that all twin studies of behavioral characteristics-like those defining "schizophrenia" are fundamentally flawed because identical twins have been clearly shown to be raised more similarly than are non-identical ones. Hence, higher rates of the co-existence of "schizophrenia" among identical twins can be explained by their having been raised in more similar environments. Even then their rates run only about 35%vs.10% for non-identicals)

Joseph, J. (1999). A critique of the Finnish Adoptive Family Study of Schizophrenia. *Journal of Mind and Behavior*, **20**, 133-154. (Joseph points out that the adoption study methodology depends on random adoption-that is the adoption agency does not know the mother's background when placing the child. The Finnish study, the most elegant and sophisticated of all, suffers from the fact that the first half of the sample was placed with the knowledge the mothers had "schizophrenia". The widely quoted Danish adoption studies are plagued with this and a number of other important methodological problems making their findings highly questionable.)

Joseph, J. (1999). The genetic theory of schizophrenia: A critical overview. *Ethical Human Sciences and Services*, **1**, 119-145. (Conclusion: there is no evidence of a specific or important genetic component in "mental illness")

Neuropathology:

Chua, S. E. and McKenna, P.J. (1995) Schizophrenia-a Brain Disease? A critical review of structural and functional cerebral abnormality in the disorder. *Brit. Jour. Psych.*, **166**: 563-582. (no consistent specific structural or functional abnormalities found).

Zakzanis, K. et al (2000) Searching the Schizophrenic Brain for Temporal Lobe Deficits: a systematic review and meta-analysis. *Psychol. Med.*, **30**: 491-504. (No specific findings).

Brain Damage Associated with Neuroleptic Drug Treatment:

Ballesteros J, Gonzales-Pinto A, & Bulbena A. Tardive dyskinesia associated with higher mortality in psychiatric patients: results of a meta-analysis of seven independent studies. *J Clin Psychopharmacology*, **20**:2, 188-194, 2000.

E Christensen. "Neuropathological investigations of 28 brains from patients with dyskinesia." *Acta Psychiatrica Scandinavica*, **46**,14-23, 1970. (TD patients have structural abnormalities in the basal ganglia, enlarged ventricles, and sulcal markings.)

OO Famuyiva. Tardive dyskinesia and dementia. *British Journal of Psychiatry*, **135**, 500-504, 1979. (TD associated with cognitive impairment.)

JT Wegner. Cognitive impairment in tardive dyskinesia. *Psychiatry Research*, **16**, 331-337. 1985. (TD associated with cognitive impairment.)

James Wade. Tardive Dyskinesia and Cognitive Impairment. *Biological Psychiatry*, **22**, 393-395, 1987. (Association between TD and cognitive impairment. "The relationship appears to be linear: individuals with severe forms of the disorder are most impaired cognitively.")

JL Waddington. Cognitive dysfunction, negative symptoms, and tardive dyskinesia in schizophrenia. *Archives of General Psychiatry*, **44**, 907-912, 1987. (TD associated with cognitive impairment and worsening of negative symptoms.)

Waddington J et al, Mortality in schizophrenia: Antipsychotic polypharmacy and absence of adjunctive anticholinergics over the course of a 10-year prospective study, *Br J Psych*, 1998, **173**; 325-329. (This study found that a reason that schizophrenics have a shorter life expectancy was neuroleptic drug treatment)

JB Wade. Cognitive changes associated with tardive dyskinesia. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology*. **1**, 217-227. 1989. (TD associated with cognitive impairment. The researchers conclude: "TD may represent both a motor and dementing disorder.")

R. Yassa. Functional impairment in tardive dyskinesia: medical and psychosocial dimensions. *Acta Psychiatr Scand* **80**, 64-67. 1989. (TD associated with gait, speech difficulties, and psychosocial impairment.)

Michael S. Myslobodsky. Central Determinants of Attention and Mood Disorder in Tardive Dyskinesia (Tardive Dysmentia.). *Brain and Cognition*, **23**, 88-101. 1993. (TD patients lose the motor part of their "road map of consciousness." TD may represent "larval dementia.")

Herbert Spohn. The effect of attention/information processing impairment of tardive dyskinesia and neuroleptics in chronic schizoprhenics." *Brain and Cognition* **23**, 28-39, 1993. (TD exacerbates cognitive impairment.)

Jacinthe Baribeau. Tardive dyskinesia and associated cognitive disorders: a convergent neuropsycological and neurophysiological approach. *Brain and Cognition* **23**, 40-55, 1993. (TD associated with cognitive dysfunction.)

John Waddington. Cognitive dysfunction in schizophrenia: organic vulnerability factor or state marker for tardive dyskinesia? *Brain and Cognition* **23**, 56-70, 1993. (He reviews 22 studies from 1979 to 1991 that concluded that patients with TD were cognitively impaired on a variety of measures, which include learning, memory, cognitive function, intellectual function, visual retention, orientation, etc.)

James Wade. Factors related to the severity of tardive dyskinesia. *Brain and Cognition* **23,** 71-80, 1993. (A review of research shows that "biochemical and neuropathological changes associated with TD indicates that similar alterations are associated with Hungtington's disease and or Parkinson's." In their own research, "cortical dysfunction, characterized by impairment in nonverbal function, is associated with TD severity.")

Emmanuelle Pourcher. Organic brain dysfunction and cognitive deficits in young schizophrenic patients with tardive dyskinesia. *Brain and Cognition* **23**, 81-87, 1993. (This is a study of patients under 40. They find that TD is associated with cerebral dysfunction, which in turn is associated with exposure to neuroleptic drugs.)

Thomas Gualtieri. The problem of tardive akathisia. *Brain and Cognition* **23**, 102-109, 1993. (He states that tardive akathisia may be thought of as a disease of the basal ganglia, much like Parkinson's, Huntington's and Wilson's. MRI studies have demonstrated basal ganglia lesions in TD patients, especially in the caudate nucleus. Basal ganglia diseases all cause behavioral instability and intellectual impairment (even psychosis and dementia)).

Miranda Chakos. Increase in Caudate Nuclei Volumes of First-Episode Schizophrenic Patients Taking Antipsychotic Drugs. *Am Jour Psych* **151**, 1430-1435. 1994. (Neuroleptics increase caudate volumes 5.7% during first 18 months of treatment in first-episode schizophrenic patients. Higher dosage is associated with larger increase in caudate volumes.)

J.S. Paulsen. Neuropsychological impairment in tardive dyskinesia. *Neurospsychology* **8**, 227-241. 1994. (Review of 31 studies that compared cognitive function in schizophrenics with and without TD. In 24 studies, TD patients were found to do worse. The more severe the TD, the greater the impairment in cognitive function. They conclude that "TD involves an alteration of brain function that affects both motor and cognitive control.")

P. Sachdev. Negative symptoms, cognitive dysfunction, tardive akathisia and tardive dyskinesia." *Acta Psychiatr Scand*. **93**, 451-459. 1996. (Both tardive akathisia and tardive dyskinesia are associated with more cognitive deficits and negative symptoms. This association is stronger with TA than with TD. The implication is that movement disorders seen in TA and TD are "but one feature of complex syndromes that include motor and cognitive features. A comparison must be made with other movement disorders, such as Parkinson's disease and Huntington's disease, in which neuropsychological deficits, and indeed subcortical dementia are known to occur.")

John Waddington. Cognitive dysfunction in chronic schizophrenia followed prospectively over 10 years and its longitudinal relationship to the emergence of tardive dyskinesia. *Psychological Medicine*, **26**, 681-688. 1996. (Progressive deterioration in cognitive function is seen even late in chronic phase of schizophrenic illness. Deterioration derives primarily from emergence of TD. They find that marked deterioration in cognitive function occurs at same time as emergence of movement disorder.)

Rupert McShane. Do Neuroleptic Drugs Hasten Cognitive Decline in Dementia? Prospective Study with Necropsy Follow Up. *British Medical Journal*, **314**, 266-270. 1997. (The decline in cognitive function in dementia patients who take neuroleptics is twice the decline in patients who did not take he drugs.)

Raquel Gur,et. Al. Subcortical MRI Volumes in Neuroleptic-Naï ve and Treated Patients with Schizophrenia. *American Journal of Psychiatry*, **155**, 1711-1717. 1998. (Drugs cause hypertrophy of the caudate, putamen, and thalamus, which is thought to be "structural adaptation to receptor blockade." The drug-induced hypertrophy is also "mildly associated with greater severity of both negative and positive symptoms.")

Raquel Gur, et. Al. A follow-up of magnetic resonance imaging study of schizophrenia. *Archives of General Psychiatry*, **55**, 145-151, 1998. (Use of neuroleptics is associated with volume reduction (or atrophy) of frontal lobes and temporal lobes. As the brain atrophies in this way, here is said to improvement in delusions and thought disorder (the brain-damaging principle at work). A greater rate of reduction in volume is associated with higher dose. At the same time, reduction in volume is associated with decline in some neurobehavioral functions.)

Al Madsen. Neuroleptics in progressive structural brain abnormalities in psychiatric illness. *The Lancet*, **352**, 784-785. Sept. 5, 1998. (Neuroleptic use is associated with atrophy of cerebral cortex. The estimated risk of atrophy increases by 6.4% for each additional 10 grams of neuroleptic drug.)

G. Tsai. Markers of glutamergic neurotransmission and oxidative stress associated with tardive dyskinesia. *American Journal of Psychiatry*, **155**, 1207-1213. 1998. (This study suggests that neuroleptics cause neuronal damage as a result of oxidative stress, and that this is the degenerative process that produces TD.)

Conclusion: the brain abnormalities attributed causal significance in mental illness are most likely the result of neuroleptic drug treatment.

Long Term Follow-up Studies:

Bleuler, M. (1968). A 23 Year Follow-up Study of 208 Schizophrenics. In Rosenthal and Kety (eds.) *The Transmission of Schizophrenia*. Oxford: Pergamen Press.

Ciompi, L. (1980) Catamnestic Long Term Study of the Life Course and Aging of Schizophrenics. *Schiz. Bull*. **6**, 606-618.(30 year follow-up).

Harding, C. et. Al. (1987). The Vermont Longitudinal Study of Persons with Severe Mental Illness. (32 year follow-up). *Am. J. Psychiatry*, **144**, 718-726. (A remarkable study because in contrast to the European ones-Ciompi and Bleuler- who studied consecutively admitted cohorts- Harding et.al. studied a group of so-called "chronic back-ward" patients discharged with an individualized rehabilitation program to the community.)

Hegarty, J.D.et. al. (1994) One Hundred Years of Schizophrenia: a meta-analysis of the outcome literature. *Am. J. Psychiatry* **151**: 1409-1416. (Poorer outcomes in last third of the 20th century and best in the middle third.)

Cross-Cultural Studies:

Jablensky, A.; Sartorius, N.; Ernberg, G.; Anker, M.; Korten, A.; Cooper, J.E.; Day, R.; and Bertelsen, A. (1992) *Schizophrenia: Manifestations, incidence, and course in different cultures. A World Health Organization ten-country study*. Psychological Medicine, Monograph Supplement 20:97 pp.

Lin, K.M., and Kleinman, A.M. (1988) Psychopathology and clinical course of schizophrenia: A cross-cultural perspective. *Schizophrenia Bulletin*, **14**(4): 555-567.

Leff, J.; Sartorius, N.; Jablensky, A.; and Korton, A. (1992) The international pilot study of schizophrenia: Five-year follow-up findings. *Psychological Medicine*, **22**(1): 131-145.

Murphy, H.B. and Raman, A. C. (1971) The Chronicity of Schizophrenia in Indigenous Tropical People: Results of a 12-year Follow-up. *Brit. Jour. Psych*. **118**: 489-497.

Warner, R. (1994) *Recovery from schizophrenia: Psychiatry and political economy.* (2nd Edition) London: Routledge and Kegan Paul.

World Health Organization, (1979) *Schizophrenia: An international follow-up study*. New York: John Wiley & Sons.

(all these studies find relatively benign long term outcomes -- 50 to 75% full and social recoveries -- before neuroleptics or when they were little used. Also, striking cross-cultural differences in outcome were found favoring "developing" countries -- best explained by little or no neuroleptic drug use in those countries.)

Alternatives to Psychiatric Hospitalization:

Braun, P.B., Kochansky, G., Shapiro, R., Greenberg. S., Gudeman, J.E., Johnson, S., & Shore, M.F. (1981) Overview: Deinstitutionalization of psychiatric patients: A critical review of outcome studies. *American Journal of Psychiatry*, **138**, 736-749.

Kiesler, C.A. (1982a) Mental hospitals and alternative care: Noninstitutionalization as potential public policy for mental patients. *American Psychologist,* **37**, 349-360.

Kiesler, C.A. (1982b) Public and professional myths about mental hospitalization: An empirical reassessment of policy-related beliefs. *American Psychologist*, **37**, 1323-1339.

Mosher LR. (1999) Soteria and other alternatives to acute hospitalization: A personal and professional review. *Jour. Nerv. Ment. Dis*. **187**: 142-149.

Mosher LR, Burti L (1994) *Community mental health: A practical guide*. N.Y.: W.W. Norton.

Straw, R.B. (1982) *Meta-analysis of deinstitutionalization*. (Doctoral dissertation). University Microfilms, Ann Arbor, MI: Northwestern University.

Warner, R. (Ed.) (1995) *Alternatives to the mental hospital for acute psychiatric treatment*. Wash. DC: American Psychiatric Press.

(Conclusions: every study shows alternatives to be as, or more effective, than hospital treatment and less costly.)

Psychosocial Treatment with minimal or no drug use:

Alanen, Y.O.; Ugelstad, E.; Armelius, B.A.; Lehtinen, K.; Rosenbaum, B.; and Sjostrom, R., Eds. (1994) *Early treatment for schizophrenic patients: Scandinavian psychotherapeutic approaches*. Oslo, Norway: Scandinavian University Press.

Alanen, Y.O.; Lehtinen, V.; Lehtinen, K.; Aaltonen, J.; and Rakkolainen, V. (2000) The Finnish model for early treatment of schizophrenia and related psychoses. In: Martindale, B., Bateman, A., Crowe, M., and Margison, F., Eds. *Psychosis: Psychological approaches and their effectiveness*. London: Gaskell. (The centerpiece of their approach is rapid in-home family and social network intervention to avoid hospitalization and medicalization.)

Ciompi, L., Duwalder, H.-P., Maier, C., Aebi, E., Trutsch, K., Kupper, Z., & Rutishauser, C. (1992). The pilot project "Soteria Berne": Clinical experiences and results. *British Journal of Psychiatry*, 161(suppl. 18), 145-153. (A replication of Mosher and co-workers Soteria Project in California. Similar results-about 2/3rds of newly diagnosed psychotics recovered without neuroleptic drug treatment)

Lehtinen, V. et. al. (2000). Two-Year Follow-up of First Episode Psychosis Treated According to an Integrated Model: Is immediate neuroleptisation always needed? *European Psychiatry*, 15(5): 312-320. (44% of the randomly assigned subjects received no neuroleptic drug treatment-vs. 6% of the controls- over the two-year period and their outcomes were comparable or better than those treated with drugs.)

Matthews SM, Roper MT, Mosher LR, and Menn AZ. (1979) A non-neuroleptic treatment for schizophrenia: Analysis of the two-year post-discharge risk of relapse. *Schiz. Bull.* 5: 322-333. (Soteria treated patients-as compared with hospital treated- had a significantly lower rehospitalizaton rate over two years despite few being neuroleptic maintained. First cohort analysis)

Mosher, L.R. & Bola, J.R. (2000) The Soteria Project: Twenty-five Years of Swimming Upriver. *Complexity and Change*, 9: 68-74. (Soteria patients-43%- who received no neuroleptics over the two year follow-up period did substantially better than those who did. As a group the Soteria treated patients had better outcomes than a control group that received "usual" hospital and drug treatment. The subgroup of "poor prognosis" subjects treated at Soteria had better outcomes than the Soteria group as a whole. First combined cohort analysis)

Mosher LR & Menn A Z (1978) Community residential treatment for schizophrenia: Two-year follow-up. *Hosp Comm Psych* 29: 715-723. (Better psychosocial outcomes for Soteria treated 1st and 2nd episode patients compared with control subject receiving "usual" treatment. First cohort.)

Mosher LR, Vallone R, and Menn AZ .(1995) The treatment of acute psychosis without neuroleptics: Six-week psychopathology outcome data from the Soteria project. *Int. J. Soc. Psych.* 41: 157-173. (2nd cohort: as was true of the 1st cohort, at six weeks the Soteria group had improved as much without meds as the hospital group-all of whom received neuroleptics.)

Tuori, T. et al (1998) The Finnish National Schizophrenia Project 1981-1987: 10 year evaluation of its results. *Acta. Psychiatrica Scandinavica* 97: 10-18. (In the presence of comprehensive "need adapted"psychosocial treatment, drugs are unneccesary for the most part and may, in fact, prevent recovery.)

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