4. The Psychiatric Dichotomy and the Proliferation of Models

This chapter begins by identifying the fundamental aetiological controversy within the medical model for schizophrenia — the dichotomy between theories that speculate about a biological cause for schizophrenia and theories which focus on an environmental/experiential cause. A description is given of the major biological theories and the drug treatments that support these theories. The range of enviro-experiential theories are then discussed together with the talking therapies they support.

Introduction

The wide ranging variety of scientific/psychiatric models that are currently used to explain the cause of schizophrenic symptoms[1] is indicative of the high level of confusion that persists within the medical model. These proliferating hypotheses can be readily divided into two different types: those which assume a biologically-based aetiology[2]; and psychodynamic theories rooted in assumptions that the cause is to be found in stresses resulting from the past experience, and/or the past/present environment of the sufferer.[3] This dichotomy is an echo of the old nature/nurture debate about human psychological attributes.[4] As with the nature/nurture debate, there is also a seemingly balanced and common sense view of schizophrenia aetiology which assumes both sides of the dichotomy will eventually be found to contribute to the answer.[5]

Associated with this dichotomy over aetiology is a fundamental difference of opinion within the psychiatric profession over the best forms of treatment for schizophrenia.[6] Supporters of a biological aetiology normally hold a view that proper treatment requires some form of physical intervention, usually with neuroleptic medication.[7] Subscribers to the environmental/experiential theories, on the other hand, usually prefer one of the many forms of talking therapy.[8]

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[1] Donald W. Black, William R. Yates and Nancy Andreasen, 'Schizophrenia, Schizophreniform Disorder, and Delusional (Paranoid) Disorders', in John A. Talbott, Robert E. Hales and Stuart Yudofsky, eds., Textbook of Psychiatry, American Psychiatric Press, Washington, 1988, pp. 378-385.

[2] Norman L. Keltner, 'Schizophrenia and Other Psychoses', in Norman L. Keltner, Lee Hilyard Schwecke and Carol E. Bostrom, eds., Psychiatric Nursing, Mosby, St. Louis, 1995, pp. 366-368.

[3] S. Marchevsky and E. Baram, 'Is a diagnosis of occupational post-traumatic schizophrenia possible?', Medicine and Law, Vol. 11, Nos. 1 and 2, 1992, pp. 127-136.

[4] Keltner, op.cit., p. 366.

[5] G. O. Gabbard, 'Mind and brain in psychiatric treatment', Bulletin of the Menninger Clinic, Vol. 58, No. 4, 1994, pp. 427-446.

[6] John M. Kane and Thomas H. McGlashan, 'Treatment of schizophrenia', The Lancet, Vol. 346, No. 8978, 23 Sept, 1995, pp. 820-826.

[7] L. H. Lindstrom and I. M. Wieselgren, 'Schizophrenia and antipsychotic somatic treatment', International Journal of Technology Assessment and Health Care, Vol. 12, No. 4, 1996, pp. 573-584.

[8] J. Zubin, 'Suiting therapeutic intervention to the scientific models of aetiology', British Journal of Psychiatry, No. 5, July 1989, pp. 9-14.

But the situation is not as clear cut and as logical as a simple *biological aetioliogy=drugs treatment* versus *enviro-experiential aetiology=talk therapy* debate. Sometimes it seems apparent that the two psychiatric factions have reached their aetiological and treatment associations in the opposite order. That is, the psychiatrists whose training has favoured drug therapies have simply assumed a biological aetiology as a convenient rationale for the forms of treatment they have been trained to apply.[9] Similarly, psychiatrists who have undertaken training in psychoanalytic and psychotherapeutic forms of talking therapy appear to have little choice but to assume that the cause of the problem they have been trained to talk through can be found in the past experience or environment of the patient.[10]

But even when logic would appear to require both camps within the psychiatric community to be permanently locked into the alternatives of an *either or* situation — *either* biology plus drugs *or* enviro-experiential plus talk — the actual practice of psychiatry on schizophrenics does not follow such logical patterns. In practice most psychiatrists are prepared to supervise treatment plans that mix both drugs and talk.[11] However, it is probably fair to assume that when a psychiatrist supervises such a mixed plan of treatment, one form of treatment will be seen as the essential therapy while the other is just a convenient supplement.[12]

For a biopsychiatrist — i.e. a psychiatrist who favours a biological aetiology for schizophrenia and drug therapy — the supplement of talking therapy is most likely to be useful when it involves teaching a patient some kind of living skills.[13] This can be a convenient supplement to the medication used for treating schizophrenia because the efficacy of combined treatment is largely measured by the ability of the patient to return to a position of at least partial social functioning. While the drugs supposedly re-balance a patient's brain chemistry, so that he or she *wants* to return to normal, supplementary living skills-type therapy can supposedly teach the person *how* to be normal.[14] If a semblance of normality is achieved then the efficacy of the medication is confirmed and the psychiatrist can claim a successful outcome.

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[9] R. L. Martin, 'Outpatient management of schizophrenia' American Family Physician, Vol. 43, No. 3, March, 1991, pp. 921-933.

[10] H. L. Provencher, J. P. Fournier and N. Dupuis, 'Schizophrenia: revisited', Journal of Psychiatry and Mental Health Nursing, Vol. 4, No. 4, August, 1997, pp. 275-285.

[11] G. E. Gomez and E. A. Gomez, 'Chronic schizophrenia: the major mental health problem of the century', Perspectives on Psychiatric Care, Vol. 27, No. 1, 1991, pp. 7-9.

[12] P. Weiden and L. Havens, 'Psychotherapeutic Management Techniques in the Treatment of Outpatients with Schizophrenia', Hospital & Community Psychiatry, Vol. 46, No. 6, 1994, pp. 549-555.

[13] A. E. Farmer and P. McGuffin, 'The pathogenesis and management of schizophrenia', Drugs, Vol. 35, No. 2, February, 1988, pp. 177-185.

[14] F. A. Wiesel, 'Neuroleptic Treatment of Patients with Schizophrenia. Mechanisms of Action and Clinical Significance', British Journal of Psychiatry, No. 23, 1994, pp. 65-70.

Similarly, a talking therapist might find medication a useful supplementary tool to calm a patient as a necessary prelude to achieving a therapeutic relationship:[15] i.e. a relationship in which the patient submits to the dominance of the therapist. However, this type of convenience is not appreciated by all talking therapists and an argument is sometimes mounted that no useful talking therapy can be undertaken so long as the patient is under the influence of neuroleptics.[16] This sort of argument is most likely to be made by therapists who are seeking the cause of the schizophrenia in the past experience of the patient, where memory and accurate recall are important, rather than by therapists who specialise in teaching adaptation skills.

Theories of a Biological Aetiology

There are so many different hypotheses speculating on a biological aetiology for schizophrenia that it is impossible to review them all. A search of the database Medline, for instance, which combined keywords 'schizophrenia' and 'etiology', just for the years 1990-1997, produced 1577 abstracts. Most of these articles describe scientific research seeking to confirm one or more of the hundreds of different variations on the biological hypothesis. This section of the thesis reviews some of the more influential hypotheses and demonstrates that there is very little consensus amongst scientific researchers.

Biochemical Hypotheses — and Associated Drug Treatments

The most influential of the biological hypotheses are biochemical theories that assume schizophrenic symptoms are caused by an imbalance in the chemistry of the sufferer, most particularly by an imbalance of brain chemistry. The reason for the influential position of biochemical theories is not because schizophrenic symptoms can be directly linked in laboratory work with a chemical imbalance. Rather, it is a convenient rationale derived from observations that neuroleptic medication appears to ameliorate some of the symptoms of schizophrenia. As a result it can be reasoned that the cause of the symptoms is a shortage of the chemicals contained in the medication.

This reasoning is not very sound and it could just as easily be argued that a person who counters shyness by drinking alcohol apparently has a shortage of alcohol in the brain. But the weakness in reasoning has not inhibited research based on this type of deduction. The 'dopamine theory' is the most prominent of the biochemical hypotheses and a great deal of research has been undertaken to explore the relationship between the positive symptoms of schizophrenia and the supposed hyperactivity of the dopamine system in the brains of schizophrenics.

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[15] J. H. Zahniser, R. D. Coursey and K. Hershberger, 'Individual Psychotherapy with Schizophrenic Outpatients in the Public Mental Health System', Hospital & Community Psychiatry, Vol. 42, No. 9, 1991, pp. 906-913.

[16] Peter Breggin, Toxic Psychiatry, Fontana, London, 1993, pp. 481-483.

The dopamine hypothesis is principally derived from two kinds of observation. The first is that drugs which increase the supply of dopamine, like amphetamines[17] and the anti-parkinson drug L-dopa,[18] can cause a person to enter a psychotic state. The second is that neuroleptic drugs has been observed to block dopamine receptors in laboratory animals and thereby inhibit the supply of dopamine.[19] The hypothesis derived from these observations argues that untreated schizophrenics have hyperactive dopamine systems and require neuroleptic medication to inhibit the supply of dopamine in their brains.

An often cited weakness[20] with this theory is that, whereas the dopamine receptors in the central nervous system are blocked within 20 minutes of neuroleptic medication, the drugs usually take days, sometimes many weeks, or even months, before they show any clinical effect.[21] A second weakness is that,

> although the correlation of dopamine blocking effects with the clinical potency has led to the dopamine hypothesis of schizophrenia, it is also true that these drugs reduce psychotic symptoms regardless of the diagnosis. The therapeutic effects of dopamine receptor blockade, therefore are not unique to the pathophysiology of schizophrenia.[22]

In other words, just as alcohol affects garrulous people in much the same way as it affects shy people — and therefore makes improbable a 'lack of alcohol' theory to explain the cause of shyness — so neuroleptics have much the same effect on people whether they have a prior diagnosis of schizophrenia, or not. Everyone who is treated experiences "some degree of (often total) indifference and apathy".[23] This means that although neuroleptic medication might ameliorate some of the florid features of schizophrenia, all the same, it is not a cure and therefore the dopamine hypothesis is actually quite doubtful.

On top of this the effects of neuroleptic medication on brain chemistry can be quite various, and unpredictable, depending on the type that is prescribed and the individual tolerance levels of the patient. Apart from blocking dopamine receptors various neuroleptics also blockade noradrenergic,

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[17] Black et al., op.cit., p. 382.
[18] Keltner, op.cit., p. 367.
[19] Black et al., op.cit., p. 382.
[20] Harold I. Kaplan and Benjamin J. Sadock, Synopsis of Psychiatry, Williams and Wilkins, Baltimore, 1991, p. 639.
[21] Keltner et al., op.cit., p. 367.
[22] Kaplan and Sadock, op.cit., p. 639.
[23] David Richman, 'Pursuing Psychiatric Pill Pushers', in Sherry Hirsch, Joe Adams, Leonard Frank, Wade Hudson, and David Richman, eds., Madness Network News Reader, Glide, San Francisco, 1974, p. 113. (For a more extensive description of the effects of neuroleptics see Chapter 6.)

chlonergic and histaminergic receptors, and a number of adverse effects can sometimes manifest as a result.[24]

There are many variables involved in the prescription of neuroleptics. A match has to be found for a particular patient, through a combination of heuristics and trial and error, with a particular type and brand of neuroleptic according to the individual tolerance of the patient; an appropriate dosage has to be determined for each individual patient — with the right combination of anti-side effect drugs; and the treatment has to be continued for an indefinite period to suppress psychotic symptoms that tend to fluctuate over time.

According to a number of critics,[25] the mainstream of the psychiatric profession was in a state of semi-denial until recently regarding the seriousness of the side effects caused by neuroleptic medication. This situation seems to have changed with a new openness about the problem being displayed in the latest version of the American Psychiatric Association's Diagnostic and Statistical Manual (DSM IV). The revised manual contains an appendix of "psychiatric subjects recommended for further study". Prominent amongst them is a detailed survey of medication-induced movement disorders. These are a group of diseases caused by the use of neuroleptics.[26] The list includes Neuroleptic-induced Parkinsonism which features a variety of tremors and muscle rigidity mimicking Parkinson's disease. It afflicts some 50% of patients on long-term neuroleptic treatment.[27] Neuroleptic Malignant Syndrome is an acute toxic reaction to the drugs and occurs in 0.07% to 1.4% of patients treated with neuroleptics. Of these 10%-20% die from it.[28]

Neuroleptic-Induced Acute Dystonia features abnormal positioning of the head and neck in relation to the body, spasms of the jaw muscles, impaired swallowing, thickened or slurred speech, tongue protrusion, eyes deviated up, down or sideward and abnormal positioning of the limbs or trunk. Fear and anxiety are also often a symptom and it occurs most commonly in young males.[29] If a person looked sane before treatment they certainly would not after developing this side effect.

Neuroleptic-Induced Acute Akathisia features symptoms of compulsive restlessness like fidgety movements, walking on the spot and inability to sit still. The reported prevalence of this side effect in people receiving neuroleptics varies widely from 20%-75%.[30] Once again a set of physical

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[24] Kaplan and Sadock, op.cit., p. 639.
[25] See for instance, Pam Gorring, 'Drugs and Madness', in Erica Bates and Paul R. Wilson, eds., Mental Disorder or Madness, University of Queensland Press, St. Lucia, Qld., 1979, pp. 217-233.
[26] American Psychiatric Association, Diagnostic and Statistical Manual of Mental Disorders (DSM), Fourth Edition, American Psychiatric Association, Washington, 1994, pp. 735-751.
[27] Ibid., p. 736.
[28] Ibid., p. 740.
[29] Ibid., p. 743.
[30] Ibid., p. 745.

symptoms induced by the treatment contributes dramatically to the person's appearance of being mad.

The side effect of greatest concern is Neuroleptic-Induced Tardive Dyskinesia. The indications of tardive dyskinesia are involuntary movements which might be rapid and jerky, slow and sinuous or rhythmic. They might involve the tongue, jaw, trunk or extremities. 20%-30% of people receiving neuroleptics develop tardive dyskinesia with the percentage ranging up to 50% amongst elderly people.[31] The reason why this side effect is so serious is because there is no supplementary drug treatment with which to mask its symptoms and if the neuroleptic treatment is discontinued the tardive dyskinesia symptoms remain permanently in 50% of cases. This permanency is much higher in elderly people in whom it remains unremitted in up to 95% of cases.[32]

These neuroleptic-induced movement disorders are collectively known as the central nervous system extrapyramidal side effects (EPSEs). It is perhaps incorrect to call these disorders side effects because they occur as a direct result of blocking the dopamine receptors and most of the patients receiving neuroleptics develop EPSEs. In fact it is partly through the existence of EPSEs that scientists have been able to work out how neuroleptics affect brain chemistry.

Atypical Neuroleptics

There are a number of different types of dopamine receptors and the conventional neuroleptics under discussion have been shown to be most effective in blocking D2 receptors. But a new generation of neuroleptics, called 'atypicals', have recently become available which target other types of receptors. It is claimed that the atypicals will prove to be safer than the conventional neuroleptics and will reduce the incidence of neuroleptic-induced disorders.[33] At this stage the evidence for this claim is equivocal since most of the atypicals are very new. If the new drugs do turn out to cause the same types of problems as the conventional neuroleptics it may take some years of usage before the problems become fully apparent.

However, one of the atypicals called clozapine is not a new drug and some of the major problems attached to its usage have been known for some time. Clozapine dates back to the early 1970s: "clinical trials of clozapine were begun in the United States in 1972, but they were temporarily halted in 1975 following reports of clozapine-induced agranulocytosis".[34] Agranulocytosis is "a life-threatening blood disorder that reduces the white blood cell count"[35] and it was found that some

[31] Ibid., pp. 747-749.
[32] Ibid., p. 748.
[33] William Harwell Wilson and Arvilla M. Clausen, '18-Month Outcome of Clozapine Treatment for 100 Patients in a State Psychiatric Hospital', Psychiatric Services, Vol. 46, Number 4, April 1995, p. 386.
[34] Moisey Wolf, Solomon Wolf and William Harwell Wilson, 'Clozapine Treatment in Russia: A Review of Clinical Research', Psychiatric Services, Vol. 46, No. 3, March 1995, p. 256.
[35] Breggin, op.cit., p. 106.

1-2% of patients treated with clozapine were afflicted with this condition and of these some 35% died from it.[36]

Clozapine earned such a bad reputation from its early usage that a Reagan-era drug deregulation campaigner even blamed it for causing some of the legal constrictions that he claimed inhibit drug research. He described clozapine as being one of a number of "major tragedies which have created public alarm and fear and which have led to the condemnation of drugs".[37]

By the end of the 1980s, however, as the accumulation of evidence about disorders induced by conventional neuroleptics began to overwhelm the psychiatric establishment in the United States, clozapine was still the only neuroleptic medication with an atypical profile which gave hope for a resolution to the EPSE problem. And so, in some desperation, clozapine was rehabilitated. It was approved for clinical use in the United States in 1990 after FDA-controlled trials which "lasted only six weeks".[38]

When it first went back on the market the manufacturer Sandoz,[39] as a precautionary measure, required that all the recipients undergo a weekly blood test conducted by the company's laboratories. This procedure was very expensive and pushed up the price of treatment to about $9000 a year. Under pressure from the consumer lobby, however, Sandoz was forced to make some concessions about the rigour of these blood tests and the cost of clozapine treatment was soon lowered to about $4000 a year in the US.[40]

The combination of agranulocytosis risk, the inconvenience of regular blood tests and excessive cost led to a situation in which clozapine was only being recommended for use with patients who did not respond to the traditional neuroleptics or who could not tolerate the adverse effects associated with these drugs. The clinical advice in the early 1990s described "a patient who has been unsuccessfully treated with three different antipsychotic drugs from different classes in sufficient doses, each for a least two months, is probably a candidate for treatment with clozapine."[41]

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[36] Keltner et al., op.cit., p. 245.
[37] M. Weatherall, 'An end to the search for new drugs', Nature, Vol. 296, April 1, 1982, p. 387.
[38] Breggin, op. cit., p. 447.
[39] Sandoz has since merged with Ciba-Geigy and the new conglomerate is named Novartis. For an analysis of the implications of the merger for the pharmaceutical industry see, Joan Harrison, 'Gaining critical mass to stay on top: a Ciba/Sandoz combo would have the size and cash for new R&D areas', Mergers & Acquisitions, Vol. 30, No. 6, May-June 1996, pp. 53-56.
[40] Keltner, et al., op. cit., pp. 245-246.
[41] Kaplan and Sadock, op.cit., p. 648.

During the period of its banning in the US clozapine remained available in some European countries where it continued to earn a bad reputation with some psychiatrists. Peter Breggin claimed: "Clinicians I have spoken to in Europe feel that clozapine produces a particularly profound lobotomy effect, adding to concern about long-term dangers of tardive psychosis and dementia."[42]

This negative view, however, was not shared by all European psychiatrists with long-term experience of clozapine. Two Russian psychiatrists who emigrated to the United States claimed that the drug had been used successfully in their home country for more than 20 years where "it was not reserved for neuroleptic-resistant disorders but instead was used with some success as a first-line treatment in acute disorders".[43] However, they admitted that their information was largely anecdotal and that "the Russian studies did not include controlled clinical trials, standardised diagnostic criteria, random assignment, double-blind conditions, standardised rating instruments, and other methodological approaches that we associate with scientific rigour".[44]

Clozapine survived the early scares and is now generally considered to be less dangerous than was first thought while being at least as efficacious as the conventional neuroleptics in reducing positive symptoms.[45] It is also claimed that clozapine causes less impairment of cognitive functioning than conventional neuroleptics[46] although there are apparently some severe problems with a psychotic rebound effect attached to withdrawal from clozapine.[47]

Most of the evidence indicates that the incidence of EPSEs is reduced with clozapine and for some time research has been turning to explore the link between clozapine's ability to reduce some negative symptoms of schizophrenia and the serotonin levels in the brains of schizophrenics.[48] This new line of research in schizophrenia treatment has helped to spawn a growing array of atypical neuroleptics to complement clozapine. All of these atypicals share similar features:

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[42] Breggin, op. cit., p. 105.

[43] Wolf et al., op. cit., p. 256.

[44] Ibid., p. 258. The human rights record of Soviet psychiatry poses some interesting questions in regard to the reliability of anecdotal information like that supplied by the Wolfs. There are also ethical considerations about using any scientific knowledge derived from procedures and conditions that may have been in violation of human rights.

[45] Robert Kerwin, 'Adverse Reaction Reporting and the New Antipsychotics', The Lancet, Vol. 342, No. 8885, 11 December, 1993, p. 1440.

[46] D. E. Fujii, I. Ahmed, M. Jokumsen and J. M. Compton, 'The effects of clozapine on cognitive functioning in treatment-resistant schizophrenic patients', Journal of Neuropsychiatry and Clinical Neuroscience, Vol. 9, No. 2, 1997, pp. 240-245.

[47] T. M. Shiovitz, T. L. Welke, P. D. Tigel, R. Anand, R. D. Hartman, J. J. Sramek, N. M. Kurtz and N. R. Cutler, 'Cholinergic rebound and rapid onset psychosis following abrupt clozapine withdrawal', Schizophrenia Bulletin, Vol. 22, No. 4, 1996, pp. 591-595.

[48] Anon., 'Atypical Meds Help Treatment-Refractory Patients', Psychopharmacology Update, Vol. 6, No. 11, November, 1995, pp. 1-3.

> While it was formerly believed that EPSEs and the antipsychotic benefits were inexorably bound, the atypical antipsychotic agents have been found to decrease EPSE liability through serotonin antagonism, reduced [D_2]/5[HT_2] receptor activity ratios, intrinsic anticholinergic activity, and dopamine receptor selectivity.[49]

One of the more recently launched atypicals is olanzapine (Zyprexa) made by Eli Lilly. Although quite a lot is already known about the effects of olanzapine on chemical receptors in the brain it is not understood whether any of these known effects contribute in any way to the observed reduction in schizophrenic symptoms: "The mechanism of action of olanzapine (oh lan' za peen) is unknown. The drug binds to serotonin type 2, 3 and 6, dopamine D_1, D_2 and D_4, histamine H_1, adrenergic alpha$_1$, and muscarinic (particularly M_1) receptors."[50]

The binding properties of the atypicals are giving rise to wide speculation about the aetiology of schizophrenia which extend well beyond the old dopamine theory inspired by the conventional neuroleptics.[51] Some researchers think the mechanisms of the atypicals will eventually make it possible to trace the cause of schizophrenic symptoms while others believe that little aetiological evidence will emerge from atypical usage.[52]

Despite the claimed benefits for schizophrenics from atypical usage enthusiasm for the new drugs amongst pharmaceutical researchers might be best understood in economic terms. By the end of the 1980s, as patents for the conventional neuroleptics expired and generics became available, the continued reliance on conventional neuroleptics threatened to undermine pharmaceutical profits . As a result of this situation the quest for a new generation of schizophrenia drugs was driven at least as much by a need to ensure the long-term profitability of pharmaceuticals for schizophrenia treatment as it was to discover safer drugs which targeted the cause of schizophrenia more precisely. In fact when the cost of the atypicals is compared to that of conventional neuroleptics, and the equivocal nature of some claims for atypicals is borne in mind — particularly in regard to improved safety and aetiological insights — then the significance of the economic factors is brought into perspective.

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[49] Walter Alexander, 'For schizophrenia: atypical agents offering promise', Drug Topics, Vol. 140, No. 11, 10 June, 1996, p. 71.

[50] Anon., 'Olanzapine For Schizophrenia', Medical Letter on Drugs & Therapeutics, Vol. 38, No. 992, 17 January, 1997, pp. 5-7.

[51] Maude Campbell, 'New Atypical Antipsychotics Focus of International Workshop on Schizophrenia', Psychiatric Times, May 1995, Available URL, http://www.mhsource.com./edu/psytimes/p950523.html

[52] Editorial, 'Atypical treatments for schizophrenia' The Lancet, Vol. 339, No. 8788, 1 Feb, 1992, pp. 276-278.

The table below shows the cost to pharmacists in the United States for 30 days treatment with usual dosage based on the average wholesale price in 1996 and January 1997.[53]

COST OF SOME DRUGS FOR SCHIZOPHRENIA

Drug	Usual Dosage	Cost
Conventionals		
Thorazine (SK Beecham)	200 mg	$64.65
Haldol (haloperidol) (McNeil)	5 mg	$85.78
Generics		
Haloperidol	5 mg average generic price	$36.01
Chlorpromazine	200 mg average generic price	$8.89
Atypicals		
Risperidone - Risperdal (Janssen)	3 mg	$241.62
Clozapine - Clozaril (Sandoz)	100 mg	$307.80
Olanzapine - Zyprexa (Lilly)	10 mg once daily	$232.20

Table adapted from: 'Olanzapine For schizophrenia', Medical Letter on Drugs and Therapeutics, Vol. 38, No. 992, 17 January, 1997, pp. 5-7.

Other Biochemical Theories

Only the dopamine theory is directly linked to the array of neuroleptic medications that are routinely prescribed for schizophrenia. However, many psychiatric researchers are willing to uncouple their assumptions of cause from standard forms of treatment. Beside the theories derived from research associated with the usage of conventional and atypical neuroleptics there are also a number of other biochemical theories. But without any specific drug treatment to support them none of these other biochemical theories is as persuasive as the dopamine hypothesis.

As well as extending the aetiological search to serotonin neurotransmitters researchers have also targeted the neurotransmitters concerned with norepinephrine, glutamate, and related excitatory amino acids, and the neuropeptides cholecystokinin and neurotensin, but have only found a "fragmentary body of data which provides neither consistent nor conclusive evidence for any specific etiologic theory".[54]

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[53] Anon., 'Olanzapine For schizophrenia', op.cit., pp. 5-7.

[54] J. A. Lieberman and A. R. Koreen, Neurochemistry and Neuroendocrinology of Schizophrenia: A Selective Review', Schizophrenia Bulletin, Vol. 19. No. 2, 1993, pp. 371-429.

It has also been argued that heightened concentrations of the neuromodulator phenylethylamine at the post-synaptic dopamine receptor could be involved in the aetiology of schizophrenia. A study carried out on Indian patients with paranoid schizophrenia has indicated a correlation[55] but the findings have not been confirmed.

Another neuromodulator theory involves a speculation that the system of nigral enkephalin peptides may be disordered in schizophrenia[56] while another speculates that the source of the problem might be found with the regulation of opioid peptides.[57] A further neuromodulator theory is based on an assumption that there are essentially two states of mind — a cognitive processing mode and an analogical mode — and that noradrenergic (NE) neurons located in the locus coeruleus switches the mind to the analogical mode while dopaminergic (DA) neurons located in the ventral tegmental area alternatively switch the mind to the cognitive mode. It is proposed that schizophrenics with positive symptoms have an excess of NE and are therefore analogically inclined while schizophrenics with negative symptoms have an excess of DA which inclines them to the cognitive mode.[58]

There is a theory that a mechanism in the hippocampus might cause nicotinic stimulation of gamma-aminobutyric acid (GABA) which in turn could be the cause of the decreased responses of schizophrenics to repeated stimuli.[59] Essential fatty acids have also been linked to schizophrenia and there is indirect evidence of impaired metabolism of prostaglandin in schizophrenics.[60] It has also been suggested that prostaglandin hyposensitivity resulting from a prostaglandin deficiency is a characteristic of schizophrenics.[61]

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[55] B. A. Davis, S. Shrikhande, V. P. Paralikar, S. R. Hirsch, D. A. Durden and A. A. Boulton, 'Phenylacetic Acid in CSF and Serum in Indian Schizophrenics', Progress in Neuro-Psychopharmacology & Biological Psychiatry, Vol. 15, No. 1, 1991, pp. 41-47.

[56] M. J. Iadarola, D. Ofri and J. E. Kleinman, 'Enkephalin, Dynorphin and Substance P in Postmortem Substantia Nigra from Normals and Schizophrenic Patients', Life Sciences, Vol. 48, No. 20, 1991, pp. 1919-1930.

[57] G. B. Stefano, B. Scharrer, T. V. Bilfinger, M. Salzet and G. L. Fricchione, 'A novel view of opiate tolerance', Advancements in Neuroimmunology, Vol. 6, No. 3, 1996, pp. 265-277.

[58] J. P. Tassin, 'NE/DA Interactions in Prefrontal Cortex and Their Possible Roles as Neuromodulators in Schizophrenia', Journal of Neural Transmission, Vol. 36, No. 1, 1992, pp. 35-62.

[59] S. Leonard, C. Adams, C. R. Breese, L. E. Adler, P. Bickford, W. Byerley, H. Coon, J. M. Griffith, C. Miller, M. Myles-Worsley, H. T. Nagamoto, Y. Rollins, K. E. Stevens, M. Waldo and R. Freedman, 'Nicotinic receptor function in schizophrenia', Schizophrenia Bulletin, Vol. 22. No. 3, 1996, pp. 431-445.

[60] D. F. Horrobin, 'The Relationship Between Schizophrenia and Essential Fatty Acid and Eiconsanoid Metabolism', Prostaglandins Leukotrienes & Essential Fatty Acids, Vol. 46, No. 1, 1992, pp. 71-77.

[61] H. Kaiya, 'Prostaglandin E1 Suppression of Platelet Aggregation Response in Schizophrenia', Schizophrenia Research, Vol. 5, No. 1, 1991, pp. 67-80.

There is also a theory about a possible imbalance in the relationship between dopamine and acetylcholine in the brains of schizophrenics[62] as well as theories arguing that histamine plays a role and that an impairment of the histamine receptors might be the cause.[63]

The possible role of norepinephrine in schizophrenia has been extensively explored. One study found a purported link in the interaction between dopamine and norepinephrine.[64] The norepinephrine hypothesis is closely linked to the dopamine hypothesis since norepinephrine is thought to regulate dopamine and an excess of norepinephrine is possibly the cause of a dopamine excess.[65] Other researchers have extended the dopamine/norepinephrine link with schizophrenia to include serotonin.[66]

The dopamine metabolite homovanillic acid (HVA) has also been found at abnormal levels in schizophrenics,[67] and even in the parents of schizophrenics.[68] But HVA is mainly used as an indirect tool to assess changes in dopamine levels[69] so this area of research is also still closely linked to the dopamine hypothesis.

Uncertainties in Schizophrenia Research

The search for a definitive link between biochemistry and schizophrenia goes on. But as with all the biological hypotheses, although they can be explored using various methods of 'hard' science, they must always rest on swampy ground. The swamp on which schizophrenia researchers have to base their theories is two-fold. On the one hand there is the subjectivity of the diagnostic process which means that the search for a biological common denominator amongst groups of schizophrenics is founded on the dubious assumption that all the members of a schizophrenic cohort will actually have a common denominator, other than a history of neuroleptic medication, that makes them

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[62] M. Lyon, N. Lyon and M. S. Magnusson, 'The Importance of Temporal Structure in Analysing Schizophrenic Behaviour: Some Theoretical and Diagnostic Implications', Schizophrenia Research, Vol. 13, No. 1, 1994, pp. 45-56.

[63] T. Nakai, N. Kitamura, T. Hashimoto, Y. Kajimoto, N. Nishino, T. Mita and C. Tanaka, 'Decreased Histamine H1 Receptors in the Frontal Cortex of Brains From Patients With Chronic Schizophrenia', Biological Psychiatry, Vol. 30, No. 4, 1991, pp. 349-356.

[64] D. P. van Kammen and M. Kelley, Dopamine and Norepinephrine Activity in Schizophrenia. An Integrative Perspective', Schizophrenia Research, Vol. 4, No. 2, 1991, pp. 173-191.

[65] D. P. van Kammen, 'The Biochemical Basis of Relapse and Drug Response in Schizophrenia: Review and Hypothesis', Psychological Medicine, Vol. 21, No. 4, 1991, pp. 881-895.

[66] J. N. Joyce, 'The Dopamine Hypothesis of Schizophrenia: Limbic Interactions With Serotonin and Norepinephrine', Psychopharmacology, Vol. 112, No. 1, pp. S16-S34.

[67] F. Amin, M. Davidson and K. L. Davis, "Homovanilic Acid Measurement in Clinical Research. A Review of Methodology', Schizophrenia Bulletin, Vol. 18, No. 1, 1992, pp. 123-148.

[68] J. Wei, H. M. Xu and Hemmings, 'Studies on Neurochemical Heterogeneity in Healthy Parents of Schizophrenic Patients', Schizophrenia Research, Vol. 10, No. 2, 1993, pp. 173-178.

[69] M. Davidson, R. S. Kahn, R. G. Stern, J. Hirschowitz, S. Apter, P. Knott and K. L. Davis, 'Treatment With Clozapine and Its Effect on Plasma Homovanillic Acid and Norepinephrine Concentrations in Schizophrenia', Psychiatric Research, Vol. 46, No. 2, 1993, pp. 151-163.

different from normal people. Since there is no diagnostic certainty about schizophrenia there is also no certainty that a common aetiological factor has prior existence, which makes the quest for this common cause a tenuous enterprise.[70]

The second problem is the history of neuroleptic medication which applies to practically all schizophrenics. Routine psychiatric practice normally requires that a person be medicated immediately upon diagnosis. This means that any group of schizophrenics which might be available to a scientific researcher will inevitably be comprised of people whose brain functioning has been modified by powerful chemicals. The subtle deviations from normal biochemistry and normal brain architecture that are detected by researchers can usually be better explained as artefacts of neuroleptic medication.[71]

This problem is readily apparent to researchers and, while most of them choose to ignore it and continue their research programmes as if it were not a factor, occasionally one researcher will add extra significance to findings by claiming to have carried out the research on schizophrenics who had no experience with neuroleptics.[72] However, such claims consistently avoid describing how the groups of 'never-medicated' schizophrenics were assembled so there is some doubt about the validity of these claims. Occasionally, however, is it suggested that they were people recruited on first-admission entry to hospital.[73] This method of recruitment raises an interesting ethical question about a hospital admission procedure which requires people who are apparently in acute distress to undergo research testing before attention can be given to their own problems.[74] On top of this ethical problem there is also the doubt that a distressed person's treatment history can be accurately obtained from the person at the point of entry into hospital.

Brain Imaging

There are a number of brain imaging techniques commonly used in schizophrenia research. Some of these techniques are only useful for assessing the structure of the brain while others can assess both the structure and certain functions, like blood flow.[75] The application of various forms of brain imaging to people who have been diagnosed with schizophrenia has allowed researchers to compare

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[70] David E. Sternberg, 'Schizophrenia', in A. James Giannini, The Biological Foundations of Clinical Psychiatry, Medical Examination Publishing Company, New York, 1986, p. 148.

[71] Breggin, op.cit., pp. 138-141.

[72] Monte S. Buchsbaum, 'Frontostriatal disorder of cerebral metabolism in never-medicated schizophrenics', JAMA, The Journal of the American Medical Association, Vol. 269, No. 17, 5 May, 1993, p. 2204.

[73] Jarmo Hietala, Erkka Syvalahti, Klaus Vuorio, Viljo Rakkolainen, Jorgen Bergman, Merja Haaparanta, Olof Solin, Mikko Kuoppamaki, Olli Kirvela, Ulla Ruotsalainen and Raimo K. R. Salokangas, 'Presynaptic dopamine function in striatum of neuroleptic-naive schizophrenic patients', The Lancet, 28 Oct, 1995, Vol. 346, No. 8983, pp. 1130-1132.

[74] For a discussion on the ethics of taking schizophrenics off medication to facilitate research see, W. T. Carpenter Jr., 'The risk of medication-free research', Schizophrenia Bulletin, Vol. 23, No. 1, 1997, pp. 11-18.

[75] Kaplan and Sadock, op.cit., p. 76.

the results with the brain-structure and the brain-functioning of normal people and to observe certain patterns of difference. These perceived differences have become the basis for a wide variety of hypotheses about the aetiology of schizophrenia.

Computed tomography (CT) is a method of x-raying living brains and is many times more sensitive than conventional radiography. In CT scanning x-ray photons are passed through the tissue under examination while the x-ray tube and the detector are rotated around the head as well as moved in parallel lines. The data that results from this process are entered into a computer which is programmed to provide a three dimensional reconstruction.[76] This imaging can detect lesions on the brain. Schizophrenia studies have largely focussed on a perceived enlargement of ventricles, brain atrophy and unusual brain asymmetries. Although CT does not cause physical discomfiture patients are exposed to radiation by the process.[77]

Magnetic resonance imaging (MRI) is not a form of x-ray and works on a different principle to that of CT. MRI applies a strong magnetic field to the brain under investigation which causes a realignment of hydrogen atoms. The realignment in turn causes an alteration to the radio frequency emitted by the hydrogen atoms and this alteration is read by a computer. The result is an image of the brain that is clearer and more complete than a CT scan but which is more expensive to produce.[78] In schizophrenia research MRI is used for the same purposes as CT. One of the advantages of MRI is that a patient is not exposed to radiation by the process.[79]

Positron emission tomography (PET) is the most advanced and expensive form of brain imaging. It requires a patient to be dosed with glucose containing radioactive atoms.[80] Once the radioactive substance has reached the brain positrons are emitted which collide with electrons in the brain. The radio emission that result from this process can be detected by probes from a PET camera. The information is analysed by a computer and topographical maps of the brain are generated. It is possible to use PET to measure the number, and to assess the state, of neurotransmitters in the brain.[81]

Single photon emission computed tomography (SPECT) works in a similar way to PET in that a patient is required to be dosed with a radioactive substance. In the case of SPECT a single photon emitting isotope is used that generates activity in the brain which can be detected.[82] The resulting

[76] Ibid., p. 78.
[77] Keltner, op.cit., p. 367.
[78] Ibid., p. 367.
[79] Kaplan and Sadock, op.cit., p. 79.
[80] Keltner, op.cit., p. 367.
[81] Kaplan and Sadock, op.cit., p. 84.
[82] Ibid., p. 84.

data is processed by a computer and maps of the brain can be produced. SPECT results are not as detailed as those of PET scans but they are less expensive.[83]

Scanning For Causes

Developments in brain imaging techniques have given rise to a growing body of speculation that the cause of schizophrenia will be found in abnormalities in the brain architecture of schizophrenics:

> The kaleidoscopic images on PET scans suggest that there are structural defects in certain regions of schizophrenic brains which may lead them to process and retain information differently from healthy brains. Such alterations can produce behaviour from the extravagantly bizarre to intense withdrawal, prolonged apathy, and other emotional or affective disturbances.[84]

This type of brain scanning has revealed that the ratio of blood in the frontal lobes, between the front and the back, is lower in the brains of schizophrenics than in the brains of normal people. This has led to speculation that it is frontal lobe dysfunction that causes the positive symptoms of schizophrenia. Another theory that has developed out of apparent abnormalities detected by brain scans is that the cause of schizophrenia is to be found is some kind of trauma that has been experienced in the womb. Brain scans have revealed,

> increased amounts of cerebrospinal fluid (CSF) in the brain of many schizophrenics. A correspondingly slightly smaller brain volume has also been found. Since physical brain abnormalities do not progress further as the patient ages, levels of neuropathology may be present before birth. [85]

Apparent evidence from CT scans of degeneration in a particular part of the brain called the cerebellar vermis has contributed to a brain atrophy hypothesis for schizophrenia.[86] MRI scans have indicated that schizophrenic brains have a smaller average volume of total brain tissue than do the brains of normal people. The smaller brain volume is also found to be offset by an increase in the volume of cerebrospinal fluid in schizophrenic brains. The frontal lobes is the area where specific decreases seem most apparent. These findings have led researchers to conclude that:

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[83] Keltner, op.cit., p. 367.
[84] Anon., 'The anatomy of madness', Psychology Today, Vol. 25, No. 6, Nov-Dec, 1992, p. 16.
[85] Daniel R. Weinberger, 'From neuropathology to neurodevelopment', The Lancet, Vol. 346, No. 8974, 26 August, 1995, pp. 552-558.
[86] R. Sandyk, S. R. Kay and A. E. Merriam, 'Atrophy of the Cerebellar Vermis: Relevance to the Symptoms of Schizophrenia', International Journal of Neuroscience, Vol. 57, Nos. 3 and 4, 1991, pp. 205-212.

> In addition to the generalised brain abnormalities observed in schizophrenia, a regional abnormality may be present in frontal regions. Since the frontal lobes integrate multimodality information and perform a variety of 'higher' cognitive and emotional functions that are impaired in schizophrenia, the frontal abnormality noted is consistent with the clinical presentation of the illness. Impaired frontal function and a disruption in its complex circuitry (including thalamocortical projections) may explain why patients with schizophrenia often have significant deficits in formulating concepts and organising their thinking and behaviour.[87]

There is no mention, however, in the description of this research as to whether the schizophrenics had been treated with neuroleptic medication prior to the MRI scans. It therefore has to be assumed that they had been medicated and that the findings might be artefacts of the treatment.

One group of researchers used MRI to scan the brains of 15 pairs of identical twins where one twin in each pair had schizophrenia. The schizophrenic twin in 14 of the 15 pairs was found to have enlarged ventricles. To deal with the problem of prior neuroleptic treatment the degree of ventricle enlargement was cross-checked to see if it correlated with the length of time the schizophrenic twins had been on medication: "Brain abnormalities were not more severe among the schizophrenics with a long history either of the disorder or of antipsychotic drug treatment. Thus, the changes appear linked directly to schizophrenia."[88]

These findings have led to speculations that early in brain development there might be some kind of viral infection, birth injury or autoimmune disorder which underlies abnormal brain development in schizophrenics. These abnormalities could go unnoticed until late adolescence when the central nervous system undergoes maturational changes. This is the time of life when the symptoms of schizophrenia most commonly emerge.

There are, however, significant problems with this type of assumption. One problem is that the possible contribution of neuroleptic medication to the abnormalities is not properly discounted simply by finding no correlation between the duration of medication and the degree of brain structure abnormality. This approach presupposes that if neuroleptics play a role then the longer they are used the greater would be the abnormality. But no evidence is supplied to support this assumption.

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[87] Nancy C. Andreasen, Laura Flashman, Michael Flaum, Stephan Arndt, Victor Swayze II, Daniel S. O'Leary, James C. Ehrhardt and William T. C. Yuh, 'Regional brain abnormalities in schizophrenia measured with magnetic resonance imaging', JAMA, The Journal of the American Medical Association, Vol. 272, No. 22, 14 December, 1994, pp. 1763-1770.

[88] B. Bower, 'Brain Anatomy Yields Schizophrenia Clues', Science News, Vol. 137, No. 12, 24 March, 1990, p. 182.

Another problem is that in most groups of schizophrenics there are some schizophrenics who do not have large ventricles. One investigation found that it was only the males in a group of schizophrenics who had large ventricles.[89] On top of this it has been found that some people with Alzheimer's disease and others with manic depression also have large ventricles so that this abnormal feature is neither necessary for schizophrenia, nor is it associated solely with schizophrenia.[90]

A medical scientist who reviewed the research literature on the neuropathology of schizophrenia in the early 1990s wrote:

> Neuroscientific inquiry into this most devastating of mental illnesses is, then, bearing hard won fruit. Few would now dispute that a substantial proportion of patients with schizophrenia do indeed have consistent structural and physiological brain abnormalities. The challenges for research workers are to establish the specificity of these abnormalities to schizophrenia and to clarify the relations between biological, aetiological, and diagnostic heterogeneity.[91]

But the above statement is riddled with complications and the sought-after heterogeneity that would positively define aetiology is still eluding the researchers. More recently the enlarged ventricles of schizophrenics have been associated with prenatal exposure to influenza,[92] but the evidence is equivocal.

Infection Theories

The theory of an infectious organism as the cause of schizophrenia is not new. In the early decades of the 20th century between a quarter and a third of patients admitted to mental hospitals in industrialised countries were suffering from general paresis, a condition produced in the tertiary stage of syphilis.[93] For a long time there was considerable confusion within mental hospitals between people who manifested the symptoms of neurosyphilis and those who had schizophrenic symptoms.

The advent of penicillin in the 1940s seems to have focussed a belief amongst some psychiatric researchers that a similar infectious cause and medical remedy might be found for schizophrenia.

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[89] Ibid.
[90] Ibid.
[91] L. S. Pilowsky, 'Understanding schizophrenia: structural and functional abnormalities of the brain are present in the condition', British Medical Journal, Vol. 305, No. 6849, 8 August, 1992, pp. 327-329.
[92] N, Takei, S. Lewis, P. Jones, I. Harvey and R. M. Murray, 'Prenatal exposure to influenza and increased cerebrospinal fluid spaces in schizophrenia', Schizophrenia Bulletin, Vol. 22, No. 3, pp. 521-534.
[93] Thomas Szasz, Schizophrenia: The Sacred Symbol of Psychiatry, Syracuse University Press, Syracuse, New York, 1976, p. 7.

Viral theories of aetiology are particular attractive to some researchers because they can tie up a number of loose ends like the perceived seasonality of schizophrenic births,[94] the difficulties some schizophrenic women are said to have in childbirth, the frequency of auditory hallucinations and an assumed genetic component of the disease.[95]

The viral theories are essentially of two types: those which postulate an active but undetected virus that directly affects the brain and gives rise to unusual psychological phenomena; and theories which postulate a past infection that, although no longer active, caused abnormalities in brain development. Retroviruses have been suggested as the likely culprit for the first type of possibility but researchers have been unable to find any positive link between schizophrenia and this type of virus.[96] Borna disease was recently discounted as a virus that might be active in schizophrenics.[97]

One explanation for the second type of possibility is that schizophrenia is linked to viral epidemics that have occurred prenatally. It is argued that schizophrenics might be part of a sub-population with special resistance to disease. Although the resistance itself may not be a factor of schizophrenia it might result in fetal vulnerability to hormonal disturbances during prenatal viral infection. This vulnerability might in turn lead to neurodevelopmental damage. It is further argued that if this were so then schizophrenia could be seen as one of the prices a population has to pay for surviving epidemics.[98]

Enthusiasm for the viral epidemic theory has recently focussed on polio. There seem to be a number of attractive features to the polio theory — a claimed decrease in numbers of schizophrenics coinciding with the advent of polio vaccine, a higher number of winter births confirming the possibility of second trimester infection in summer months, when polio is most active, and a higher incidence of schizophrenia amongst immigrants to the United Kingdom coinciding with higher incidence of polio in countries of origin. Once again it is postulated that prenatal infection with

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[94] R. L. O'Reilly, 'Viruses and Schizophrenia', Australian & New Zealand Journal of Psychiatry, Vol. 28, No. 2, 1994, pp. 222-228.

[95] E. F. Torrey, 'Viral-Anatomical Explanation of Schizophrenia', Schizophrenia Bulletin, Vol. 17, No. 1, 1991, pp. 15-18.

[96] M. A. Coggiano, R. C. Alexander, D. G. Kirch, R. J. Wyatt and H. Kulaga, 'The Continued Search for Evidence of Retroviral Infection in Schizophrenic Patients', Schizophrenia Research, Vol. 5, No. 3, 1991, pp. 243-247.

[97] J. A. Richt, R. C. Alexander, S. Herzog, D. C. Hooper, R. Kean, S. Spitsin, K. Bechter, R. Schuttler, H. Feldmann, A. Heiske, Z. F. Fu, B. Dietzschold, R. Rott and H. Koprowski, 'Failure to detect Borna disease virus infection in peripheral blood leukocytes from humans with psychiatric disorders', Journal of Neurovirology, Vol. 3, No. 2, 3 April, 1997, pp. 174-178.

[98] G. Rubinstein, 'Schizophrenia, Infection and Temperature. An Animal Model For Investigating Their Interrelationships', Schizophrenia Research, Vol. 10, No. 2, 1993, pp. 95-102.

polio might cause developmental problems in the brain that do not emerge until sexual maturity.[99] But no significant evidence has been found yet to provide confirmation for the polio hypothesis.

Nutrition

Nutrition is an area of speculation with a substantial following. Like the viral theories nutritional theories divide into theories that postulate prenatal nutritional deficiencies, causing developmental abnormalities, and theories arguing that a deficiency in the current diet of the patient is the cause. Whereas the first group of theories generally do not have a direct remedy, the second group often does. (These are usually in the form of dietary supplements. This form of 'treatment' does not give rise to human rights problems).

A considerable amount of research has focussed on historically recorded famines and these events have been used to explore a hypothesised link between the starvation of pregnant mothers and schizophrenia in offspring.[100] The Dutch Winter Famine of 1944-1945 has provided Dutch researchers with the opportunity to explore this connection and one group has concluded that starvation during pregnancy can be a factor in the development of schizoid personalities in offspring.[101]

Researchers give four reasons for supporting the prenatal nutrition hypothesis: 1) the known effects of prenatal starvation are not incompatible with the observed features of schizophrenia; 2) brain abnormalities can develop as a result of these events; 3) general malnutrition has also been observed to cause abnormalities in areas of the brain that have been linked to schizophrenia; 4) it is known that proper prenatal nutrition is essential for the correct development of the fetal nervous system.[102] But the circumstantial nature of this reasoning readily demonstrates that no hard evidence has yet been found to support this type of hypothesis. Indeed, it actually seems quite implausible when there appears to be no evidence of a higher incidence of schizophrenia in countries where malnutrition has been endemic for generations. Nevertheless this area seems particularly attractive to some researchers and the Dutch Winter Famine has been statistically linked to a two-fold increase in the risk of schizophrenia.[103]

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[99] R. F. Squires, 'How a poliovirus might cause schizophrenia: a commentary on Eagles' hypothesis', Neurochemical Research, Vol. 22, No. 5, May, 1997, pp. 647-656.

[100] J. O. Davis and H. S. Bracha, 'Famine and schizophrenia: first-trimester malnutrition or second-trimester beriberi', Biological Psychiatry, Vol. 40, No. 1, 1 July, 1996, pp. 1-3.

[101] H. W. Hoek, E. Susser, K. A. Buck, . H. Lumey, S. P. Lin and J. M. Gorman, 'Schizoid personality disorder after prenatal exposure to famine', American Journal of Psychiatry, Vol. 153, No. 12, December, 1996, pp. 1637-1639.

[102] A. S. Brown, E. S. Susser, P. D. Butler, R. Richardson-Andrews, C. A. Kaufmann and J. M. Gorman, 'Neurobiological plausibility of prenatal nutritional deprivation as a risk factor for schizophrenia', Journal of Nervous and Mental Disorders, Vol. 184, No. 2, February, 1996, pp. 71-85.

[103] E. Susser, R. Neugebauer, H. W. Hoek, A. S. Brown, S. Lin, D. Labovitz and J. M. Gorman, 'Schizophrenia after prenatal famine. Further evidence', Archives of General Psychiatry, Vol. 53, No. 1, January, 1996, pp. 25-31.

Amongst the second line of nutritional theories, which argue that schizophrenia is caused by deficiencies in the diets of adult schizophrenics, one theory claims that low concentrations of PGE-1, n-6 fatty acids, vitamin C and zinc have been linked to schizophrenia and can be rectified with a high wheat diet.[104] But the recommendation for a high wheat diet directly contradicts information publicised recently by the Schizophrenia Association of Great Britain:

> The kinds of cereal grain from products customarily eaten may be a factor in the production of psychiatric symptoms. There might be a relationship between schizophrenia and coeliac disease, a disease of known sensitivity to wheat and sometimes to milk. The wheat and rye-eating areas of the world have the highest incidence of schizophrenia, with oats and barley areas next, followed by the rice-eating areas (with approximately 60% of the incidence of the wheat areas). In sorghum and maize-eating areas the incidence of schizophrenia was approximately 25% of the wheat areas and in the highlands of New Guinea a practically nil incidence is found. Here no grains are eaten. William Philpott, an American psychiatrist, found that half his sample of schizophrenic patients could not tolerate milk and 64% were wheat sensitive.[105]

The confusion over whether wheat might have a beneficial or detrimental effect on people inclined towards psychosis is fairly typical of the many contradictions that surround schizophrenia. Perhaps this particular area of confusion might in part be explained by the well-known relationship between vitamin B12 deficiency and pellagra, a disease that effects the skin, the digestive and nervous systems, and which commonly presents with schizophrenia-like symptoms.

> In 1937, Elvehjen identified niacin deficiency as the cause and niacin the cure for pellegra, after which large numbers of pellagra psychotics recovered and were found not to be schizophrenic. As a result of this discovery, niacin is now routinely added to bread.[106]

Despite this long-known association between vitamin B12 deficiency and psychotic symptoms contemporary researchers still occasionally announce its re-discovery. One recent example from Singapore reported a case study involving an observed link between vitamin B12 deficiency,

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[104] R. S. Smith, 'The GI T-lymphocyte theory of schizophrenia: some new observations', Medical Hypotheses, Vol. 37, No. 1, January, 1992, pp. 27-30.

[105] Brian Hyfryd, 'Neglect of the Body to the Detriment of the Patient: Management Notes', Journal of Nutritional & Environmental Medicine, Vol. 7, No. 1, March, 1997, pp. 47-53.

[106] James S. Howard, 'Requiem For Schizophrenia', Integrative Physiological & Behavioral Science, Vol. 31, No. 2, April-June 1996, pp. 148-155.

anaemia and schizophrenia and recommended supplementing neuroleptic medication with a substance to compensate.[107]

Genetic Theories

Genetic theories underlie many of the biological theories since it is often assumed that only those people with a genetic vulnerability will manifest the neuropathological condition that causes schizophrenia, or succumb to the virus, or the malnutrition, that causes the neuropathology that in turn causes schizophrenia. Indeed, a genetic factor is fairly essential to give credibility to any biological explanation of aetiology because one of the few uncontested features of schizophrenia, to which most observers agree, is that schizophrenia tends to run in families. Without a plausible genetic argument the phenomenon of family propensity for schizophrenia might always be better explained by environmental causes.

The idea that a single dominant genetic component might be solely responsible — that there could be a 'schizophrenia gene' — has to be discounted by other observable phenomena. Breggin argues that when geneticists go in search of a dominant gene as the cause for a disease like Huntington's chorea the quest makes sense because there is prior knowledge from the set pattern of family inheritance that a dominant gene is responsible.[108] But this is clearly not the case with schizophrenia. Although there is a family association with schizophrenia, there is no set pattern of inheritance.

The prior existence of a logical refutation of any claims to have discovered a 'schizophrenia gene' has not deterred some genetic researchers from embarking on the quest and occasionally announcing success. In 1988 it was announced that chromosome 5 was the site of the schizophrenia gene: "For the first time, scientists have obtained evidence that a specific chromosome mutation contains a gene predisposing its bearers to schizophrenia and closely related mental disorders".[109]

The announcement was made in the November 10, 1988 issue of Nature and a refutation came so swiftly it was even published in the same edition of the same journal:

> Another study in the same NATURE indicates, however, that schizophrenia is too complex to result from a single gene. Psychiatrist James L. Kennedy of Yale University and his co-workers report that the same region of chromosome 5 is unrelated to schizophrenia in several generations of a large Swedish family. The researchers suggest

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[107] S. M. Ko, and T. C. Liu, 'Psychiatric syndromes in pernicious anaemia — a case report', Singapore Medical Journal, Vol. 33, No. 1, February, 1992, pp. 92-94.

[108] Breggin, op.cit., p. 122.

[109] Bruce Bower, 'Schizophrenia: genetic clues and caveats', Science News, Vol. 134, No. 20, 12 Nov, 1988, p308.

> there may be several genes, each causing a different biochemical abnormality, that together result in a 'final common pathway' to schizophrenia.[110]

More recently the search for the schizophrenia gene has concentrated on Irish families and in 1995,

> a team of scientists headed by Scott Diehl of the National Institute of Health found a specific region of Chromosome 6 that appears to contain a gene for the disorder. "If our finding holds up, it means that, contrary to what a lot of researchers have thought, there is at least one major gene that predisposes a person to schizophrenia," said Diehl.[111]

The equivocal statement, "If our finding holds up," was a definite warning that Diehl himself was uncertain about the claims he was making for Chromosome 6. Ten days later the reason for this uncertainty was revealed. It seems that Diehl had published the results of work he had been engaged in as a junior research scientist at the Medical College of Virginia under the direction of Dr. Kenneth Kendler. But Diehl had moved on to another research facility in 1993. A simmering dispute had continued between Diehl and Kendler between 1993 and 1995 over whether Diehl had any rights to the research he had undertaken while working for Kendler. Diehl's 1995 announcement of having discovered the schizophrenia gene turned out to be a pre-emptive strike to claim ownership of the intellectual property. But it seems that Diehl had only completed 10% of the gene mapping work envisioned in Kendler's research scheme and Diehl's article only described preliminary findings. At stake were millions of dollars of research funding to complete the project.[112]

Many genetic researchers were not convinced that Kendler and Diehl are even on the right track in pursuing chromosome 6. Some researchers have targeted chromosome 22 as being the most likely site for the schizophrenia gene.[113] Most genetic research, however, now assumes that there is no single schizophrenia gene and that a variety of separate genetic factors are involved.[114]

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[110] Ibid.

[111] Mark Bowden, 'Study finds evidence for gene that may help cause schizophrenia,, Knight-Ridder/Tribune News Service, 1 May, 1995 p. 501.

[112] Mark Bowden, 'Top human geneticists argue over ownership of schizophrenia research', Knight-Ridder/Tribune News Service, 11 May, 1995, p. 511.

[113] K. C. Murphy, A. G. Cardno and P. McGuffin, 'The molecular genetics of schizophrenia', Journal of Molecular Neuroscience, Vol. 7, No. 2, 1996, pp. 147-157.

[114] Peter McGuffin, Michael J. Owen and Anne E. Farmer, 'Genetic basis of schizophrenia', The Lancet, Vol. 346, No. 8976, 9 September, 1995, pp. 678-683.

The basis for the genetic theory is a well-observed pattern of psychiatric morbidity in the families of people with schizophrenia.[115] Psychiatric textbooks routinely present these concordance rates as being established genetic risk factors for schizophrenia.

Genetic Risk for Schizophrenia

Identical twin affected	50%
Fraternal twin affected	15%
Brother or sister affected	10%
One parent affected	15%
Both parents affected	35%
Second-degree relative affected	2-3%
No affected relative	1%

Source: Norman L. Keltner, 'Schizophrenia and Other Psychoses', in Norman L. Keltner, Lee Hilyard Schwecke and Carol E. Bostrom, eds., Psychiatric Nursing, Mosby, St. Louis, 1995, p. 368.

These concordance rates, however, can also support arguments for an environmental cause. In cases, for instance, where one or both parents have schizophrenia it can be argued that it is not the transmission of genes that passes the condition on to offspring but rather bad parenting. Similarly, if one child develops schizophrenia due to an environmental cause in family life then it is likely other children in the family will also be affected in the same way.[116]

This exploitation of concordance research by advocates for an environmental cause has prompted genetic researchers to explore the concordance rates of siblings who have been reared separately. The adoption studies that are most frequently cited, however, are quite old dating back to the 1960s and 1970s. Of these the most important was published in 1975 by a team led by Seymour Kety.[117]

The Kety study was sponsored by the National Institute for Mental Health in the United States and involved locating Danish schizophrenics who had been adopted as children, before their schizophrenia had become apparent. Denmark was chosen because of the efficiency of the official system of record-keeping. The second step to the investigation required locating and psychiatrically assessing the biological relatives of the adopted schizophrenics to see if there was a higher incidence of schizophrenia amongst them than there was amongst the relatives of a control group of

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[115] S. L. Varma, A. M. Zain and S. Singh, 'Psychiatric morbidity in the first-degree relatives of schizophrenic patients', American Journal of Medical Genetics, Vol. 74, No. 1, 21 February, 1997, pp. 7-11.

[116] P. J. Tienari and L. C. Wynne, 'Adoption studies of schizophrenia', Annals of Medicine, Vol. 26, No. 4, August, 1994, pp. 233-237.

[117] S. S. Kety, D. Rosenthal, P. H. Wender, et al, 'Mental illness in the biologic and adoptive families of adoptive individuals who have become schizophrenic: a preliminary report based on psychiatric interviews', in R. Fieve, D. Rosenthal and H. Brill, eds, Genetic Research in Psychiatry, John Hopkins University Press, Baltimore, 1975, pp. 147-165.

non-schizophrenic adoptees. The findings showed that schizophrenia and 'schizophrenia spectrum' disorders were more prevalent amongst the relatives of the schizophrenic adoptees and this was taken to be positive evidence of a genetic link for schizophrenia.[118]

However, this study has been severely criticised. Claims have been made that there was a double sleight-of-hand involved in the presentation of the results.[119] Whereas there was no increase in schizophrenia amongst the close relatives — mothers, fathers, sisters and brothers — there was an increase amongst paternal half-siblings. But this increased incidence was all contained within one large family.

Furthermore, the increase was largely due to the inclusion of 'schizophrenia spectrum' disorders in the comparative study. DSM II was used to define the relevant range of disorders and this edition of the manual included a category, which has since been dropped, called 'latent schizophrenia'. This was a supposed tendency towards schizophrenia without a history of psychosis. It was similar to the Soviet concept of 'sluggish schizophrenia' and almost anybody could be fitted into it. Fourteen of a total of eighteen half-siblings diagnosed with schizophrenia spectrum disorders only had this latent form.

Breggin argues that the four cases of full-blown schizophrenia within the one family could be best explained by an environmental cause, perhaps sexual abuse. He dismisses the cases of latent schizophrenia as irrelevancies.

Adoption studies have also been severely criticised because of concerns that adoption practices may have been influenced by eugenics policies in the countries where the most influential adoption studies were carried out. Jay Joseph[120] has analysed the development of eugenics policies, and their translation into laws sanctioning sterilisation of mentally ill people, in Denmark, Finland and Oregon. He argues that a number of adoption studies undertaken in Denmark in the 1960s and 1970s, others undertaken in Finland in the 1970s and 1980s, and another in Oregon in the 1960s, all used schizophrenic adoptees who were placed for adoption at a time when eugenics legislation was in force.

Jay's argument is that adoption agencies would have been influenced by eugenics policies in regard to the type of families children were placed in. If a child who was given up for adoption had a natural mother who had schizophrenia, or if there was any known mental illness in the family of the mother or father, then the child would most likely have been placed in a family of lower

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[118] Black et al., op.cit., p. 379.
[119] Breggin, op.cit., pp. 118-121.
[120] Jay Joseph, 'The Genetic Theory of Schizophrenia: A Critical Overview', Ethical Human Sciences and Services, Vol. 1, No. 2, 1999, pp. 119- 145.

socio/economic status. This in turn would have increased the likelihood that these children would have been reared in a more stressful environment, and they would therefore have been more likely to develop schizophrenia. Jay concluded:

> If all schizophrenia adoption studies are considered in the context of the social and political environments from which they obtain their participants, the following can be concluded: The great majority of adoptees under investigation by the schizophrenia adoption studies were given up for adoption at a time when the compulsory sterilisation of "schizophrenics" for eugenics purposes was permitted by law in the country or state in which their adoptions took place (Denmark, Finland, Oregon). Leaving aside all other problems, the evidence suggesting that the selective placement of adoptees occurred in these studies is reason enough to disregard their findings until evidence can be produced that such placements did not occur.[121]

The most persuasive evidence for a genetic link comes from the study of concordance rates in twins. The twin method is widely used to determine whether a particular trait has any connection with genetic inheritance. The method involves comparing the concordance rates of monozygotic (MZ) twins (one egg, identical) with concordance rates of dizygotic (DZ) twins (two egg, fraternal). In schizophrenia studies these comparisons are confined to twins where both members have been reared together. The object is to determine whether the 100% genetic concordance of monozygotic twins, compared to the 50% genetic concordance of dizygotic twins, reflects in similarly divergent concordance rates for schizophrenia. The results are quite impressive.

When the results are pooled of 14 twin studies conducted between 1928 and 1998 the pooled concordance rates for monozygotic twins is 44% compared to 9% for dizygotic twins.[122] At first glance this offers convincing evidence for a genetic factor in schizophrenia. However, critics of the studies point to two major problems with this approach. The first is that the studies themselves all have methodological problems. The most serious of these methodological problems are the familiar ones concerning diagnostic criteria and subjective methods of diagnosis for schizophrenia. It is pointed out that all of these studies were undertaken by researchers who set out to confirm the genetic hypothesis and that in most cases non-blinded diagnostic procedures were used. Given the subjective nature of schizophrenia diagnosis this gives rise to concerns that diagnoses might have been inflated for one group and deflated for the other.

The other major problem concerns the possibility that concordance rates might have been confounded by environmental factors. Analysis of the studies has shown that, although there is no

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[121] Ibid., p. 136.
[122] Ibid., p. 123.

sex-link to the genetic hypothesis for schizophrenia, the twin studies show a distinct pattern of sex-linked concordance: "female MZ pairs were more concordant than male MZ pairs; female DZs were more concordant than male DZs; DZ same sex-sex twins were more concordant than opposite-sexed DZs; and DZ twins were more concordant than ordinary siblings, despite sharing the same genetic relationship".[123]

These results suggest that something other than genes is responsible for the concordance patterns. It is suggested that the most likely explanation is that family environments tend to compress the identities of twins so that they experience a phenomena called "ego-fusion".[124] This occurs as a result of families endeavouring to treat them equally which often means duplication of clothing and experience. The more similar the twins are, whether MZs or same sex, the more duplicated their experience tends to be. The result is that if one twin experiences madness the other will have a propensity to follow depending on their history of duplicated experience: "It is therefore concluded that there is no reason to accept that the twin method measures anything other than the environmental differences distinguishing identical and fraternal twins".[125]

It is not only the gene sceptics who warn about excessive optimism pervading the genetic quest. Some of the leading researchers in the field also find it occasionally necessary to tone down the rhetoric in order to keep the issue in perspective:

> The search for genes of major effect in schizophrenia, however, is premised not so much on hard evidence that they exist, as on the absence of evidence that they do not. .... Recent work suggests that such genes of major effect exist in other common disorders, but linkage studies in schizophrenia must still be regarded as acts of faith. Clearly, therefore, we must explore both avenues and continue to apply to schizophrenia a range of sophisticated techniques that do justice to the intricacies of the problem.[126]

Theories of an Environmental/Experiential Aetiology

Enviro/experiential theories are very different to biological theories. They begin from an altogether different premise. Biological theories largely disregard or discount any concept of mind, preferring instead to assume that abnormalities of thought are caused by abnormalities in brain functioning. Enviro/experiential theories, on the other hand, are based on the concept that the symptoms of schizophrenia are manifestations of a person's mind, rather than their brain.[127]

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[123] Ibid., p. 126.
[124] Ibid.
[125] Ibid., pp. 136-137.
[126] Michael Owen and Peter McGuffin, 'The molecular genetics of schizophrenia: blind alleys, acts of faith, and difficult science', British Medical Journal, Vol. 305, No. 6855, 19 September, 1992, pp. 664-666.
[127] Silvano Arieti, 'From schizophrenia to creativity', American Journal of Psychotherapy, Vol. 33, No. 4, October, 1979, pp. 490-505.

A major weakness of the enviro/experiential side of the debate is the inability to adequately describe what is meant by mind.[128] Since the biological determinists have claimed as their own territory all the biological organs on which mind might depend[129] the enviro/experiential determinists are only left with a vague concept of a disembodied collection of memories and feelings which apparently malfunctions when it has been assembled, or is required to operate, under inclement conditions. Although most of the enviro/experiential theories are accessible to some form of scientific investigation they normally do not demonstrate the same kind of hard scientific edge as the biological theories.

An individual's experience within the family environment is the matrix from which many enviro/experiential theories of aetiology arise.[130] Family life, particularly family life during infancy and early childhood, is often seen as the place and time where the fundamental characteristics of a person's mind are formed. It is malformations of mental characteristics that are variously blamed by the environmental determinists as causes of schizophrenia. However, although it is usually experience gained within the family which is hypothesised as the cause of schizophrenia, because this experience is in the past, and can not be re-run to achieve a more desirable outcome, the solutions devised by talking therapists are not always directed at readjusting family relationships.[131]

Talking therapies can be divided into a number of different types: there are those which assume the fault is a problem of intrapsychic development: i.e. that it is in the psychological makeup of the schizophrenic, and that it can be corrected by making the affected individual more aware of the problem;[132] there are those which assume the fault is with the schizophrenic's family, or a particular member of the family, and can be corrected by making adjustments to family structures;[133] and there are those which assume the fault is in a competitive/hostile social environment to which the schizophrenic is maladapted.[134]

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[128] Michael D. Lemonick, 'Glimpses of the Mind', Time, Vol. 146, No. 3, July 17, 1995, pp. 44-53.
[129] Judith Hooper, 'Targeting the brain: the 3-lb. organ that rules the body is finally giving up its secrets. Goodbye, Oedipus', Time, Vol. 148, No. 14, 1996, pp. 46-51.
[130] Ian R. Falloon, 'Family stress and schizophrenia: Theory and practice', Psychiatric Clinics of North America, Vol. 9, No. 1, March, 1986, pp. 165-182.
[131] J. Zubin, 'Suiting therapeutic intervention to the scientific models of aetiology', British Journal of Psychiatry Supplement, July 1989, pp. 9-14.
[132] D. B. Diamond, 'The fate of the ego in contemporary psychiatry with particular reference to etiologic theories of schizophrenia', Psychiatry, Vol. 6, No. 1, 1997, pp. 67-88.
[133] Matti Keinanen, Hilkka Virtanen and Anne Kaljonen, 'Structural couplings between individual development and the epigenesis of family relations in schizophrenia: An eight-year follow-up', Contemporary Family Therapy, Vol. 11, No. 2, 1989, pp. 75-88.
[134] Howard N. Garb, 'Race bias, social class bias, and gender bias in clinical judgment', Clinical Psychology Science and Practice, Vol. 4, No. 2, 1997, pp. 99-120.

Developmental Theories

The first type of therapy often assumes some kind of developmental hypothesis for the aetiology of schizophrenia. Sigmund Freud's ideas about schizophrenia form the basis for many developmental theories.[135] Freud based many of his ideas about schizophrenia on an analysis of a distinguished jurist named Daniel Paul Schreber who developed a psychosis in mid-life.[136] Schreber's psychosis involved paranoid delusions of persecution which Freud interpreted as manifestations of latent homosexual attraction to his father.

Freud theorised that because the homosexual attraction was too unbearable for Schreber to acknowledge it had instead been transformed into hatred for his father. Hatred for his father, in turn, caused Schreber to see him as a persecutor. This simple rationale became the basis for a general explanation of the paranoia which is commonly associated with schizophrenia. Similarly, the equally common phenomena of hallucinations was explained conversely as "wish fulfilment of unbearable ideas rejected by the ego".[137]

Freud observed that problems with interpersonal relationships were associated with schizophrenia and he applied his libido theory to find an explanation. He speculated that a schizophrenic's inability to properly relate to other people is caused by the withdrawal of libido into the self. This withdrawal of libido is a regression to the infantilism of primary process thinking and the ensuing focus on self gives rise to delusions and hallucinations as compensation for the deficit in interpersonal relations.[138]

Freud believed that schizophrenics could not be treated by psychoanalytical means because their inability to form interpersonal relationships meant they were unable to engage in transference, which is essential to the process of psychoanalysis.[139] This declaration of untreatability led to the marginalisation of Freud's theories on schizophrenia and to a quest by successive theorists for a developmental hypothesis that would support some form of therapeutic intervention.

Harry Stack Sullivan's theories extended Freud's considerably. Although Freud and Sullivan had similar approaches to schizophrenia they came from very different cultural backgrounds that led them to different conclusions. While Freud came from a Jewish, middle class, sophisticated

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[135] B. B. Wolman, 'New ideas on mental disorders', American Journal of Psychotherapy, Vol. 31, No. 4, October, 1977, pp. 546-560.

[136] Sigmund Freud, 'Psycho-analytic Notes on an Autobiographical Account of a Case of Paranoia (Dementia Paranoides)', The Standard Edition of the Complete Psychological Works of Sigmund Freud, Vol. 12, Hogarth Press, London, 1958, pp. 3-82.

[137] Black et al., op.cit., p. 380.

[138] Ibid.

[139] Laurie M. Post, 'A study of the dilemmas involved in work with schizophrenic patients', Psychotherapy Theory, Research and Practice, Vol. 19, No. 2, 1982, pp. 205-218.

Viennese background, which gave him a detached, scholarly, perspective, with an observer's status that was not so much a personal attribute as one he had inherited with his ethnic identity, Sullivan, on the other hand, had grown up as a lonely outsider in a rural area of the United States.[140]

Sullivan's personal struggle through childhood and adolescence, with a cold rejecting mother and a shy distant father, apparently gave him a certain empathy with the schizophrenics he encountered in his adult career as a psychiatrist in the 1920s to 1940s.[141] Although he began his career accepting many of Freud's beliefs his technique of closely observing and empathising with his patients led to many revisions.[142]

Sullivan came to believe that many psychiatric problems were due to the fraud and hypocrisy which he thought were endemic to society. He believed the Oedipal complex, for instance, on which Freud's theory of schizophrenia was based, "must be recognised as a distortion, not a biological development, in the normal male child. It is a fraudulent symbol situation commonly the result of multiple vicious features of our domestic culture".[143]

Sullivan's version of the developmental theory conceived by Freud was that schizophrenia is the outcome of interpersonal problems. To Sullivan personality development is dependent on a person monitoring the appraisal of significant people. When significant people are perceived to have a negative opinion, or when there are no significant people in a person's life, then there is a risk of developing a personality deficit or schizophrenia.[144] Sullivan's theory contributed to a change in the focus of the developmental theories so that schizophrenia was no longer seen as an intrapsychic problem but instead became an environmental problem.[145]

Family Environment

On the non-biological side of the psychiatric dichotomy over schizophrenia the theories that have evolved about environmental causes for schizophrenia usually focus either on the family environment or the larger social environment. Those which focus on the family environment demonstrate a clear pattern of evolution.[146] At first mothers were the focus of research under the

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[140] Jane Pearce, 'Harry Stack Sullivan: Theory and practice', Managed Environment Systems, Vol. 14, No. 4, July, 1984, pp. 159-166.

[141] Clara Thompson, 'Harry Stack Sullivan, the Man', in Harry Stack Sullivan, Schizophrenia as a Human Process, W. W. Norton and Company, New York, 1962, p. xxxii.

[142] Harry Stack Sullivan, Schizophrenia as a Human Process, W. W. Norton and Company, New York, 1962, p. 147.

[143] Harry Stack Sullivan, 'Erogenous Maturation', Psychoanalytical Review, Vol. 61, 1926, pp. 1-15.

[144] Pearce, op.cit., pp. 159-166.

[145] Black et al., op.cit., p. 380.

[146] Yrjo O. Alanen, 'An attempt to integrate the individual-psychological and interactional concepts of the origins of schizophrenia', British Journal of Psychiatry, Vol. 164, Supplement 23, April, 1994, pp. 56-61.

assumption that some fault in the mother/child bonding was the cause of schizophrenia.[147] Then the focus of research moved on to examine marital relationships between mothers and fathers with the assumption that some kind of distortion in these relationships might impact on children and cause schizophrenia.[148] Finally researchers began to take account of the family environment as a whole assuming that any member of a family, or all the members of a family together, might somehow create conditions of stress that produced schizophrenia in a family member.[149]

The term 'schizophrenogenic mother' was first coined in 1948 by a psychiatrist named Frieda Fromm-Reichman: "The schizophrenic is painfully distrustful and resentful of other people due to the severe early warp and rejection he encountered in important people in his infancy and childhood, as a rule mainly the schizophrenogenic mother."[150] There were two ideas embodied in the concept of the schizophrenogenic mother which made this terminology a powerful message for the times. These two ideas were the notions of maternal rejection and maternal over-protection.[151]

The post World War II period was one of rapid cultural change where attention was often focussed on the relationship between mothers and children.[152] Uncertainty had developed about the quality of mother-infant bonding in industrialised countries as a result of a variety of factors like a rising divorce rate, adolescent pregnancies, and working mothers who left their babies with surrogate minders. The satisfying pre-war cultural image of a young mother successfully nurturing an infant was being eroded.[153]

In these circumstances it became popular for social commentators to blame mothers for any troubles children had with social adjustment and also any problems the society might have with maladjusted or delinquent children. It was discovered that mothers could be conveniently blamed for many kinds of social stresses. As a result the stereotypical suburban housewife was often portrayed as a frustrated, repressed, disturbed, martyred, never satisfied, unhappy woman — a demanding, nagging, shrewish wife — and a rejecting, over-protecting, dominating mother.[154]

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147 James S. Grotstein, 'Deciphering the schizophrenic experience', Psychoanalytic Inquiry, Vol. 3, No. 1, 1983, pp. 37-69.

148 C. Peter Rosenbaum, The Meaning of Madness: Symptomatology, Sociology, Biology and Therapy of the Schizophrenias, Science House, New York, 1970, pp. 140-163.

149 Anthony Clare, Psychiatry in Dissent: Controversial issues in thought and practice, Tavistock Publications, London, 1976, pp. 189-196.

150 Frieda Fromm-Reichman, 'Notes on the Development of Treatment of Schizophrenics by Psychoanalytic Psychotherapy', Psychiatry, Vol. 11, 1948, pp. 263-273.

151 Gordon Parker, 'Re-searching the schizophrenogenic mother', Journal of Nervous and Mental Disease, Vol. 170, No. 8, August, 1982, pp. 452-462.

152 Carol Eadie Hartwell, 'The schizophrenogenic mother concept in American psychiatry', Psychiatry Interpersonal and Biological Processes, Vol. 59, No. 3, August, 1996, pp. 274-297.

153 J. Kagan, Unstable Ideas, Harvard University Press, Cambridge, 1989, p. 80.

154 Betty Friedan, The Feminine Mystique, Bantam Books, New York, 1964.

This changing cultural identity of women enhanced the significance of the role of motherhood in the eyes of psychiatrists. As faith in the competence of mothers declined advice was increasingly sought from professionals on matters concerning nurturing and child care. But mothers remained powerful. Although they might be perceived as failing to produce healthy well-adjusted children they could still wreak social havoc by producing deviants.[155]

On top of this, at the same time as women were increasingly seen as becoming dominant, masculine power was thought to be on the decline. Popular literature complained about the emasculation of men due to factors like the bureaucratisation of work, the rise of the corporate 'man in the grey flannel suit,' and the demise of individualism. The newly enfeebled men of the 1950s had castrating women waiting for them in every suburban home.[156]

The significance of these cultural trends to the discovery of the idea of the schizophrenogenic mother, despite the discoverer herself being a woman, is that most of the psychiatric researchers who pursued this line of research were men. The general notion they were pursuing was of a dominating, over-protective, but basically rejecting mother who somehow induced a schizophrenic reaction in her offspring.[157] A considerable number of uncontrolled studies were undertaken that seemed to confirm this premise. These were usually in the form of interview studies or case-record studies without control groups.[158]

However, by the early 1980s the concept of the schizophrenogenic mother had definitely run its course. A researcher could argue in 1982, after reviewing the literature on the subject, that "[t]he most plausible explanation is that there is no *sui generis* schizophrenogenic mother; instead, there is a parental type distinguished by hostile, critical, and intrusive style and it is not particularly over-represented in the parents of schizophrenics."[159] With further shifts in cultural values over the intervening years it had become apparent that only a small percentage of women who might arguably fit the criteria of schizophrenogenic mother had actually produced schizophrenic children. Conversely, many schizophrenics were found to have mothers who did not fit the criteria.

By the early 1980s some psychiatric researchers were ready to include the schizophrenogenic mother on a list with other "dangerous psychosocial hypotheses" that supposedly had retarded the

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[155] Stella Chess, 'The "blame the mother" ideology', International Journal of Mental Health, Vol. 11, Nos. 1-2, 1982, pp. 95-107.

[156] B. Ehrenreich, and D. English, For Her Own Good, Doubleday, New York, 1987, p. 241.

[157] Frank Simmers and Froma Walsh, 'The nature of the symbiotic bond between mother and schizophrenic', American Journal of Orthopsychiatry, Vol. 47, No. 3, July, 1977, pp. 484-494.

[158] See for example, A. C. W. Whal, 'Some Antecedent Factors in the Family Histories of 392 Schizophrenics', American Journal of Psychiatry, Vol. 110, 1954, pp. 668-676.

[159] Parker, op.cit., pp. 452-462.

progress of psychiatry.[160] Yet despite the hostility that had developed against the idea within the psychiatric profession, and despite the lack of evidence to support it, the schizophrenogenic mother was still being presented as a viable concept in psychology textbooks up to the end of the 1980s.[161]

Double Bind Theory

The schizophrenogenic mother was only one of a number of possible complications in the childhoods of schizophrenics that might account for the disorder. The search for a distortion in family experience that could be described in finite terms, measured, and positively connected with schizophrenics was something of a holy grail for psychiatrists in the decades following World War II. Perhaps the most seductive idea that arose from this quest was the 'double bind' theory.[162]

In 1956 Gregory Bateson and his colleagues at Stanford University published a "report on a research project which has been formulating and testing a broad, systematic view of the nature, etiology, and therapy of schizophrenia".[163] The double bind theory which arose from this research was based in communications theories. Bateson's view was that the inner turmoil experienced by schizophrenics is associated with a habit of routinely communicating in metaphorical language without first flagging that a metaphor was being used: "The peculiarity of the schizophrenic is not that he uses metaphors, but that he uses *unlabelled* metaphors. He has special difficulty in handling signals of that class whose members assign Logical Types to other signals".[164]

The "Logical Types" referred to are derived from a theory of Bertram Russell which argues that there is a discontinuity between a class and its members. Bateson had adapted Russell's theory to the realm of ideas and to the communication of ideas.[165] Bateson argued that there are numerous classes of ideas used in human communication which each dictate different modes of communication within their fields of influence. Examples given of these classes of ideas are play, non-play, fantasy, sacrament and metaphor.[166] According to the theory it is imperative that a discontinuity prevails between the class and the members: i.e. between a meta idea like 'play' and the communication of playful ideas:

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[160] Humphry Osmond, 'Dangerous psychosocial hypotheses', Journal of Orthomolecular Psychiatry, Vol. 11, No. 3, 1982, pp. 216-218.

[161] Otto F. Wahl, 'Schizophrenogenic parenting in abnormal psychology textbooks', Teaching of Psychology, Vol. 16, No. 1, February, 1989, pp. 31-33.

[162] John H. Weakland, 'The development and significance of the double-bind theory', Japanese Journal of Family Psychology, Vol. 6, December, 1992, pp. 25-38.

[163] Gregory Bateson, Don D. Jackson, Jay Haley, and John Weakland, 'Towards a Theory of Schizophrenia', Behavioral Science, Vol. 1, Number 4, October 1956, reproduced in Milton M. Berger, Beyond the Double Bind, Brunner/Maze, New York, 1978, pp. 3-27.

[164] Ibid., p. 8.

[165] Leena Roy and Suby Roy, 'Does the theory of logical types inform a theory of communication?', Journal of Genetic Psychology, Vol. 148, No. 4, December, 1987, pp. 519-525.

[166] Bateson et al., op.cit., p. 6

> Although in formal logic there is an attempt to maintain this discontinuity between a class and its members, we argue that in the psychology of real communications this discontinuity is continually and inevitably breached and that *a priori* we must expect a pathology to occur in the human organism when certain formal patterns of the breaching occur in the communication between mother and child. We shall argue that this pathology at its extreme will have symptoms whose formal characteristics would lead the pathology to be classified as a schizophrenia.[167]

The reference to the mother/child relationship was only a convenient example of a relationship in which this type of breaching could occur. Communications which can give rise to pathogenic breaching are theoretically possible with any close member of a family. "The hypothesis which we offer is that the sequences of this kind of external experience of the patient are responsible for the inner conflicts of Logical Typing. For such unresolvable sequences of experiences, we use the term 'double bind'."[168]

Unlike the somewhat obscure reasoning of the theoretical packaging of Logical Types, the description of the double bind situation, from which schizophrenics were assumed to contract their mental pathology, was persuasive and logical. Six ingredients were specified for a double bind situation.

(1) The "victim", i.e. the schizophrenic person, must have had a childhood relationship with one or more family members whose communication techniques induced inner conflict.
(2) The double bind communications were a repeated rather than a single traumatic experience. The repetition is necessary in order to induce in the victim a habitual expectation of double bind forms of communication.
(3) The double bind communication first takes the form of a primary negative injunction. "This may take either of two forms: (a) 'Do not do so and so, or I will punish you', or (b) 'If you do not do so and so, I will punish you'."[169] The punishment that is threatened might take the form of either withdrawal of love or the expression of anger.
(4) The primary negative injunction is followed by a secondary injunction which conflicts with the first. The secondary injunction is on a more abstract level and, although like the first it is enforced by an implication of punishment, it is usually communicated in a more subtle fashion which might involve nonverbal means like posture, gesture or tone of voice.
(5) A tertiary negative injunction prevents the victim from escaping from the situation.
(6) When the victim has learned to anticipate double bind patterns in all communications the complete set of ingredients is no longer necessary and "almost any part of the double bind sequence

[167] Ibid.
[168] Ibid., p. 9.
[169] Ibid.

may then be sufficient to precipitate rage or panic. The pattern of conflicting injunctions may even be taken over by hallucinatory voices."[170]

Although the mother/child relationship is not the only one with double bind potential Bateson and his colleagues prefer to illustrate their theory by depicting mothers:

> we hypothesise that the mother of a schizophrenic will be simultaneously expressing at least two orders of message. These orders can be roughly characterised as (a) hostile or withdrawing behaviour that is aroused whenever the child approaches her, and (b) simulated loving or approaching behaviour which is aroused when the child responds to her hostile and withdrawing behaviour, as a way of denying she is withdrawing.[171]

Bateson gave an example of the double bind situation taken from observations made during clinical practice. This example is much-cited and has been frequently used by other writers to illustrate in summary the mechanism of double bind.

> A young man who had fairly well recovered from an acute schizophrenic episode was visited in the hospital by his mother. He was glad to see her and impulsively put his arm around her shoulders, whereupon she stiffened. He withdrew his arm and she asked, "Don't you love me any more?" He then blushed, and she said, "Dear you must not be so easily embarrassed and afraid of your feelings." The patient was able to stay with her only a few minutes and following her departure he assaulted an aide and was put in the tubs.[172]

The simplicity of the double bind argument was very appealing but, while many schizophrenics appeared to have a history of some kind of double bind situation,[173] so did many non-schizophrenics. In fact, the popularity of the idea might be attributable to the fact that most people have experienced the frustration of a double bind relationship with a person of authority at some time in their lives, and can easily recognise the problem.

Perhaps it happens in many situations where one person is required to exercise authority over somebody else. In a work place, for instance, egalitarian camaraderie might be encouraged while at the same time a person in authority might need to conceal incompetence, or a disinterest in a subordinate, under a veneer of authoritarian role playing. The consequence for the subordinate

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[170] Ibid., p. 10.
[171] Ibid., p. 15.
[172] Ibid., p. 18.
[173] Susan L. Jones, 'The "damned if you do and damned if you don't" concept: The double bind as a tested theoretical formulation', Perspectives in Psychiatric Care, Vol. 15, No. 4, 1977, pp. 162-169.

person could be a double bind situation in which a show of camaraderie is met with an authoritarian response when the person in authority perceives that an apparent lack of discipline might expose managerial incompetence. A submissive approach by the subordinate person, on the other hand, might be met by teasing jocularity, or disdain, by the person in authority, as a response to a perceived excess of obsequiousness.

Many people can respond to the frustration of the subordinate person in this type of situation, perhaps through having past experience of it themselves. However, this only begs the question: if the double bind is a factor in the aetiology of schizophrenia, why are only some people vulnerable? The answer to this question proved to be too elusive[174] and by the end of the 1970s researchers had largely moved on to focus on other hypotheses.

Family Stress

Another line of research, explored over the same time period as the schizophrenogenic mother and the double bind, concerned theories that distortions in the marital relationship of a mother and father might impact adversely on a child and cause schizophrenia. Theodore Lidz[175] was one of the leading researchers in this field. Lidz hypothesised that there are two different kinds of distortion in parental marital relationships which alternatively selected boys and girls as candidates for schizophrenia.

The first kind of distortion Lidz called "marital skew".[176] This occurs when one parent yields to the idiosyncrasies and over-bearing dominance of the other. This situation was thought to be particularly relevant to the cause of schizophrenia in male children.[177] In families with marital skew the dominant parent was thought to be usually the mother and in contrast to her the father was perceived as being a weak passive type of person who provided a poor role model for his son. In these families the mother was believed to turn away from her husband as a source of emotional comfort and to fixate on her son in a search for solace. The combination of a poor paternal role model and a fixated, dominant and often eccentric mother, was thought to be a frequent cause of schizophrenia in male children.[178]

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[174] David M. Dush and Marvin Brodsky, 'Effects and implications of the experimental double bind', Psychological Reports, Vol. 48, No. 3, June, 1981, pp. 895-900.

[175] Theodore Lidz, Stephen Fleck and Alice R. Cornelison, Schizophrenia and the Family, International Universities Press, New York, 1965.

[176] Ibid., pp. 142-145.

[177] Ibid., p. 249.

[178] Julian Leff, 'Social and Psychological Causes of the Acute Attack', in J. K. Wing, ed., Schizophrenia: Towards a New Synthesis, Academic Press, London, 1978, p. 143.

The cause of schizophrenia in female children was thought to be usually caused by a variation on this theme and due to a condition called "marital schism".[179] Marital schism occurred when there was conflict between the mother and father but neither party yielded to the other.[180] In this situation each partner was constantly striving to satisfy their own needs while ignoring the other partner's needs. This perpetual battle for ascendancy between parents inevitably involved the children as the parents competed for their affections and enrolled them as supporters. The schismatic family was thought to be far more selective in causing schizophrenia in females than in males.[181]

In both the skewed and schismatic types of family Lidz hypothesised that children are reared in an abnormal environment because there is an absence of parental cooperation and the normal delineation's between generations are not observed. He believed these conditions could lead to anxieties in children involving the induction of incestuous feelings. Lidz's basic approach to schizophrenia was similar to Bateson's in that he believed it is the manifestation of inappropriate behaviour that has been learned in the family environment.[182]

Although a considerable amount of research has been conducted over the years to test Lidz's theories, most of the results have not supported them. Perhaps the most thorough of these studies was carried out by Sharan in the middle 1960s.[183] Sharan's study involved 12 families with a schizophrenic son, and 12 with a schizophrenic daughter. Each of these groups of families were symmetrically balanced by dividing them into 6 families with a healthy sibling of the same sex as the schizophrenic, and six families with a healthy sibling of the opposite sex to the schizophrenic.

The core of the study required each family to complete a questionnaire. This was done under tape-recorded supervision with the family members assembled in groups of three — firstly comprising the two parents and the schizophrenic child — and secondly comprising the two parents and the healthy child. Different questionnaires were used each time and answering the questionnaires required discussion amongst each separate group. The objective was to score the individual parents for indications of dominance by assessing how often one parent's answers became the group's decision. Support between individuals was also scored by recording how often supportive and non-supportive remarks were directed at individual family members.

Sharan could find no clear pattern confirming Lidz's theories about either the relationship of parental marital skew to schizophrenia in male children or the relationship of marital schism to

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[179] Lidz et al., op.cit., p. 264.
[180] Ibid., pp. 136-142.
[181] Leff, op.cit., p. 143.
[182] Theodore Lidz, 'Patients whose children became schizophrenic', Journal of Nervous and Mental Disease, Vol. 172, No. 7, July, 1984, pp. 408-411.
[183] Leff, op.cit., p. 144.

female schizophrenia. Nor could he uncover any clear pattern of parental support or non-support for schizophrenic children as compared to their healthy siblings.[184]

Another line of research assuming an environmental aetiology involved studying the families of schizophrenics as whole units to see if the aetiology of schizophrenia could be found in group deviance, rather than in the deviations of individual members.[185] One influential hypothesis postulated that when there are mutual expectations amongst family members of reciprocal fulfilment, which have no basis in reality, the false atmosphere in the family is often accompanied by disjointed forms of communication and irrational shifts in the focus of family attention. This situation was thought to give rise to conditions where all family communications were polarised between superficiality at one pole and fragmented, disjointed thinking at the other. These conditions in turn influenced the cognitive development of children who were subjected to them. Schizophrenia was hypothesised as one of the outcomes.[186]

More recently deviance in the language of schizophrenic patients has been compared to similar deviations in the language of their parents in the hope that some light may been shed on aetiology by understanding how the language deviations of schizophrenia are learned.[187] Research is now also turning to focus on positively identifying a link between genetic vulnerability and environmental stresses in family life that might trigger schizophrenia. One recent study compared adopted children of schizophrenic mothers, who were thought to have an enhanced genetic risk, with a control group of adoptees with normal genetic risk profiles, to see whether there were any consistent patterns of thought deviation in the two groups of children that might be associated with environmental triggers. However, no clear pattern has emerged from this research yet.[188]

Stress in the family environment has been extensively researched as both an originating cause of schizophrenia and as a factor in relapse. Two types of stresses have largely been the focus of attention: the ambient stresses of everyday life and abnormal stresses brought on by important life events like a death in the family.[189] Ambient stresses are often measured in the form of 'expressed emotion' (EE). Schizophrenics are thought to come from families with higher than normal levels of

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[184] Ibid., p. 145.
[185] Deborah J. Lieber, 'Parental focus of attention in a videotape feedback task as a function of hypothesised risk for offspring schizophrenia', Family Process, Vol. 16, No. 4, December, 1977, pp. 467-475.
[186] Leff, op.cit., p. 145.
[187] N. M. Docherty, 'Communication deviance, attention, and schizotypy in parents of schizophrenic patients', Journal of Nervous and Mental Disease, Vol. 181, No. 12, December, 1993, pp. 750-756.
[188] K. E. Wahlberg, L. C. Wynne, H. Oja, P. Keskitalo, L. Pykalainen, I. Lahti, J. Moring, M. Naarala, A. Sorri, M. Seitamaa, K. Laksy, J. Kolassa and P. Tienari, 'Gene-environment interaction in vulnerability to schizophrenia: findings from the Finnish Adoptive Family Study of Schizophrenia', American Journal of Psychiatry, Vo. 154, No. 3, March, 1997, pp. 355-362.
[189] Ian R. Falloon, 'Family stress and schizophrenia: Theory and practice', Psychiatric Clinics of North America, Vol. 9, No. 1, March, 1986, pp. 165-182.

EE and some researchers claim that when there is a high level of EE between a mother and child, for instance, this deepens the emotional bond, but it also puts the child at higher risk of developing schizophrenia.[190] Comparisons have been drawn between the key components of EE research — critical comments and the over involvement of family members in the schizophrenic's life — with the rejection and over-protection that was formerly attributed to the schizophrenogenic mother.[191] This suggests that EE researchers might be merely extending the schizophrenogenic concept from the mother to the whole family.

One type of important event that has attracted research attention is the death of a grandparent within two years of the birth of a schizophrenic person. Researchers found that a grandparent of 41% of a large sample of schizophrenics had died within this period. This rate was significantly higher than the rate in a control group of normal people and it was hypothesised that the additional stresses introduced into family life by two major events — a birth followed by death, or vice-versa — might confuse the parenting and mourning roles. In this situation a bereaved parent might be emotionally unavailable to an infant and a spouse or, alternatively, a child might be used as a distraction from mourning and as a result could inadvertently absorb the painful feelings of the bereaved parent.[192]

Social Stress

Comparisons have been made between schizophrenics and normal people to determine whether personal experience of negative life events is a significant factor. It has been found that schizophrenics have a higher incidence of these negative experiences in the areas of work, health, family and social relationships.[193] It has also been observed that the incidence of schizophrenia is higher in urban centres but recent research has been unable to confirm that the stress of city living is implicated as a cause.[194]

Stresses arising from social class have been a subject for research into the aetiology of schizophrenia. There has been consistent evidence of a higher incidence of schizophrenia amongst people of lower social classes. Two principal hypotheses have been presented to account for this. The first is that stresses induced by social conditions like poverty, unemployment and welfare

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[190] William L. Cook, Angus M. Strachan, Michael J. Goldstein and David J. Miklowitz, 'Expressed emotion and reciprocal affective relationships in families of disturbed adolescents', Family Process, Vol. 28, No. 3, September, 1989, pp. 337-348.

[191] Parker, op.cit., pp. 452-462.

[192] Froma W. Walsh, 'Concurrent grandparent death and birth of schizophrenic offspring: An intriguing finding', Family Process, Vol. 17, No. 4, December, 1978, pp. 457-463.

[193] Graziano Canton and Ida G. Fraccon, 'Life events and schizophrenia: A replication', Acta Psychiatrica Scandinavica, Vol. 71, No. 3, March, 1985, pp. 211-216.

[194] Hugh Freeman, 'Schizophrenia and city residence', British Journal of Psychiatry, Vol. 164, Supplement 23, April, 1994, pp. 39-50.

dependency can cause schizophrenia.[195] The second is that the confused state of mind experienced by schizophrenics makes them socially uncompetitive which in turn leads to downward social drift.[196]

Stresses arising from racial identity have also been explored as possibly contributing to the cause of schizophrenia. A recent study conducted in the UK compared the incidence and outcomes of schizophrenia amongst whites, Afro-Caribbeans and Asians. Afro-Caribbeans and Asian women were found to have a higher incidence of schizophrenia and Afro-Caribbeans were more disabled by the experience. The only significant variable the researchers could find to explain these results, other than racial identity, was a higher level of unemployment amongst the Afro-Caribbeans.[197]

An increasing trend amongst psychiatric practitioners is to advocate an end to the mind/brain dichotomy[198] and to argue that a more sophisticated approach to schizophrenia is an assumption that the aetiology has both biological and environmental components. This is sometimes called a biopsychosocial approach and it is often vaguely endorsed by psychiatrists who are currently treating schizophrenics with a mixture of drugs and talking therapy.[199] However, it is not a position that appears to be particularly attractive to aetiological researchers because it tends to multiply the already vast field of variables.

Conclusion

The medical model is split into two fundamentally different approaches to the aetiology of schizophrenia — the biological approach and the environmental/experiential approach. The biological approach assumes that schizophrenic symptoms are manifestations of brain disorder. Speculations about underlying brain abnormalities are various and they in turn support a range of theories about the possible causes of the brain abnormalities. The enviro/experiential approach, on the other hand, assumes that people with schizophrenia have normal brains and that the symptoms of abnormality are manifestations of developmental problems or stressful experience. There is a wide variety of environmental hypotheses which argue for different types of stressful experience as being the universal or principal cause of schizophrenia.

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[195] P. N. Wold and S. Soled, 'The family history of mental illness and welfare dependence', Journal of Clinical Psychiatry, Vol. 39, No. 4, April, 1978, pp. 328-31.

[196] Leigh Silverton and S. Mednick, 'Class drift and schizophrenia', Acta Psychiatrica Scandinavica, Vol. 70, No. 4, October, 1984, pp. 304-309.

[197] D. Bhugra, J. Leff, R. Mallett, G. Der, B. Corridan and S. Rudge, 'Incidence and outcome of schizophrenia in whites, African-Caribbeans and Asians in London', Psychological Medicine, Vol. 27, No. 4, July, 1997, pp. 791-798.

[198] Philippe Khouri, 'Continuum versus dichotomy in theories of schizophrenia', Schizophrenia-Bulletin, Vol. 3 No. 2, 1977, pp. 262-267.

[199] G. O. Gabbard, 'Mind and brain in psychiatric treatment', Bulletin of the Menninger Clinic, Vol. 58, No. 4, 1994, pp. 427-446.

When the various aetiological theories are critically analysed none of them provide convincing evidence of any movement towards early closure of the aetiological controversy. Although the biological side of the dichotomy is currently in the ascendancy this has not always been the case in the past. So long as the aetiological controversy remains open it is quite possible there might be a future shift in the emphasis of research back to the enviro/experiential side.

In the early 1990s Theodore Sarbin wrote about his long career in pursuit of definitive evidence that might explain the cause of schizophrenia. He related how in the early 1970s he had analysed the various aetiological theories that had been postulated up to that time. He said that "the rise and fall of theories of schizophrenia led me to conclude that such theories have a half-life of about five years. The conclusion applied to somatic theories and psychological theories alike."[200] Sarbin went on to cite more recent research that came to similar conclusions but which found that biological theories have shorter life-spans than psychological theories.

The sheer number of hypotheses, both past and present, on both sides of the dichotomy, is evidence of deep confusion within the medical model on the subject. This confusion is reflected in the proliferation of therapies but it is not always evident in the confidence levels of individual psychiatrists, particularly those who are willing to impose their treatments on involuntary patients.

[200] Theodore R. Sarbin, 'Towards the Obsolescence of the Schizophrenia Hypothesis', Journal of Mind and Behaviour, Vol. 11, No. 3 and 4, 1990, p. 264.