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BIOLOGY AND PSYCHIATRY: SOME MISSING PIECES IN THE PUZZLE

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A. Introduction

Since starting my explorations in psychiatry last summer, I have often attempted to find a unifying principle which would reconcile what I was learning about human behaviour, with observations gleaned from a variety of areas and prior experience ranging from systems engineering to my interactions with my own children. My background in computers has given me a mechanistic bias: we should be able to find biological and structural correlates to explain all aspects of behaviour; we are limited in this endeavour by the coarseness of our measuring instruments and by a lack of appropriate theoretical models. For example, no satisfactory explanation exists for how an individual is consciously aware of what he perceives.

F.H.C. Crick (1979) recounted his difficulty in attempting to convince an intelligent woman of this problem. She failed to understand why anyone thought there was a problem, feeling that she probably had somewhere inside her head something like a little television set, until he asked, "So who is looking at it?" Although we may laugh, it is easy enough to slip into this fallacy of the homunculus ourselves. The path to understanding the brain is dotted with such fallacies, ready, like the potholes on Queen Mary, to trap the unwary.

In this exercise, I am trying to pull together various ideas which I feel may bear on the problem of understanding behaviour, but in many ways it's like driving at dusk, trying to find the headlight switch while swerving around potholes. Although I'm probably being overinclusive of concepts at this early stage, I hope that by presenting them in an academic seminar my focus will sharpen.

The presentation is structured as follows: first, a discussion of some of the concepts I've encountered during my eight months in psychiatry, which have been difficult for me to understand or reconcile; second, an examination of some ideas, largely based on animal studies, which need to be fitted into a theory of behaviour; next a presentation of theoretical constructs based on these ideas; and lastly, an exploration of how these constructs can be applied to clarify the contentious concepts and hopefully resolve some contradictions.

B. Puzzles

The psychodynamic approach to psychiatry has generated lots of concepts which are difficult to understand, perhaps because psychoanalytic theories are by their nature hard to prove or disprove, resulting in new formulations being tacked onto earlier ones, with the discarding of something a very rare occurrence.

An example is Freud's concept of the Oedipus complex, a series of generalizations which apparently affect every component of man's behaviour. During the phallic stage, a boy's interest in mother becomes sexually tinged and he has fantasies in which he uses his penis to have intercourse with her. He also becomes concerned with getting rid of father so as to take exclusive possession of mother. However, he then becomes anxious about provoking his father into castrating him if he continues to seek after mother. This "castration anxiety" arises in part from the boy's observation that girls lack a penis, which he fantasizes is due to their having been castrated. In order to avoid losing his penis, he unconsciously decides to abandon his sexual intent towards mother, redirecting it towards other feminine objects, and identifying with father, his rival.

The girl, who lacks a penis, turns towards father with a fantasy that he will impregnate her, the potential child being compensatory in that it is unconsciously equated with the penis. Resolution of the Oedipal dilemma is for her less definite and more gradual than for a boy, so that her superego becomes less well-formed and less stringent. For her, fear of loss of love is a more powerful motivator in this resolution than for the boy, where castration anxiety plays a predominant role.

In the sequence as described by Freud, the identification process for the girl is more complex, as she has to shift from mother as the original love object to father and other heterosexual love objects, whereas the boy does not have to shift gender in moving from mother to other love objects. Moreover, a sense of body inferiority and penis envy would continue to be present in most women.

Since Freud's theories were based on clinical observation, it would seem reasonable that when translated back to their behavioral correlates, many of his tenets would be supported by research based on observation of these correlates, as Fisher and Greenberg (1977) discovered in their massive review of scientific evidence for and against Freud's ideas. However, they did find that some of Freud's conclusions were not supported by the research. For instance, there is no evidence that women experience their bodies as inferior to men's. Moreover, the empirical literature suggests that the female has less, not more, difficulty than the male in evolving a sex role. A major difference in superego severity between male and female was not supported.

For boys, the research indicates that identification with father is stronger and superego severity is more intense, if father is seen as nurturant, rather than a competitor as posited by Freud.

Other concepts put forward by Freud demonstrate the extent to which culture played a role in circumscribing his observations and subsequent conclusions. The "primal scene" and its influence on later sexual behaviours is one example. Although there is a general human preference for privacy during coitus (Katchadourian and Lunde, 1972) in most of the world children sleep in the same room, and frequently in the same bed, as their parents. Freud himself recognized that the latency period was not universal, even in the European society in which he worked; therefore, it was due to a subcultural pattern (Davenport, 1976).

Psychotherapy is frequently a topic of discussion and research. It seems that whenever an individual has had a particular success in helping people in therapy, he has identified the idiosyncracies of his style, technique, or theoretical stance into a new philosophy or school of psychotherapy. If so many different schools continue to flourish, perhaps it's because there is little to choose between them. With respect to psychoanalysis, for example, Jerome Frank (1973) points out the lack of evidence for its therapeutic superiority. He suggests that psychoanalytic theory is all-inclusive and not susceptible to disproof, observing that no training institute has yet disbanded because it concluded that its theory was inferior to that of another school. Other theories of psychotherapy also share the characteristics that they cannot be refuted by the patient's productions.

In particular, the supposition made by Freud that a process whereby the patient gains significant insight, will by itself help the patient change for the better, has not been shown to be true (Fisher and Greenberg, 1977). Is this because it is not possible to "make the unconscious conscious" by an intellectual process? Behaviour therapies show that insight is not necessary to produce lasting change.

Some types of psychopathology is of a type, such as antisocial personality, chronic schizophrenia, or some borderlines, are notoriously resistant to improvement by any form of therapy. Do these represent biological deficits which are beyond the capacity of the brain's plasticity to compensate for?

"Nature versus nurture" issues continue to be the object of much research. Although most would agree that constitutional factors are extremely important in determining the effect that a pathological environment will exert on a particular individual, are there instances in which adverse childhood experiences will ensure mental illness, in spite of inherited strengths? The effect of maternal deprivation resulting in a "detachment" phenomenon as described by Bowlby (1970) immediately comes to mind. But E. James Anthony was able to show that some children, in spite of a horrendous home environment, managed to pull thorugh, apparently untouched by the severe psychopathology which affected their siblings (Anthony, 1981). He reported, however, that these same "survivors" frequently came to psychotherapy as adults, indicating that psychic compensation was incomplete.

I have been troubled, too, by theoretical attempts to explain what motivates behaviour. The concept of two organizing factors, the pleasure principle and the reality principle, the former explaining those instincts from the id demanding direct and immediate gratification, and the latter directing ego instincts which order behaviour necessary to survive, makes sense, as does a "life instinct". But acceptance of a "death instinct" (Freud, 1920) to explain senescence, requires some mental gymnastics.

Before beginning psychiatry, I was quite willing to accept theories of child rearing such as Dr. Spock's, who believed that in the early months at least, an infant was entirely absorbed in physical sensations, ignorant of the boundaries between himself and those who cared for him. But now the emphasis has shifted to the "competent" baby, activated not only by internal pain and appetite but by language and smiles and particular people. Susan Quinn (1982) reports on T. Brazelton's studies of newborns who turn preferentially to the female voice if a man and woman speak simultaneously. G. Carpenter discovered that infants at one month are capable of distinguishing between mother's face and others. In the same article, D. Stern argues that babies need to be "understood" by their parents in order to develop properly. He bases this on videotapes of mother-infant interactions which are microscopically analysed to examine the "fit" between the pair. L. Sander's team at Boston University is engaged in a followup study of 28 adults who were minutely studied as infants 25 years ago, in an attempt to predict outcomes based on infant characteristics.

In all these studies, the assumption is that infants from birth have an extensive behavioural repertoire which may possibly be "pre-programmed"; that is, genetically predetermined.

I will conclude this section with my observations as a father that my young, breast-fed infants, asleep in another room, would frequently wake up shortly after my wife and I engaged in sexual foreplay which included breast stimulation. We experimented by controlling possible stimuli such as noise or light, and came to the conclusion that baby was most likely awakened by smelling its mother's secretions, probably milk.

As McCartney (1968) points out, there is evidence that our sense of smell has degenerated much less than is frequently supposed. He reminds us that Helen Keller's writings indicate the immense value of this sense even for those whose other senses are unimpaired.

C. Puzzle Pieces from Ethology

In this section I describe some findings from animal studies which I feel may help to explain the theoretical inconsistencies outlined in the previous section.

The first of these is "imprinting", a term originally used for the attachment behaviour exhibited by goslings, who will follow its human caretaker rather than its mother if immediately after hatching it is taken into human care, in the famous experiments of Konrad Lorenz (Katchadourian and Lunde, 1972).

Research on certain forms of pseudohermaphroditism provides some evidence for an analogy between the establishment of gender role in early childhood and imprinting phenomena (Katchadourian and Lunde, 1972). Test cases consisted of pairs of hermaphrodites with chromosomal, gonadal, and diagnostic equivalence, but antithetical in sex assignment, biographical history, and gender identity (Money, 1976). The contrast between two such young adults with respect to gender role and gender identity is complete. In general, sex reassignment begun prior to the age of 18 months will be successful, but reassignment between 18 months (the onset of language acquisition) and three to four years bodes less well for psychosexual differentiation. In such a case, the child may never differentiate the appropriate new gender identity so as eventually to fall in love appropriately in it. After four years, forced changes will likely result in psychopathology.

The foregoing suggests not only that early experiences influence later behaviour in a relatively fixed way, but that the early experiences must take place during a specific stage in development; that is, during a "critical period". Another example occurs in puppies studied by Scott and reported on by Bowlby (1970), who took progressively longer to become accustomed to humans, the longer after birth that they were kept isolated from humans. Puppies not exposed until fourteen weeks of age continued to be timid of humans in later life. Similarly, rhesus monkeys isolated from other monkeys from birth for sixteen months are unable to relate to other monkeys for at least two or three years, in experiments by Mason and Sponholz (Bowlby, 1970). The case for critical periods is more solid for sexual behaviour in rhesus monkeys. Harlow found that juveniles who were deprived from peer contact and play during the interval from about three to six months postnatally seemed to be unable to learn to relate sexually and copulate successfully as adults, even if they were reared with normal mothers (Gadpaille, 1980).

In human infants, Bolwby (1970) concluded in a review of studies relating to maternal attachment and deprivation that the period from three to six months is optimal for forming a discriminated attachment; after a year, there are great difficulties which do not diminish. Vulnerability to disruption of an established attachment is especially great for several years after the first birthday.

One very interesting aspect of certain critical period phenomena is typified by the condition known as amblyopia, in which a longlasting impairment of vision occurs due to disturbances of the optics of the eye during early life, even if the defect is later corrected. For kittens reared in the dark, minor physiological changes were found in the retina and lateral geniculate nucleus, but the retina showed pronounced physiological aberrations, as well as anatomic changes such as a 9% overall shrinkage, shorter and fewer dendrites, and a 30% reduction in synapses. Similar experiments with partial visual environments (for example, only vertical

lines and no horizontal lines) appear to support the hypothesis that a degeneration or involution takes place of those neural structures which would ordinarily be employed in sensing that environment which has been restricted (Lund, 1978). Once the critical period is over, reversibility or recovery of responsiveness is minimal except in special cases.

Although this type of neural structure involution has been demonstrated most conclusively for visual and auditory systems which are to a large extent "prewired", i.e. genetically coded for a relatively fixed pattern of development, provided appropriate external stimuli are provided at appropriate critically timed periods, the same mechanisms may operate for stereotyped behaviours which are subject to critical periods.

Human and animal nervous systems are structured hierarchically, from functional, phylogenetic, and developmental points of view. For example, peripherally located ganglia control local activities such as gut peristalsis; the spinal cord contains the mechanisms subsuming coordinated muscular activities such as withdrawal reflexes and (in "spinalized" cats, at least), walking.

The hypothalamus and thalamus, together with lower levels of the nervous system, control stereotyped behaviours necessary for basic existence. For example, cats or dogs whose brains are sectioned above these structures are able to walk, eat and sleep, and would be hard to differentiate from normal animals except for their inability to adapt behaviour; for example, they can show rage during irritant stimulation, but the rage disappears as soon as the stimulus is withdrawn (Lawrence, 1978).

The addition by evolution of higher levels in the hierarchy has not detracted from the functionality of lower levels; the mechanism used by

higher levels to exert control is one of inhibition. That this is so can be appreciated not only from the animal preparations described above, but also from "natural experiments" such as traumatic transection of the spinal cord, strokes affecting the brain, dementia, or metabolic conditions which influence the cerebral cortex, for example alcohol intoxication. In all of these conditions behaviours may be observed which are mediated by lower nervous system levels freed from inhibition by higher levels. It might seem surprising that humans have retained the capacity for behaviours which are usually associated with lower animals (for example, dominance and aggression signals seen in undisguised form in an uninhibited intoxicated man), but unless behaviours which originally evolved in permitting our phylogenetic forebears to survive have become antithetical to survival, they will not be deleted by parsimonious evolution; their expression will simply be inhibited.

D. Fabricating Some Missing Pieces

The previous section has described a number of disparate observations with little to connect them. In this section, I will attempt to fill a few of the gaps with hypotheses, some original, others gleaned from various sources. I will not try to substantiate them at this point.

1. Homo Sapiens comes equipped from birth with an immense repertoire of pre-programmed behaviours. Many of these have been retained in humanity's genetic pool in unchanged form from our evolutionary ancestors, including primates and lower species. If no longer needed for survival, these behaviours lie dormant, inhibited by hierarchically higher neural structures.

2. An important part of evolutionary development involves not only modifying old behavioral patterns or adding new ones which enhance adaptability and therefore survival, but also forming links between neural substrates which control the behaviour and other parts of the brain such as the cerebral cortex and the affective centres for pleasure and pain. The latter is almost mandatory: for example, a mother who derives greater pleasure from breast-feeding than other mothers because of enhanced connections to pleasure centres (which may have arisen by random mutation, chromosomal rearrangement, etc.), will be more likely to breast-feed her children than her sisters; consequently, her genetic endowment will be more likely to become distributed throughout the gene pool (at least before the availability of bottle feeding). Connections to cerebral cortex would arise for behaviours which would be more adaptable if less stereotyped and more available for modification by ongoing experiences. An example is the whole repertoire of social behaviours in animals which live in groups; here, it is useful to be able to recognize rapidly individuals in the group by visual identification of facial features, etc. A male animal who can do this better, because of enhanced connections to

cerebral visual association cortex, will be more successful at inserting his genes for this change into the gene pool if it enables him to exert greater dominance over other males and females and therefore engage in coitus more frequently and with more sexual partners.

However, some behavioural patterns might do quite well without extensive connections to neocortex; possible examples would be those depending on the sense of smell: an infant who becomes aroused and cries when it smells its mother's secretions if hungry will not be more adaptable if this behaviour becomes less stereotyped; similarly for a male's sexual arousal when smelling a female's oestral secretions. Another example could be the submission and fear behaviours demonstrated by social animals with respect to larger, more fierce animals of their own species, behaviours which keep them alive but which would not function better if the aggressor could be rapidly individually identified; it is enough to be able to recognize relative size and cues of aggressive intent.

At the human level, the evolution of capability for complex and highly adaptable communication in the form of speech, along with the ability to plan for speech by manipulation of symbols, that is, consciousness, has led to the development of a large cerebral cortex, the "seat of consciousness". The existence of this large neural structure does not automatically mean that more primitive structures and behaviours will therefore be linked to it; this will occur only if beneficial in an evolutionary sense.

3. Following the above line of reasoning, humans are likely to have a whole repertoire of primitive behaviours, inherited from primal forebears, which may not have cortical connections and therefore no consciousness. Simply being unconscious in this way unfortunately does not guarantee that such behaviour is not widely pervasive and influential; it just means that the individual could not become aware of it directly, but only

through observation of the behavioural correlates in him or herself: not only the motor components, including "body language" such as body habitus, gestures, and facial expression, but also intrapsychic feeling states, whether pleasure, fear, or anxiety including their physiological manifestations in heart rate, flushing, piloerection, sweating, gastrointestinal activity, laughing, and so on. Someone who is particularly psychologically minded might also attend to symbolism and other primary process indicators in his associations, parapraxes, or wit, whether suppressed or verbalized. An extreme example might be behaviours cued by olfaction: it seems that there are hardly any emotionally neutral odours, they all carry various degrees of connotation of pleasantness or unpleasantness (Gloor, 1978). When odours are sufficiently strong we quickly become conscious of these emotions; when less strong, we may only be aware of behavioural correlates such as sexual arousal, without being at all conscious initially that the cue is an odour.

4. For these unconscious behaviours to develop properly in a given individual, suitable environments must exist and interactions occur during the appropriate critical or optimal period for each set of behavioral repertoires. If the pre-programming is not suitably exercised or "primed", all or some of the neural components which mediate the behaviour may involute or degenerate. The amount of damage and its reversibility is determined partly by the extent and duration of deprivation from appropriate interactions with parents, siblings, or peers.

5. The neural components which are subject to involution can include those which mediate:

a) sensing of environmental stimuli or behaviour cues from others;

- b) recognition of stimuli or cues, including the culling of inappropriate cues;
- c) motor components of the behaviour;
- d) pleasure or pain affects associated with the behaviour (necessary for learning to occur);
- memorization or learning connected with performing the behaviour with specific individuals or under specific circumstances;
- f) inhibition of the behaviour by higher levels in the brain.

E. Do The Pieces Fit?

In this final section, I will attempt to apply the hypothetical constructions of the foregoing section to reduce some of the uncertainty highlighted in section B. Firstly, let's re-examine the constellation of behaviours generally ascribed to the Oedipus Complex, to see if they can be explained by preprogrammed behavioral repertoires inherited from lower animals in our phylogenetic chain, behaviours which may not be directly accessible to consciousness.

For example, aggression and fear of aggression is a common part of life for some sub-human primates, especially baboons. The "alpha" male maintains order and discipline among females, juveniles and subordinates by threats and punishments such as bites on the neck, which are apparently very effective in eliciting fear in underlings so that they keep their place (Morgan, 1972). In such a society, the more aggressive the male, the more likely he is to mate frequently and thus propagate his aggressive gene. Similarly, a male who can rapidly recognize a threat from a larger, more powerful male and show the appropriate submission behaviour is more likely to avoid all-out confrontation which would result in being expelled from the band, resulting in little possibility of mating, let along surviving. Thus, at least among male primates, behaviours associated with aggression and with fear of aggression are adaptive and evolutionarily sound. Although there would appear to be little need for such behaviours in the female of the species, studies suggest that sexual dimorphism is never absolute; there is a coexistence of both male and female systems in mammalian brains of both sexes (Gaidpaille, 1980).

If human infants are born with such behaviours already pre-programmed, oral rage would appear equally in both males and females prior to identity differentiation; later on, male children would demonstrate behaviours corresponding to aggression directed at dominant males, particularly the one with whom he has most frequent contact, whether it be father, or his mother's brother in the case of the matrilineal Trobrianders studied by Malinowski (Parsons, 1964). Conversely, he would fear aggression from these same dominant objects. To a lesser extent, perhaps, a girl's male part-brain would react with similar aggression and fear when triggered by such "masculine" behaviours in others, whether male or female. One might hypothesize that if such behaviours are pronounced in a girl, there has been a failure of normal development during what may be an "optimal period" corresponding to the Oedipal period, when research indicates that each sex identifies more with the same than the opposite-sex parent (Fisher and Greenberg, 1977). The normal line of development might involve a partial involution of the neural substrates involved in aggressive behaviour, unless involution is prevented by the exercising of these pathways by inappropriate aggressive behaviours (or parental behaviour which indicates fear of the child's aggression) during the optimal period.

In later life, a male might demonstrate both submission and aggression behaviours in relation to more dominant individuals (supervisors, professors, or therapists) which could be interpreted as inappropriate transferences based on unresolved Oedipal conflicts with father; they might also be considered, more parsimoniously, as examples of genetically determined behaviour. Studies which demonstrate that adult males have a greater degree than females of "castration anxiety", (concern about sustaining body damage) particularly in response to sexual stimuli (Fisher and Greenberg, 1977), can be explained by sexual repression (e.g. parental aggressive responses to infantile self-stimulation) which are more likely to evoke fear in males compared to females. This view is supported by Stephens' cross-cultural studies in which he found a strongly significant correlation between extensiveness of menstrual taboos (which he equated with castration anxiety) and severity of punishment for masturbation, and also with a society's intensity of sex anxiety (Stephens, 1962).

Predominantly female behaviours observed in lower animals should also have been transmitted to humans; for example, nurturing behaviour, which is motivated not only by pleasure, as in suckling, but also by anxiety related to separation from the dependent infant. This same anxiety may be responsible for the research findings that women are much more concerned than men about loss of love (Fisher and Greenberg, 1977). These authors have difficulty explaining studies of penis envy in women using the Oedipal conflict model; highly masculine women did not produce more themes of penis envy than low masculine females; women who pursue masculine goals demonstrate on the Rorschach more confusion with regard to sex identity, and more phallic symbolism. Such results are easily explained using the hypotheses previously described.

I won't belabour this point any longer, other than to suggest that there are likely other research findings related to Freud's theories which are more adequately explained by such a biologically based theory.

Is it possible to apply these hypotheses to explain psychopathology? We can take the various neural components which are subject to involution and consider what might occur if some degeneration did take place as a result of faulty interaction or environment during optimal periods. It may be entirely too simplistic to state that psychotic hallucinatory phenomena reflect difficulty in sensing and recognition of environmental stimuli and cues; that catatonic states are symptomatic of motor problems; that the flatness of affect of schizophrenia, or the borderline's wrist slashing so as to feel pain and "be real" is related to inadequate functioning of pain and pleasure correlates of behaviour; or that the reappearance of infantile reflexes and "second childhood" phenomena in dementia reflect loss of inhibition of primitive behaviours by higher levels.

An important question is, how permanent are such changes? Does the "amblyopia" model indicate a poor prognosis? Character disorders, especially psychopathy, drug addictions, and chronic schizophrenia are difficult to "cure" by any therapy. If these are caused to some extent by infantile or childhood exposure to inappropriate stimuli during critical or optimal developmental periods, an examination of the length and severity of such experiences should give prognostic information.

A final point relates to whether behaviours can be made conscious. In line with the above model, there are a class of behaviours which are barred from direct access to conscousness, and possibly also direct control by consciousness, except in an "over-riding" sense. Therapies which attempt to "make the unconscious conscious" are likely to be no more effective with such behaviours than directive, behavioural, or other forms of therapy. However, the indirect mechanisms which one can use to become aware of unconscious behaviour, which are employed by the analyticallyoriented therapist in understanding his or her patient, could also be applied by the patient himself. Thus, the content of an interpretation would take second place to conveying an understanding of how the interpretation was arrived at. The patient's appreciation of process may be as important as the therapist's.

F. Conclusion

The next step in developing this model would be to frame hypotheses in a way amenable to testing. One area would be to seek commonalities in behavioural repertoires observed in interactions between parent and child and between adults, perhaps employing videotape. Another is too apply some of the newer technologies, e.g. computer counting of synapses from micrographs of brain tissue, to autopsy specimens segregated as to psychopathology according to the present model. Yet a third area involves studies to examine the extent to which olfaction affects behaviour in humans.

Whatever the outcome, I believe that the path to greater knowledge of human behaviour lies with biological approaches, with a "metapsychology" based on today's science.

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