New Zealand Journal of Psychology, 1994, 23, 18-27

The Emerging Biology Of Social Intervention In The Treatment Of Psychological Disorders

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*Parts of this paper were presented at the 'Issues in Psychiatry' Satellite Symposium of the Annual Meeting of the Royal Australian and New Zealand College of Psychiatrists, Dunedin, New Zealand, 1990.
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Drug therapy and social intervention are often regarded as separate approaches in the treatment of psychological disorders. If a materialist theory of mind is accepted, then social intervention and drug therapy must be regarded as having a common site of action, ie. the central nervous system (CNS). Since continuous change is one of the salient characteristics of social behaviour, it follows that for a materialist theory of mind to be true, the CNS must be continuously modified as part of its normal function. There is abundant evidence from neuroscience to suggest that continuous, rapidly-induced plasticity is a normal function of the CNS in response to changing environmental conditions. There seems no reason why social stimuli should not also stimulate this kind of CNS plasticity. A biological approach to social intervention should lead to greater emphasis on research into aspects of biological function which are most relevant to social behaviour; such an approach may ultimately lead to new insights into the biological mechanisms which mediate the effects of social intervention on psychological disorders.

Psychological disorders such as schizophrenia and depression are commonly treated with some combination of drug therapy and psychotherapy (egs., see Goldstein & Kopeikin, 1981; Falloon, Boyd, McGill, Williamson, Razani, Moss, Gilderman & Simpson, 1985; Erickson, Beiser, Iacono, Fleming & Lin, 1989; Glass, Katz, Schnitzer, Knapp, Frank, & Gunderson, 1989; Herrman, 1989; Keck, Cohen, Baldessarini & McElroy, 1989; Miller, 1989; Reynolds, 1989; Crow, 1990; Kavanagh, 1992; Post, 1992 for reviews). These two forms of treatment are used in conjunction with one another, in recognition of the contribution of 'biological' and 'social' factors to the induction and/or maintenance of psychological disorders (egs. Kavanagh, 1992; Post, 1992). However, 'biological' and 'social' treatments are often regarded as separate approaches (see Herrman, 1989; Ciompi, 1989; Sabelli & Carlson-Sabelli, 1989 for reviews); this may lead to an underestimation of the interaction between the two forms of treatment. For example, a schizophrenic with little social support from family members may be less responsive to antipsychotic medication (egs. Falloon, Boyd, McGill, Williamson, Razani, Moss, Gilderman & Simpson, 1985; Kavanagh, 1992; see Glick, 1992; Kane, 1992; Goldstein, 1992 for reviews).

Although many psychiatrists and psychologists accept the materialist view that the mind is identical with, or a function of, the central nervous system (CNS), and therefore that 'social' interventions must exert their effects through neural consequences, some prominent advocates of mentalist philosophy (ie. that the mind is something separate from the CNS) still exist (eg.

Eccles, 1989). Even materialists may appear to support a mentalist philosophy of mind as a result of the pervasive influence of mentalist concepts in psychiatric and everyday language. The argument of this review is that mentalist approaches to psychiatry and psychology lead to the conceptual isolation of the 'biological' and 'social' treatment perspectives. One consequence of this division may be the neglect of important interactions between biological and social factors in the causes and treatments of psychological disorders (Kavanagh, 1992; Post, 1992). Furthermore, it is argued that materialism is the most parsimonious resolution of the mind-body problem and from this viewpoint social intervention in psychological disorders must be interpreted as exerting its effects through the modification of CNS function.

In a recent review, Ciompi (1989) has argued for the need to reconcile the biological and social perspectives on disorders such as schizophrenia and has suggested that one phenomenon which integrates these perspectives is neural plasticity. One of the most salient characteristics of social function is the capacity to adapt to changing circumstances in interactions with other people, ie. plasticity. This ability to adapt to new social circumstances is probably an important component of what we mean by the term 'consciousness', since the resulting change in behaviour is an indication that an organism is responding to the external environment (Llinas, 1989). CNS plasticity is well-documented in connection with learning and memory and recovery from lesions (see Marshall, 1984; Agranoff, 1989 for reviews). If it is accepted that the CNS is changing continuously in order to encode and store experiences in the world (as must be assumed in a materialist philosophy of mind), then behavioural plasticity in response to social stimuli would be a particular subtype of neural plasticity. From this viewpoint, social experiences must change CNS function, and therefore biological (eg. drug therapy) and social treatments (eg. psychotherapy) of psychological disorders must have a common site of action—the CNS (Post, 1992).

The Mind-Body Problem

The 'mind-body problem' is the problem of whether the mind is of a different substance to the body; whether it is something different to and more than, the material CNS (see Hospers, 1967 for a review). The mind-body problem is an ancient philosophical problem, but even today it splits psychology and psychiatry into two factions: those who believe that the mind is more than the material CNS (ie. mentalists) and those who believe that it is either identical with, or a function of, the CNS (ie. materialists) (see Hospers, 1967; Churchland, 1990 for reviews; cf. Armstrong, 1968; Armstrong, 1970; Eccles, 1989 and Llinas, 1989). There are various versions of the two philosophical positions and in some of these (eg. double aspect theory) the distinction between materialism and mentalism becomes subtle (Hospers, 1967). In this review I will address only the most prominent forms of materialism and mentalism.

The dualist-interactionist philosophy of Descartes is probably the dominant form of mentalism and has been promoted and popularized by the Nobel Prize-winning neurophysiologist Sir John Eccles (eg. Eccles, 1989). Dualist-interactionism holds that the mind is separate from the CNS and is of a non-physical substance, but that the two interact such that mental events can influence the CNS and vica-versa (Hospers, 1967).

One of the most prominent forms of materialist philosophy is the identity theory formulated by the philosopher, David Armstrong (Armstrong, 1968; Armstrong, 1970). Identity theory holds that the mind is identical with the CNS such that a mental event like an emotion is equivalent to a particular CNS state (yet to be identified): without the CNS state what we take to be the mental state will not occur (Armstrong, 1968; Armstrong, 1970; see Borst, 1970 for a review).

Neurological and neuropsychological research has shown that the integrity of the CNS is necessary for normal behaviour and most sophisticated advocates of Dualist-interactionism would not question that conclusion: they would acknowledge that CNS function is necessary for the bodily expression of mental activity, therefore be-havioural impairment following brain damage does not constitute evidence against the Dualist-interactionist position (Eccles, 1989). Because proponents of Identity theory *and* Dualist-interactionism may regard the integrity of the CNS as necessary for normal behaviour, it is impossible to use experimental evidence from neuroscience to resolve the mind-body problem. The debate between Identity theory and Dualist-interactionism must take place largely on philosophical grounds.

Both the Dualist-interactionist and Identity theory positions have been criticised for philosophical reasons. Criticisms of Dualist-interactionism include failure to specify the proper-ties of mental events which are more than merely the absence of physical characteristics: if mental events do not have dimension and location in space like physical entities, then what properties do they have (see Hospers, 1967 for a review)? Dualist-interactionism has also been criticised for not specifying how the mental world and the CNS might interact and for the violation of the law of conservation of energy which would occur when energy is lost from the physical world in its interaction with the mental world (see Hospers, 1967 for a review).

Identity theory has been criticised mainly on the grounds that it is inconceivable that the complexity and richness of mental experience can be identical with the mere electrochemical activity of neurons (Eccles, 1989): for example, how can the CNS generate the myriad of states necessary for our constantly changing beliefs and emotions? Usually, Identity theorists are not specific about the CNS states which they believe to underlie mental experience (Armstrong, 1970). Dualists often regard this as an evasion of the important question of how CNS activity can be identical with mental experience (Eccles, 1989).

An alternative form of materialism, known as functionalism, argues that the mind is not *identical* with the CNS but is a *function* of it (eg. Llinas, 1989; see Churchland, 1990 for a review). From this viewpoint, to compare the mind and the CNS is analogous to comparing the image generated by a television with the electronic components which make the television functional. The functions of the television have their own internal dynamics (ie. one function causally linked to another) and cannot be reduced to its electronic circuitry: it is possible to completely understand the electronic substrates of a television and yet have no idea what a television picture looks like. Nonetheless, the contents and structure of the electronic circuitry are entirely responsible for the function of the television and constrain its behavioural possibilities.

In the same way, the functionalist form of materialism argues that mental activity and behaviour derive entirely from the content and structure of the CNS, however they cannot be regarded as identical with the CNS because they are functions of it (Llinas, 1989; Churchland, 1990). It is conceivable that a neuroscientist could under-stand the structure and function of all the specific components of the CNS without understanding mental experiences like embarrassment or elation, which arise from patterns of activity across the entire CNS (Churchland, 1990). The functionalist alternative overcomes some of the common objections to identity theory. For the functionalist, mental events are not reducible to CNS circuitry and it is sensible to investigate causal connections between mental events independently of CNS microstructure (Churchland, 1990). Nonetheless, the structure and function of the CNS constrain mental activity at every point; therefore, a complete understanding of mental experience and behaviour could be achieved only by understanding both the CNS and its mental functions (in the same way that a television could be completely understood only by appreciating both its electronic circuitry and its global functions such as the image on the screen).

However, functionalism does not entirely overcome the problems of identity theory: if mental events are a function of the CNS, then surely on some global level, mental events must be identical with a complex pattern of electrochemical activity across various areas of the CNS? Otherwise the functionalist seems in danger of concluding that mental events are *more than* the function of the CNS, that they are indeed something independent of it. Although the identity theory and functionalism appear similar in some ways, the functionalist version of materialism has the advantage that it recognises mental events as a particular function which the CNS does not necessarily have to fulfil; therefore, mental events, while generated by the material CNS, are not simply reducible to it (see Churchland, 1990 for a discussion).

The functionalist form of materialism seems to be the most parsimonious philosophical account of the mind available. Firstly, the above criticisms show that Dualist-interactionism is a vague theory which cannot provide an account of even the most elementary aspects of the mind-CNS interaction which it claims is taking place (see Hospers, 1967 for a review). Secondly, the main argument against materialism, that it is inconceivable that complex mental states are generated by electrochemical activity within the CNS, is based on naive assumptions about the kinds of CNS events which are likely to be responsible for rapidly changing and complex mental events

(Armstrong, 1970; Llinas, 1989). Even though empirical neuropsychological evidence cannot be used to resolve the mind-body debate (since it can be interpreted as consistent with either Dualist-interactionism or Materialism), it is nonetheless useful to consider the kinds of CNS events which might be responsible for mental events. If materialism is accepted, then it follows that our continuously changing beliefs and emotions must be caused by continuously changing CNS states. Therefore, neural plasticity must be a normal function of the CNS. From this viewpoint, social influences are just one type of environmental stimulus which can modify CNS activity.

Synaptic Plasticity As Normal CNS Function

Baker (1985) has suggested that the way in which the term 'neural plasticity' is often used is vague and implies that a change in neural function is something unusual. The problem of defining 'neural plasticity' becomes apparent when considering what kinds of neural changes should be described by this term. Most neuroscientists would agree that the sprouting of new synapses ('reactive synaptogenesis') following a lesion should be considered 'plasticity' (see Flohr & Precht, 1981 for a review); but what about transient changes in the frequency of action potential discharges in a sensory neuron following the onset of a stimulus? These two kinds of changes in neural function are distinguished by: a) whether the structure, or only the function, of the CNS is altered (although on a molecular level the distinction between functional and structural changes is one of degree); b) the period of time over which the change occurs. If the term 'plasticity' is reserved for relatively large changes in the structure of the CNS which take place over days or weeks and are relatively long-term, then many of the neural changes which underlie learning and memory would be excluded. It is clear that the processes responsible for learning and memory, at least in the short-term, consist of changes in biochemical function which take place very rapidly (see Dunn, 1980; Nestler & Greengard, 1989; Klein, Sullivan, Skorupa & Santiago Aguilar, 1989; Walaas & Greengard, 1991, for reviews); these may be followed by longer term changes involving synthesis of new protein and major structural change (see Nestler & Greengard, 1989; Walaas & Greengard, 1991, for reviews). It is therefore difficult to exclude transient changes in neural function from the definition of 'neural plasticity'; since such transient changes are occurring continuously, it follows that neural plasticity must be occurring continuously.

What empirical evidence is there to support the notion that neural plasticity is a normal function of the CNS? Because chemical synapses are the major means of communication between neurons in the CNS, and because this communication can be modulated rapidly (see Schwartz, Costentin, Martres, Protais & Baudry, 1978; Nestler & Greengard, 1989; Klein, Sullivan, Skorupa & Santiago Aguilar, 1989; Walaas & Greengard, 1991; Post, 1992, for reviews), it is likely that the main form of plasticity occurring in the CNS is synaptic plasticity.

It is well documented that synapses are modifiable (ie. have the capacity to change from one state of operation to another) under conditions such as denervation (eg. Creese, Burt & Snyder, 1977), acute (eg. Chen, Stelzer, Kay & Wong, 1990) and chronic drug treatment (eg. Menkes & Aghajanian, 1981), altered sensory input (eg. Bear, Kleinschmidt, Gu & Singer, 1990) and enhanced synaptic activation (eg. Bliss & Lomo, 1973; see Abraham, 1988 for a review). In these cases a variety of factors controlling the operation of a synapse can be altered, for example: the amount of neurotransmitter synthesised and released; the affinity and number of postsynaptic receptors for the transmitter; the efficacy with which the binding of the transmitter to the postsynaptic binding site causes the opening of an associated ion channel; the rate of metabolic breakdown of the transmitter (see Burke, 1987 for a review). Some of these factors, like the affinity, efficacy and number of postsynaptic receptors, can change very rapidly (ie. on a timescale of seconds to minutes) in response to stimuli like prolonged continuous drug treatment (eg. desensitization; Huganir, Delcour, Greengard & Hess, 1986; see Nestler & Greengard, 1989 for a review) or repetitive synaptic activation (Bliss & Lomo, 1973; see Abraham, 1988 for a review).

Schwartz, Costentin, Martres, Protais & Baudry (1978) have suggested that synapses are on a continuum from hypo- to hyper-activity, their position on this continuum being constantly regulated according to environmental demands. This concept of the continuously regulated synapse is central to the use of experimental models of memory like long-term potentiation, where a transient increase in synaptic activation (induced

by electrical stimulation) causes pre- and post-synaptic plasticity which ultimately results in long-term increases in the efficacy of the synapse (ie. the same amount of presynaptic activation results in an increased postsynaptic response (eg. Bliss & Lomo, 1973; see Abraham, 1988; Collingridge & Lester, 1989 for reviews). A number of hypotheses have been advanced by theoretical neuroscientists in an attempt to explain how changes in global mental properties may arise from plasticity in neuronal populations: these include tensor network theory (eg. Llinas, 1989), synchronization of neuronal oscillations (egs. Engel, Kreiter, Konig & Singer, 1991; Llinas, Grace & Yarom, 1991) and chaos theory (eg. Gregson, 1992).

Recently, advances have been made in understanding the biochemical basis of these kinds of synaptic plasticity. Although long-term changes may involve the synthesis of new protein, changes on a timescale of seconds to minutes may involve the activation of second messenger systems which, via protein kinases, bring about the phosphorylation of existing protein within the neuron: the modification of protein in this way can produce many effects, including the opening or closing of ion channels, or the unmasking of previously silent binding sites, in a matter of seconds (see Nestler & Greengard, 1989; Walaas & Greengard, 1991 for reviews). Some of these changes may be encoded at the level of gene expression (see Post, 1992 for a review). There seems no reason why these types of biochemical mechanisms could not bring about rapid changes in CNS function elicited by social experiences, presenting themselves to the CNS as complex, multimodal sensory stimuli.

Evidence That 'Social' Factors Can Affect Synaptic Function

Currently there are few data available on specific relationships between social experience and CNS activity. In humans, brain imaging techniques such as positron emission tomography (PET; see Haxby, Grady, Ungerleider & Horwitz, 1991 for a review) have been used to show that the distribution of metabolic activity in the brain changes rapidly and in a highly complex manner during sensory experiences (egs. Posner, Petersen, Fox & Raichle, 1988; Haglund, Ojemann & Hochman, 1992; Demonet, Celsis, Nespoulous, Viallard, Marc-Vergnes & Rascol, 1992). However, few of these studies have examined the effects of variables which have a 'social' dimension, for example, anxiety-inducing stimuli (eg. Reivich, Gur & Alavi, 1983). Since neurons increase their energy consumption when their electrical activity increases, it is assumed that areas of high metabolic activity represent high levels of electrical activity (see Haxby, Grady, Ungerleider & Horwitz, 1991 for a review); since the primary means of neuronal communication is synaptic, it is probable that synaptic changes are occurring during these metabolic events.

There is a large literature linking stress to the development of potentially adverse neurochemical changes in the human nervous system (egs. Jacobs, Mason, Kosten, Wahby, Kasl & Ostfeld, 1986; Blanchard, Kolb, Prins, Gates & McCoy, 1991; Norman & Malla, 1993a,b; see Johnson, 1990 for a review). It is well established that stress induces the release of corticosteroids by the adrenal cortex due to increased release of pro-opiomelanocortin peptides by the pituitary gland (Nichols, Masters & Finch, 1990; see Anderson, Kant & De Souza, 1993 for a review). There is also substantial evidence that stress increases the release of some catecholamine neurotransmitters: for example, bereavement has been reported to be associated with elevated levels of adrenalin and noradrenalin (Jacobs, Mason, Kosten, Wahby, Kasl & Ostfeld, 1986) and combat veterans suffering from post-traumatic stress disorder have increased noradrenalin levels in blood plasma (Blanchard, Kolb, Prins, Gates & McCoy, 1991). Social stress has been implicated in the aetiology and progression of numerous psychological disorders, including schizophrenia and depression (see Norman and Malla, 1993a,b; Duman, 1993; Valentino, 1993; Akil, 1993; Watson, 1993 for reviews).

A number of physiological and biochemical studies using animals have attempted to address the effects of social stress on CNS synaptic activity more directly. At present, most of these studies concern the effects of various kinds of stress associated with the handling of rats or mice. One interpretational problem in the case of neonates is the possibility that synaptic changes due to handling may be caused by changes in ambient temperature rather than any social factors associated with the handling procedure (Bolden, Hambley, Johnston & Rogers, 1990). However, in the case of adult animals the stimulus for stress is more likely to be a social one related to fear of the handler and isolation from

other animals (Bolden, Hambley, Johnston & Rogers, 1990). The stress experienced by experimental animals in this situation may be related to the stress which humans experience in threatening social situations.

Meaney, Aitken, van Berkel, Bhatnagar & Sapolsky (1988) have reported that handling of rat neonates results in a permanent increase in the number of glucocorticoid receptors in the hippocampus. There is also increasing evidence from animal studies that stress can cause changes in the number of postsynaptic receptors for the inhibitory amino acid, gamma-amino-butyric acid (the GABA receptor) (see Bolden, Hambley, Johnston & Rogers, 1990 for a review). GABA is the major inhibitory transmitter in the brain and is believed to have an important role in the regulation of anxiety (Biggio, Concas, Mele & Corda, 1987). Alteration of the number of GABA receptors will alter synaptic transmission at GABAergic synapses: for example, an increase in the number ('up-regulation') of GABA receptors would cause an increase in the effect that GABA has on postsynaptic neurons (Bolden, Hambley, Johnston & Rogers, 1990). It has been shown that stressful events like handling (Biggio, Corda, Concas, Demontis, Rossetti & Gessa, 1981; Biggio, Concas, Mele & Corda, 1987; Bolden, Hambley, Johnston & Rogers, 1990), injection, or immersion in water (Skerrit, Trisdikoon & Johnston, 1981; Akinci & Johnston, 1993; Montpied, Weizman, Weizman, Kook, Morrow & Paul, 1993; Park, Hitri, Lukacs & Deutsch, 1993), can increase the number of GABA receptors in rats. Some of these changes may be induced within minutes (Skerrit, Trisdikoon & Johnston, 1981) and others have been shown to last more than 100 days after the offset of the stressful event (Bolden, Hambley, Johnston & Rogers, 1990). In chicks it has been shown that acute stress can cause an increase in the number of benzodiazepine binding sites on $GABA_A$ receptors (Martijena, Salvatierra & Arce, 1992). In rats exposed to unfamiliar environments, Primus and Kellog (1991) found that both the number and affinity of benzodiazepine binding sites increased. However, Morinan, Parker, Rich, Cariuk & Horton (1992) reported no change in the number or affinity of benzodiazepine binding sites following social isolation in rats. There is increasing evidence that the handling history of animals affects their response to anxiolytic drugs (Andrews & File, 1993).

Consistent with the results from human clinical studies indicating stress-induced changes in catecholamine release, recent animal studies suggest that stress may induce changes in CNS adrenergic receptors (Gorman & Dunn, 1993; Basso, Depiante-Depaoli, Cancela & Molina, 1993). Stress induced by restraint or swimming has also been reported to increase excitatory amino acid levels in the prefrontal cortex, hippocampus and striatum (Moghaddam, 1993). As with anxiolytic drugs, stress may also alter the response of animals to stimulants such as amphetamines (Badiani, Cabib, Stefano & Puglisi-Allegra, 1992).

Another relevant line of evidence involves the effects of stress on the electrophysiological model of memory known as long term potentiation (LTP) (Bliss and Lomo, 1973). In LTP, high frequency stimulation of an afferent pathway results in a long-term increase in the efficacy of the synapses between the pre- and post-synaptic neurons (see Abraham, 1988 for a review). This type of increase in synaptic efficacy through increased use is widely regarded as a feasible model for the type of neuronal changes which underlie the formation of memories (Bliss & Lomo, 1973; see Abraham, 1988; Collingridge & Lester, 1989 for reviews). There is evidence that stress induced in rats by tail shock or restraint results in a large impairment of hippocampal LTP induced in slice preparations maintained *in vitro* (Foy, Foy, Levine & Thompson, 1990; Shors, Seib, Levine & Thompson, 1989; Shors, Foy, Levine & Thompson, 1990). More relevant to the question of whether social factors can modulate synaptic activity is the recent demonstration that the impairment of LTP is greater when the shock is inescapable than when it is escapable: these results suggest that the helplessness which is experienced with inescapable shock has particularly detrimental effects on hippocampal plasticity (Shors, Seib, Levine & Thompson, 1989; Shors, Foy, Levine & Thompson, 1990). A similar impairment of plasticity may occur as a result of learned helplessness in social situations.

Although these experimental situations are far removed from complex human social situations, humans may nonetheless experience similar kinds of stress associated with social isolation and helplessness in social interactions. The available evidence indicates that stress associated with social situations may modify synaptic function in

humans; other forms of social experience may have similar effects. Psychosocial experiences may therefore be an important influence on the neural changes which underlie the development and maintenance of psychological disorders (eg. Post, 1992).

New Directions For Research Into The Aetiology And Treatment Of Psychological Disorders

If it is accepted that social experiences modify CNS function and that social intervention exerts its beneficial effects on psychological disorders through as yet undetermined neural actions, what does this tell us about the aetiology and treatment of psychological disorders that is new?

If social factors influence the development and/or maintenance of psychological disorders through neural actions (as they must if the materialist philosophy of mind is correct), then it may be useful to examine well-documented mechanisms of CNS plasticity in order to determine whether these mechanisms might also contribute to socially-induced plasticity.

In the last decade one type of receptor which has been implicated in many different types of neural plasticity is the subtype of excitatory amino acid receptor known as the N-methyl-D-aspartate (NMDA) receptor (see Collingridge & Lester, 1989 for a review). This receptor subtype, whose endogenous agonists are the excitatory amino acid transmitters aspartate and glutamate, has been implicated in both adaptive and maladaptive plasticity phenomena ranging from the induction of LTP and epileptiform activity, to recovery from brain damage and the development of Alzheimer's disease (see Collingridge & Lester, 1989 for a review).

Recently it has been suggested that the NMDA receptor may have a function in the development of schizophrenia (Kim, Kornhuber, Schmid-Burgk & Holzmuller, 1980; Nishikawa, Takashimi & Toru, 1983; Carlsson & Carlsson, 1990; Javitt & Zukin, 1990; Wachtel & Turski, 1990; Johnson & Jones, 1990; Javitt & Zukin, 1991). This hypothesis was advanced primarily because the ion channel associated with the NMDA receptor has a binding site for phencyclidine (PCP), which has long attracted interest as a tool for experimental models of schizophrenia (see Johnson & Jones, 1990 for a review). PCP can induce a psychosis in humans which is similar to schizophrenia, and PCP and other NMDA antagonists produce stereo-typed behaviour and hallucinations in animals (see Johnson & Jones, 1990 for a review). PCP produces structural abnormalities in the cingulate cortex which are also found in the brains of schizophrenics (see Wachtel & Turski, 1990 for a review).

If in addition to its other contributions to CNS plasticity, the NMDA receptor contributes to the initiation of neural plasticity associated with social experience, then it is conceivable that the effects of social intervention in schizophrenia and other psychological disorders might be partially mediated by NMDA receptors. Recent evidence in rats suggests that NMDA receptors in the limbic system may mediate synaptic plasticity associated with fear conditioning: other types of plasticity related to social stimuli may also be NMDA receptor-mediated (Miserendino, Sananes, Melia & Davis, 1990).

Conclusions

The aim of this paper has been to present the argument that social intervention exerts its beneficial effects on psychological disorders through CNS mechanisms which are similar in principle to those involved in drug therapy ie. they both modulate synaptic function. Therefore, the separation of social and biological approaches in the treatment of psychological disorders is unnecessary and potentially hinders the understanding of interactions between social and drug effects on the CNS. The argument presented is that on philosophical grounds we should accept the materialist theory that the mind is nothing more than a function of the CNS. It follows that the behavioural plasticity which is associated with social experience is part of the normal function of the CNS. There is abundant evidence from neuroscience to support this type of rapid and continuous plasticity of CNS synapses.

A biological approach to social intervention should lead to an understanding of the neurochemistry of social influences on psychological disorders, this in turn should lead to a better under-standing of the ways that social intervention and drug therapy may interact to the benefit or detriment of the patient (Post, 1992). The predictions from this approach are twofold: firstly, the next decade will see a gradual accumulation of evidence that social factors of all types alter CNS function and specifically modify synapses (Post, 1992); secondly, the NMDA receptor may

have a pivotal function in the neural plasticity which social experiences induce during the development and/or treatment of psychological disorders.

I thank Dr C. Darlington, Dr G. Gillett, Dr Gillian Rhodes and two anonymous referees for their helpful comments on the manuscript. This research was supported by a Laurenson Award from the Otago Medical Research Foundation.

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