Mapping paradigm change in Australian psychiatry 1950-2004

Julie Henderson
School of Nursing and Midwifery, Flinders University
Julie.Henderson@flinders.edu.au

Abstract

This paper maps the extent of paradigm change in the understanding of the causes of mental illness in Australian psychiatry from 1950. The period was chosen as it coincided with deinstitutionalisation which began in Australia in 1955. The data presented in this paper comes from two sources, the recommended textbooks used to teach the psychiatric component of the medical degree at six Australian universities since 1950 and the official journal of the Royal Australian and New Zealand College of Psychiatry, the Australian and New Zealand Journal of Psychiatry which was first published in 1967. Both sources were subject to content analysis of the models of aetiology used in the texts. The models of aetiology were divided into social and biological models, where social models are those in which the source of pathology is found in the social environment and social relations and biological models find the root of pathology in individual biological factors. The findings show that Australian psychiatry was dominated by social models of aetiology during the period from 1950 to 1985 with these models reaching the height of their popularity between 1965 and 1980. There was a growing interest in biological models of aetiology from 1985, which escalated in the 1990s and continues into the present.

Introduction

While there is an extensive literature exploring the development of psychiatric knowledges in the United States and in Europe, little critical attention has been given to the development of psychiatric knowledges in Australia. A number of Australian historians have explored the relationship between psychiatry and the mental hygiene movement in the earlier part of the twentieth century, but the period from 1950 onwards has been largely unexplored. This paper addresses this gap in knowledge through mapping the relationship between social and biological explanations of mental illness in Australia. The methodology used is content analysis of the models of aetiology used in the Australian and New Zealand Journal of Psychiatry and the recommended textbooks used to teach psychiatry to medical students in Australian universities between 1950 and 1999.
A history of paradigm change

The recent history of psychiatry is one of disparate understandings of both the symptoms and the aetiology of disease entities developed in the nineteenth century (Gilman 1988). Psychiatry had two competing aetiological paradigms from this time, one favouring models of social causation and the other biological causes. Psychiatry is generally understood to have moved towards social explanations of mental illness in the interwar years. Abbott (1988) argues that psychiatry found the root of psychopathology in unsuccessful adjustment to the social environment from the 1930s. This approach had three fundamental assumptions. First, social factors are only important in so far as they affect the individual. Second, that “any violation of social rules...signified mental problems” and third, that mental problems should be managed at an individual rather than a social level (Abbott 1988: 298).

The dominant model of aetiology in the interwar period was a psychobiological model (Sashbin 1990). Psychobiology as developed by Adolf Meyer, understood mental illness as a “faulty response or substitution of an insufficient or protective or evasive or mutilated attempt at adjustment” rather than a “hypothetical cell alteration which we cannot reach or prove” (Meyer cited in Caplan 1998: 106-107). Mental illnesses were viewed as arising from a failure of adjustment leading to the development of ”bad habits”, and the role of psychotherapy was that of inculcating better habitual ways of reacting (Gilman 1988: 219).

Psychobiology was preceded by a psychoanalytic model of aetiology in the post-war period (Sashbin 1990). The defining feature of a psychoanalytical approach was the localisation of neuroses in the personal history of the patient (Prior 1991). Psychopathology was understood as arising from a failure to successfully negotiate innate developmental stages, resulting in fixation in, or regression to, earlier developmental stages (Gilman 1988). Mental illness was viewed as a process of the mind that was
precipitated by difficulties in early childhood. This understanding expanded the scope of psychiatry through shifting the focus of management to the family and early childhood experiences. The universality of these experiences meant that all people could be subject to mental illness, creating a continuum between normal and abnormal behaviour, with the separation between pathological and normal behaviour based on the degree of pathology (Horwitz 2002).

Biological models of psychopathology re-emerged in the 1970s (Gilman 1988). Abbott (1988) associates the resurgence of biological models of aetiology with an attempt by psychiatry to retain jurisdiction over mental health in the face of competition from other professions through realignment with medicine. Psychiatry was losing professional dominance over the mental health field, due in part, to the movement of mental health services into the community where professions with expertise in the social could claim a greater role. The redefinition of mental illness as a biological condition not only allowed for the reassertion of medical dominance over allied health professions, but also solidified governmental support for psychiatry as a ‘scientific’ profession (Cooksley and Brown 1998). Gilman (1988) identifies two major focuses for a renewed interest in biology: the role of biochemistry and the role of genetics in the transmission of disease. Biochemical explanations of mental illness primarily focus upon failures in the neurotransmitters, chemicals that aid the transmission of information between neurones. The new genetics focuses on molecular genetics and the identification of the genes responsible for mental illness. This presumes a causal rather than a correlational relationship between genes and mental illness (Joseph 2003). While molecular geneticists initially believed that a specific gene could be found for mental disorders they have more recently adopted a polygenic theory in which the aetiology of mental illness is believed to be found in the interaction of multiple genes, each of which have a “small effect that increase susceptibility” to disorders (Joseph 2003: 284).

Despite general agreement, there were regional differences in adoption of the models of aetiology. American psychiatry embraced social models of aetiology from the 1930s and
biological models of aetiology from the 1970s. Baldessarini (1998) conducted a content
analysis of The American Journal of Psychiatry between 1944 and 1994 for articles
concerned with biological aetiology and treatment methods. He found a reduction in
articles adopting biological models of aetiology from 1961 to 1981, accompanied by
fewer articles discussing biological treatment modalities between 1966 and 1975. This
trend reversed from the mid 1970s. Between 1976 and 1980, 38% of all articles
published in The American Journal of Psychiatry adopted a biological theme compared
with nineteen percent for the previous five years.

British psychiatry in contrast, is generally viewed as being more “empirical, eclectic,
practical and based on sound clinical observation” (Crammond 1981: 203). Moncrieff
and Crawford (2001) argue that British psychiatry maintained an interest in biological
psychiatry throughout the twentieth century. They demonstrate, through content analysis
of the British Journal of Psychiatry between 1905 and 1995, that this journal published
articles concerned with biological therapies through the twentieth century. There were
however, a growing number of articles about social psychiatry and epidemiology in 1975
while articles concerned with neuropsychiatry and community care dominated the journal

Australian psychiatry is understood to owe more to British than American psychiatry, due
to the extent to which British psychiatrists established and comprised Australian
psychiatry. In practice however, social and biological model of aetiology have often co-
existed in Australian psychiatry. Cohen (1999) argues that while Australian psychiatrists
maintained a belief in a biological basis for mental illness in the interwar years they also
expressed concern about the role of environmental factors on the mental well-being of the
individual. This was due in part, to experiences with treating shell shock in the First
World War, which contributed to a belief that mental illness arose from psychological
maladjustment (Garton 1997/98). By the 1950s there were two factions within psychiatry
those who sought inherited and physical causes for psychopathology and those who
emphasised psychological mechanisms and reactions to external events of psychological
significance (Davis 1957: 42). This tension was still evident in the early 1960s when the *Medical Journal of Australia* noted an ongoing conflict between analysts and somaticists about the scientific validity of psychoanalysis (*Medical Journal of Australia* 1966).

**Methods**

The data for this paper comes from the recommended psychiatric textbooks used within six Australian medical schools from 1950-1999 and from the *Australian and New Zealand Journal of Psychiatry*. The textbooks provide access to the taken-for-granted assumptions of the profession while the journal demonstrates, through the peer review process, the ideas which currently have credence with the profession (Kuhn 1996). The textbooks were also used due to the comparatively recent development of psychiatry as a clinical specialty in Australia. The Royal Australian and New Zealand College of Psychiatry was first formed in 1946 and the *Australian and New Zealand Journal of Psychiatry* not published until 1967 (Rubenstein and Rubenstein 1996).

The textbooks were drawn from medical schools at the University of Adelaide, Flinders University, Monash University, the University of Sydney, University of Tasmania and the University of Western Australia and were identified through reading lists published in university calendars. Data was collected from 1950 through to 1999 when reading lists were no longer being published in most university calendars. The search for recommended texts generated a list of 91 different textbooks. This list was reduced by the use of only one edition of each textbook. The sample was further reduced by removing texts that do not address the aetiology of mental illness and those textbooks which were used only once, at one campus only. This left a sample of 55 textbooks.

The second source of data was the *Australian and New Zealand Journal of Psychiatry*, which was published quarterly between 1967-1995, six times a year from 1996-2002 and monthly from 2003. The initial sample consisted of all articles published within the *Australian and New Zealand Journal of Psychiatry* to the end of 2004. The sample was
reduced to 1007 articles after examining the content of each article. The articles that were retained were those which contained a discussion of the aetiology of mental illness. These articles were initially identified through reading the abstract. Any articles that were in doubt were read in full.

Content analysis of the models of aetiology used in both sources was undertaken. The textbooks were subject to manifest coding as the models of aetiology used within the textbooks were cited and easily identifiable. The journal articles were subject to latent coding, as coding depended upon the coder's knowledge of the key concepts, theorists and focus of each aetiological model (Neumann 2000). A number of the journal articles cited social causes of mental illness without reference to a theoretical framework. These articles were coded to reflect the aspect of the environment which was seen as contributing to poor mental health. Both sources were then divided into either a social or biological category according to the focus of the model of aetiology. Textbooks using psychoanalytical, environmental, behavioural, family systems and socialisation theory as an explanation for behaviour were allocated to the social category, while those focussing upon heredity or genetics, neurophysiology, neurochemistry, endocrinology and ethology were viewed as having a biological focus. Psychobiological and biophysiological models that contain both physical and social elements were allocated to both. The journal articles were allocated to a social category if they adopted a psychoanalytic, family systems or behavioural approach or if the root of pathology is found in the family or social events or in trauma arising from natural disasters. Articles were viewed as adopting a biological focus if psychopathology was associated with genetics, neurophysiology, neurochemistry, illicit drug use, prescription drug use, other toxins, secondary to another illness, sociobiology, as having an immunological cause, innate temperament and perinatal events and trauma. Articles adopting a biopsychosocial approach were allocated to both.

**Paradigm change in Australia?**

Tables 1 and 2 demonstrate the shift between a social and biological paradigm within Australian psychiatry. Table 1 shows that the textbooks used contained both social and
biological models of aetiology, evident in the number of texts that have an 'eclectic' approach. There was a marked tendency in the period 1965 and 1974 to use textbooks that drew solely upon social explanations of pathology. Between 1965-1969, nine out of the ten recommended texts had a social focus while five (50%) dealt exclusively with social models of aetiology. A similar trend is evident in the period 1970-1974, when nineteen of the twenty recommended texts had a social focus, nine (45%) of which focussed solely on social models of aetiology. This can be accounted for by texts adopting a psychoanalytical focus (nine or 90% of all texts in 1965-69, and sixteen or 80% of all texts in 1970-74).

Table 1: Social or biological focus? - Recommended psychiatric texts 1950-1999

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Biological</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Social</td>
<td>2</td>
<td>3</td>
<td>5</td>
<td>5</td>
<td>9</td>
<td>5</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Eclectic</td>
<td>2</td>
<td>4</td>
<td>2</td>
<td>5</td>
<td>11</td>
<td>7</td>
<td>12</td>
<td>10</td>
<td>9</td>
<td>9</td>
</tr>
<tr>
<td>Total texts</td>
<td>4</td>
<td>8</td>
<td>8</td>
<td>10</td>
<td>20</td>
<td>13</td>
<td>16</td>
<td>11</td>
<td>10</td>
<td>10</td>
</tr>
</tbody>
</table>

By 1985 more of the recommended texts contained discussions of biological models of aetiology rather than social models. Of these, most discussed more than one model. The majority of texts contained both social and biological models of aetiology, with five texts in the period between 1985-1994 containing a discussion of psychoanalytic theories and seven of the ten texts used in 1985-1989 exploring environmental risk factors.

A similar pattern emerges in *The Australian and New Zealand Journal of Psychiatry* (refer Table 2). There is a noticeable tendency away from the publication of articles using biological models of aetiology in the period 1971-1980. During this period 26 percent of all articles dealing with the aetiology of mental illness favoured biological explanations. Of these, the majority dealt with physiological changes to the brain, neurochemistry and the impact of illicit drugs on mental health. The majority of articles in this period (139 of 188 [74%]) explored social causes of mental illness. Of these 67 (35.6%) found the root of pathology within family relations while 75 (39.9%) listed other aspects of the social environment, most commonly issues arising from the socio-
economic status of the individual or family.

Table 2: Social or biological? - *Australian and New Zealand Journal of Psychiatry*

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Biological</td>
<td>23 (35%)</td>
<td>20 (26%)</td>
<td>29 (26%)</td>
<td>34 (35%)</td>
<td>51 (42%)</td>
<td>64 (46%)</td>
<td>137 (53%)</td>
<td>67 (49%)</td>
</tr>
<tr>
<td>Social</td>
<td>43 (65%)</td>
<td>57 (74%)</td>
<td>82 (74%)</td>
<td>64 (65%)</td>
<td>70 (58%)</td>
<td>75 (54%)</td>
<td>122 (47%)</td>
<td>69 (51%)</td>
</tr>
<tr>
<td>Total-Aetiological</td>
<td>66 (100%)</td>
<td>77 (100%)</td>
<td>111 (100%)</td>
<td>98 (100%)</td>
<td>121 (100%)</td>
<td>139 (100%)</td>
<td>259 (100%)</td>
<td>136 (100%)</td>
</tr>
</tbody>
</table>

The period from 1980 saw a steady growth in the number of articles published in this journal with a biological focus. In the period 1996-2000, 53 percent (N=137) of published articles adopted biological models of aetiology (refer Table 2). Of these 33 (24%) discussed genetic causes of mental illness while 42 (30.6%) discussed neurophysiological causes of mental illness. Social models of aetiology were still evident in 122 articles (47%). Of these 52 (20%) dealt with the role of the family in pathology. An ongoing interest in social factors can be accounted for by epidemiological articles, which explore the impact of social risk factors upon mental health, by 23 articles about post traumatic stress disorder (PTSD), seventeen exploring the impact of child or sexual abuse and 21 about youth suicide.

A shift in psychiatric paradigm can also be demonstrated by the changing construction of the aetiology for a specific disorder, in this case schizophrenia. Table 3 shows a comparative analysis of those factors associated with the aetiology of schizophrenia in the periods 1971-1975 and 1996-2000 in the *Australian and New Zealand Journal of Psychiatry*. These periods were chosen as they reflect the height of a social and biological paradigm shift within the journal. This table provides data about the risk factors, markers and the aetiology of schizophrenia.

Risk factors are social factors seen as contributing to the probability of developing mental illness. In the period 1971-1975, the identified risk factors focus almost exclusively upon the relationship between parenting and mental health pathology. This relationship
is usually a causal one, and the data is often presented as a series of assertions without recourse to statistical evidence. Pathology is primarily seen as arising from family disruption, thus Metcalf states that:

There are the children who did not have a relationship with one mother figure in the early years and particularly before 2 years. Permanent crippling of personality growth caused by institutionalised and multiple mothering may make it impossible for this child to form a therapeutic relationship (1973: 281).

By 1996-2000, the relationship between social factors and mental health pathology was correlational, identified through the statistical correlation of social factors with schizophrenia. Many of the risk factors identified in this period relate to environmental factors such as place of birth, month of birth and class. A family history of mental illness is viewed as a risk factor but is understood as arising from a greater genetic risk of the inheritance of schizophrenia. Thus, McGrath notes that:

of the presently known risk factors for schizophrenia, family history is by far the most robust; however, as yet, no single gene has been identified....it is plausible that several genes, each of small effect, contribute to the risk of schizophrenia (2000: S61).

Markers are early indicators of the potential to develop schizophrenia, evident in biological differences in those with the condition (Russell 1995). “Biological markers can be defined as biochemical, physiological or anatomical traits that are specific to particular conditions” (Coplov and Crook 2000: S108). The identification of markers is seen as a means of detecting those at risk of developing schizophrenia at a later date, although not all people having biological markers develop the condition. In practice, many biological markers appear in a number of psychiatric conditions as well as in the healthy population (McNeil and Cantor-Graae 2000).
Table 3: Factors associated with the aetiology of schizophrenia for the periods 1971-1975 and 1996-2000

<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Risk factors</strong></td>
<td><strong>Risk factors</strong></td>
</tr>
<tr>
<td>family environment</td>
<td>urban birth</td>
</tr>
<tr>
<td>social isolation</td>
<td>month of birth</td>
</tr>
<tr>
<td>marital status</td>
<td>family history of mental illness</td>
</tr>
<tr>
<td>parental age</td>
<td>class</td>
</tr>
<tr>
<td>loss of a parent</td>
<td>stressful life events</td>
</tr>
<tr>
<td>failure to adjust to maternal role after childbirth</td>
<td>expressed emotion</td>
</tr>
<tr>
<td>social upheaval</td>
<td>disrupted family environments</td>
</tr>
<tr>
<td><strong>Markers</strong></td>
<td><strong>Markers</strong></td>
</tr>
<tr>
<td>repetitive mannerisms</td>
<td>delayed development</td>
</tr>
<tr>
<td>rigidity of thinking</td>
<td>impaired attention</td>
</tr>
<tr>
<td>failure of the executory function of the brainstem reticular function</td>
<td>neuromotor alterations</td>
</tr>
<tr>
<td><strong>Aetiology</strong></td>
<td><strong>Aetiology</strong></td>
</tr>
<tr>
<td>amphetamine use</td>
<td>genetics</td>
</tr>
<tr>
<td>maternal deprivation</td>
<td>pre-natal events</td>
</tr>
<tr>
<td>genetic</td>
<td>- poor maternal nutrition</td>
</tr>
<tr>
<td>autoimmune</td>
<td>- mid-trimester viruses</td>
</tr>
<tr>
<td></td>
<td>- pre-natal brain damage</td>
</tr>
<tr>
<td></td>
<td>- alcohol use in the first trimester</td>
</tr>
<tr>
<td></td>
<td>obstetric complications</td>
</tr>
<tr>
<td></td>
<td>head injuries</td>
</tr>
<tr>
<td></td>
<td>cannabis and stimulant use</td>
</tr>
<tr>
<td></td>
<td>convulsive disorders</td>
</tr>
<tr>
<td></td>
<td>encephalitis</td>
</tr>
</tbody>
</table>

In the period 1971-1975 the potential to develop schizophrenia was more likely to be associated with behaviours that were seen as evidence of a prodromal phase in which the
person develops symptoms. The development of brain imaging and genetic technologies allowed for the identification of a number of biological events that appear more commonly in those people diagnosed with schizophrenia. These events include brain changes, impaired attention and delayed development. As with social risk factors, no direct causal link has been made between these markers and the development of schizophrenia. The consensus is however, that technology will allow a causal link to be made in time.

The final aspect of the table lists aetiological causes of schizophrenia discussed in the journal in 1971-1975 and 1996-2000. The table demonstrates a marked shift towards biological explanations for the aetiology of schizophrenia. In the earlier period little attention is paid to the aetiology of any major mental illnesses. Each of the factors identified in Table 3 appear in one article only, although maternal deprivation was viewed as an aetiological factor for other disorders including mood and neurotic disorders. In the period 1996-2000, all factors identified as contributing to the aetiology of mental illness have a biological basis. McGrath (2000: S59) views a genetic history of schizophrenia, pregnancy and birth complications, prenatal virus infections and prenatal nutrition as risk-modifying factors as each “appear[s] to operate within the causal chain” having a direct impact upon mental health.

Conclusion

This paper has mapped the dominant psychiatric paradigm in Australia from 1950 to 2004 through content analysis of the models of aetiology discussed in the recommended psychiatric textbooks used within six Australian medical schools from 1950-1999 and the Australian and New Zealand Journal of Psychiatry. The central finding was that there was a marked movement towards the use and publication of texts adopting a social, and in particular psychoanalytical focus, from 1965 through to 1985 after which the trend reversed towards use of texts adopting a biological focus. The most frequently cited biological causes of mental illness were genetics and neurophysiological changes. This trend is reflected in the understanding of the causes of schizophrenia. Earlier literature
often implicated family relations while recent articles focus almost exclusively upon biological factors. Despite this, paradigm shift is not complete. Many of the psychiatric texts retain chapters on psychoanalysis and half of the articles published in the *Australian and New Zealand Journal of Psychiatry* in 1996-2000 cite the role of social factors in causing mental illness, although these factors are often viewed as risk rather than causative factors. These findings tally with earlier work which suggests that biological and social models of aetiology have often co-existed in Australian psychiatry.

References


