Going in through the out door: On the topic of Bateson's double bind theory

Denise M. Frank*

Abstract

A long time ago, Bateson and colleagues developed the double bind theory. As a theory of schizophrenia, it did not fare well. Research in this area has been forgotten, or better said, ignored, in academic circles and so I decided it was time to re-evaluate the theory and attempt to understand why it was so unsuccessful. This paper focuses on Bateson's understanding of the double bind theory of schizophrenia, and explores and exposes why the theory itself, was not the problem.

Key Words: *Schizophrenia, Bateson, double bind theory, communication, critical theory*

Bateson's theory of schizophrenia: the double bind

The double bind, first described by Bateson and his colleagues (Bateson, Jackson, Haley and Weakland, 1956), is a theory of schizophrenia based on the premise that poor communication styles within a family unit contribute to the development of the disorder. This simple statement 'It is hypothesized that a person caught in the double bind may develop schizophrenic symptoms' (Bateson et al., (1956, p. 1) sent the mental health community into a theoretical tizzy.

It was clear that even though Bateson focused his efforts on understanding how communicative patterns within a familial setting can affect those with a certain disposition towards the disorder, Bateson believed that the actual experience of being in a double bind, was the same for those with and without schizophrenia. This may lend a clue as to one of the reasons the theory failed to survive. Bateson stated that 'Many writers have treated schizophrenia in terms of the most extreme contrast with any other form of human thinking and behaviour. While it is an isolable phenomenon, so much emphasis on the differences from the normal – rather like the fearful physical segregation of psychotics – does not help in understanding the problems. In our approach, we assume that schizophrenia involves general principles which are important in all communication and therefore many informative similarities can be found in 'normal' communicatiol[1] (**spelling error theirs**) situations' (Bateson et al., 1956, pgs. 13–14). Per Bateson, 'We have suggested that this is the sort of situation which occurs between the pre-schizophrenic and his mother, but it also occurs in normal relationships. When a person is caught in a double bind situation he will respond defensively in a manner similar to the schizophrenic. An individual will take a metaphorical statement literally when he is in a situation where he

must respond, where he is faced with contradictory messages, and when he is unable to comment on the contradictions' (Bateson et al., 1956, p. 5).

Before we can discuss Bateson's application of the double bind to schizophrenia and the role of the family in its development, let us look at the conditions that need to exist for a double bind to occur as provided by Bateson et al. (1956), pp. 3–4:

1. There must be two or more persons involved. One of them must be the individual with schizophrenia and that person must have a relationship in which one, or possibly more members of the family, create a double bind as a function of their communicative style. Thus, the individual eventually succumbs to inner turmoil.

2. The double bind is repeated over time, creating in the individual an expectancy for this type of communication.

3. The double bind communication is initially expressed as a primary negative injunction, taking one 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' (Bateson et al., 1956). In other words: (a) Do not go to the store, or I will punish you (i.e. if you go to the store I will punish you), or (b) If you do not go to the store, I will punish you.

4. A conflicting secondary injunction follows the first; however, it contains an element of punishment as expressed through the giver's posture, tone of voice or gestures.

5. A third or tertiary negative injunction prevents the individual from avoiding/escaping the situation.

6. After conditions 1–5 have repeatedly occurred over time, the individual has learned to anticipate these types of messages in all forms of communication.

Bateson states that 'almost any part of the double bind sequence may then be sufficient to precipitate rage or panic. The pattern of conflicting injunctions may even be taken over by hallucinatory voices' (Bateson et al., 1956, p. 4).

The double bind is based on a specific communicative style in which two or more messages are presented that contain conflicting information. As one message contradicts the other, confusion mounts over time for the receiver, resulting in long term emotional distress. If the receiver can successfully respond to one of the messages, but not to the other, and is unable to either challenge or resolve what is said to them, the receiver's response will be incorrect. Keep in mind that Bateson's construction of the double bind

relies on the infant's exposure, internalisation and inability to confront or escape a situation. When the infant cannot escape, schizophrenia may later develop. Bateson argued that if confusing messages originating during infancy between mother and child remain psychically embedded throughout the child's adult life, the inability to differentiate those messages into meaningful and logical units may result. In some individuals, illogical thinking, incoherent speech and a delusional, perhaps hallucinatory belief system are the only means of escape (Bateson et al., 1956). According to Bateson et al. (1956), 'The psychosis seems, in part, a way of dealing with double bind situations to overcome their inhibiting and controlling effect. The psychotic patient may make astute, pithy, often metaphorical remarks that revea1 (**spelling error theirs**) an insight into the forces binding him. Contrariwise, he may become rather expert In (**spelling error theirs**) setting double bind situations himself' (p. 13). From this position, the process through which schizophrenia is developed during infancy is clear and we may be drawn into thinking that double bind situations can only occur in those with family members who display difficulties in communicating with one another. Unfortunately, the double bind finds its way into situations where one would hope would be the last place for it to occur – within the therapeutic setting.

Our daily dose of the double bind

One day while conducting the research phase of my dissertation at a major psychiatric hospital in Long Island, New York, my supervisor and I met one of the staff psychiatrists in the hall; a much older gentleman with a very distinct way of thinking about his patients. Upon hearing that my study was challenging the research that suggested that individuals with schizophrenia did not have self-esteem, he responded with a cynical laugh 'Self-esteem?' 'Of course they have no self-esteem –they're schizophrenic!' He looked at my supervisor and shook his head. 'She's wasting her time.' And walked away.

In this case, it was highly likely that individuals with schizophrenia who entered a therapeutic relationship with this psychiatrist were unknowingly entering into a double bind. The situation would meet the criteria of the double bind as represented by the following scenario:

1. The psychiatrist and his patient were involved in discourse that was confusing to the patient (i.e. 'you can't have self-esteem if you're schizophrenic') causing the patient to experience inner turmoil. Therapy would not be aimed at either developing or increasing the patient's self-esteem.

2. Therapy sessions were repeated over time.

3. The primary negative injunction was 'you are here to feel better about yourself but you can never feel better about yourself because you are schizophrenic'.

4. A conflicting secondary injunction followed the first in the form of whatever the tone of voice, gesture or posture the psychiatrist displayed.

5. A third or tertiary negative injunction prevented the individual from avoiding/escaping the situation as chances are the patient felt stuck with the psychiatrist.

6. The patient had learned to anticipate those types of messages in all forms of communication, leading him to experience hallucinations as a form of escape (see Bateson et al., 1956, p. 4) or at best, isolation.

No matter how positively the patient felt about his 'self', he would not have been able to convince the psychiatrist otherwise. In this case, not only does the psychiatrist develop an environment for the double bind to occur, but in doing so, manages to maintain the patient within an illness model.

Outside of therapy, we find ourselves in different types of double binds. When we modify the criteria for the double bind to apply it to our daily lives, miscommunication remains the foundation in creating a no-win situation. For example, the picture (Figure 1) reads 'Do Not Enter. Enter Only'. Do I go through the door or not? If I try to go through the door, then security will tell me 'do not go through the door'. But if I ask security 'what door can I leave through?' they will tell me 'go through that door'. We all wind up in a double bind at some point or

other during our lives but can filter information and place things in their proper context. For those individuals whose current understanding of the world is filled with confusion, misdirection, and the inability to communicate effectively, a simple encounter like the one above, has the potential to create an enormous amount of stress. Individuals who are unable to develop a strong psychological constitution to withstand the rigours of conflict and disruption within the familial environment, or perhaps are disposed to developmental or cognitive impairment, continued confrontations with conflicting messages may be enough, as Bateson hypothesizes, to either initiate a psychotic episode or even maintain a psychotic state.

Why the double bind theory is not the problem

Many times, theories fail to provide a satisfactory explanation of a phenomenon, or perhaps the theory could not withstand the rigours of scientific scrutiny. Whatever the case, theories are proposed to understand life's unexplainable occurrences. But strangely, sometimes brilliant minds wind up committing a disservice to a theory that might otherwise have had a huge impact on humanity. In our field, we are aware of the many ways to conduct research. Whatever the approach, it will fall under one of two categories – qualitative or quantitative. In the most general sense, each of these methodological approaches compliments the other. Unfortunately, it is no secret that many quantitatively grounded researchers believe that empiricism is the *only* method to unveiling and thus revealing the truth. The scientific method may not have been the best choice when analysing the double bind within the conceptual framework of behavioural genetics (Gottesman, 1991) as the significance and applicability of the theory was misunderstood and thus, severely questioned. In other areas such as family process (Lidz and Fleck, 1985) and theories of expressed emotion (Brown, Birley and Wing, 1972; Kavanaugh, 1992; Miklowitz, 1994) the double bind also came under fire.

When the key concept of the double bind reflects the development of schizophrenia through the expression of contradictory forms of communication between family members (Burston, 1998), it may have been wise to choose a framework of inquiry that worked in tandem with the core principles of the theory. Family dynamics, as they were experienced by those bound up in the bind, could have been explored through research methods that more closely resembled those undertaken by Bateson (i.e. phenomenological type methods). Nowhere in Bateson's theory of schizophrenia is the clinical data suggestive of research conducted experimentally or quantitatively. Bateson's work was based only on 'observations and descriptions' (Bateson, 1972, p. 153; Bateson et al., 1956, pgs. 10–13). It was from these very descriptions, that Bateson theorized the development of schizophrenic symptoms. For example, Bateson states, 'We are recording interviews held jointly with patients and their families, and we are taking sound motion pictures of mothers and disturbed, presumably pre-schizophrenic, children' (Bateson et al., 1956, p. 15). From this perspective, the double bind was subjected to a method of testing that was clearly outside of a framework consistent with the theory. Ironically, the phenomenon of the double bind was experienced by the researchers whose inability to correctly communicate the true nature of the theory amongst themselves, led to incorrectly selecting an appropriate research design.

As a result of analysing the double bind with unsuitable methodologies, it was concluded that the theory was too unspecific to generate a testable/researchable hypothesis. According to Schuham, 'Little theoretical agreement exists about the elements required to generate double bind situations or the relevant interactional parameters of double bind communication. The research literature fails to support the assumptions and predictions of the theory. The tenets of the double bind hypothesis require further limitation, clarification, and operationalisation for it to become a reliable phenomenon capable of empirical validation' (1967: 409–416). Although Abeles suggested that the double bind was simply 'unresearchable' (Abeles, 1976, p. 113), later attempts yielded more supportive results in its favour (Blotchky, Tittler and Friedman, 1982).

The literature also demonstrates much confusion with respect to identifying if the double

bind is a theory or a hypothesis (Abeles, 1976; Olson, 1972). This would certainly contribute to research design issues, possibly yielding conflicting or even untenable results. From this lens, it would not be at all unreasonable to suggest that since research into the double bind was concluded to be empirically unsupported (Abeles, 1976; Olson, 1972) there was no reason to further continue its investigation. So why wasn't the double bind explored via other methods? Bateson et al. never stated that the double bind was definitively the way schizophrenia developed, but rather they hypothesized that '...a person caught in the double bind *may* (**italics mine**) develop schizophrenic symptoms' (see Bateson et al., 1956, p. 1) implying that the double bind was only one of many interpretations. For some reason, no one paid much mind to this enlightening little bit of information.

Critical theory and the double bind

Because Bateson clearly disrobed the taboo surrounding poor parenting and the development of schizophrenia, no one wanted to look. It is of value to discuss the double bind within the framework of critical theory. Critical theory functions under two assumptions: (1) historical ontology in the sense that the nature of our existence is moulded by various positions (i.e. social, political, cultural, and economic, etc.) and that these positions are understood and being real (Cohen and Crabtree, 2006) and, (2) that a researcher and the object of his/her attention cannot be separated. This is known as modified transactional or subjectivist epistemology (Cohen and Crabtree, 2006). Critical theorists are in opposition to historical ontology in the sense that what is moulded or developed by a society or through political agenda is in direct conflict to the principle of subjective realism.

By applying critical theory to the assumptions of the double bind, incorrect research methodologies can be identified to eliminate guessing the disorder's occurrence, thereby helping to address its true nature as experienced first-hand by those caught up in communicative chaos. When the double bind is understood from this perspective, we might assume that another reason for its disappearance is that Bateson shifted the focus of the disorder from biological/genetic interests onto the family unit, unleashing the taboo surrounding parental accountability for behaviours that resembled individuals with schizophrenia. For example, we could contemplate that negative symptoms (e.g. isolation) might result from a stress induced response created during extended confusing and emotionally charged communicative patterns. It would stand to reason that because communication is such a powerful tool, and because different types of communicative approaches have the power to elicit various emotional and behavioural responses, the individual who can control the discourse better gains control, leaving the weaker individual exposed to verbal attacks. As Bateson stated, 'If the double binds are imposed during infancy, escape is naturally impossible' (in Collier, 1977 p. 111) suggesting that parental interactions (usually mother–infant) are responsible for the disorder's development. Through a critical theory approach, the task of the research community would have been in identifying parents who admitted that their own poor communication skills had contributed to their child developing schizophrenia.

Fixed thinking, schizophrenia and the double bind

The application of critical theory to the double bind elucidates the need to review other areas socially constructed by the mental health profession, such as the manual used by practitioners to help diagnose and label individuals, for example, the Diagnostic and Statistical Manual (DSM) (American Psychiatric Association, 2013), which is in itself problematic; one issue being that each individual interprets their symptoms from such a personal experiential position, that the disorder, regardless of similarity of description between individuals, becomes a separate disorder unto itself. Having to rely on diagnostic categories that may have been constructed as a function of the mental health community's social, political, cultural, and economic agenda is of dire concern and raises questions concerning its validity and credibility as a diagnostic tool. If the goal of critical theory is to transform negative social views, then questioning the underlying intent of the psychiatric community becomes obligatory. By questioning the assumptions that have guided the psychiatric community, critical theory challenges the fixed categories that diagnoses rely on, as well as their

effectiveness. Boyle (2007), who has extensively questioned the validity of the DSM as well the validity of empirical investigation has stated '... our body parts, after all, don't have language or emotions, form beliefs, make relationships, create symbols, search for meaning, or plan the future. Small wonder that a theoretical framework developed for understanding bodily problems, has proved so inappropriate for the task of understanding psychological experience and behaviour' (p. 290).

When schizophrenia is understood within the conceptual framework of the patient's experiences and what each of those experiences represents for him, it might not actually be schizophrenia, but simply a personal way of understanding the world, especially when taking into consideration the cultural context in which the behaviour occurs. Boyle states that 'Yet there is strong evidence that emotional distress and behavioural problems, even the most bizarre, are understandable responses to or ways of actively trying to manage adverse circumstances and relationships' (2007, p.290). Boyle's message is simple and quite clear.

The literature is replete with experiments that suggest that schizophrenia is a brain disorder and that various areas of the brain are affected. For example, research has focused on structural abnormalities (Narr et al. 2005; Spinks et al. 2005; Joyala et al. 2003), neurological abnormalities (McCreadie et al. 1996; Hoa, Mola, and Andreasen, 2004), neuropsychological abnormalities (Brickman et al. 2004), neurophysiological abnormalities (Meltzer and McGurk, 1999) and cerebral metabolic abnormalities (Lehrer et al. 2005). Biological research demonstrates that there are no definitive studies that conclusively suggest that schizophrenia is, without a doubt, located solely in one agreed upon area of the brain (Zipursky, Reilly, and Murray, 2012; Chaua and McKenna, 1995; Siebert, 1999. Given that there are presently many hypotheses regarding the aetiology of schizophrenia, the notion that schizophrenia may be a disorder that takes its developmental cues from 'somewhere else' is not far-fetched, giving us licence to explore other venues such as the role that communicative patterns between children and their parents play in its development. One area that we need to re-explore is Bateson's original premise and to first determine if, and how, poor communication and expression during language and communication development affect the ability of infants to later understand their world. If the infant experiences greater sensitivity to its immediate surroundings where messages are not what they seem, and in which family members never recognize others' personal perspectives as being correct, perhaps further research might see this as having, in part, contributed to the expression of negative symptoms. The only recourse the individual would have is to develop its own language; one free of all imposing familial verbal confusion and retreat into a world where double binds can be avoided and uncertainties do not exist.

Perhaps in placing the brain idea aside, and in opening our consciousness to alternative views, we may find a new beginning to answer an old question but in doing so, we disturb an entire intellectually stubborn community. S

Denise M. Frank PhD is a theoretical psychologist currently investigating the application of various theoretical models such as terror management and self-presentation, as they apply to the concept of self-esteem in individuals with psychotic disorder. She is currently a Research Fellow at the Institute for Social Innovation at Fielding Graduate University, Santa Barbara, California. Funding for this research was provided to Denise M. Frank from the Blackner Stone Family Fund, Palm Beach, Florida.
***dmfrank@hotmail.com**

Dr.Frank would like to dedicate this paper to Mr. Richard Stone for his generous and unwavering support in her research endeavours.

References

Abeles, G. (1976) Researching the unresearchable: Experimentation on the double bind. In C. E. Sluzki & D. C. Ransom (Eds.). *Double bind: The foundation of the communicational approach to the family.* New York: Grune & Stratton.

American Psychiatric Association. (2013) *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.

Bateson, G. (1972) *Steps to an ecology of mind. Collected essays in anthropology, psychiatry, evolution and epistemology.* Chandler Publishing Company. Retrieved from monoskop.org/images/b/bf/Bateson_Gregory_Steps_to_an_Ecology_of_Mind.pdf

Bateson, G., Jackson, D.D., Haley, J. & Weakland, J. (1956) Towards a theory of schizophrenia. *Behavioral Science, 1,* 251–264.

Blotchky, A.D., Tittler, B.I. & Friedman, S. (1982) The double bind situation in families of disturbed families. *Journal of Genetic Psychology, 141*, 129–142. DOI:10.1080/00221325.1982.10533463

Boyle, M. (2007) The problem with diagnosis. *The Psychologist* . May, 20, 290–295.

Brickman, A.M., Buchsbaum, M.S., Bloom, R., Bokhoven, P., Reshmi, P-O, Mehmet, H.M., Dahlman, K.L., Hazlett, E.A., Aronowitz, J., Heath, D., & Shihabuddin, L. (2004) Neuropsychological functioning in first-break, never-medicated adolescents with psychosis, *Journal of Nervous & Mental Disease*; *192*(9), 615–622.

Brown, G.W., Birley, J.L.T. & Wing, J. K. (1972) Influence of family life on the course of schizophrenic disorders: A replication. *British Journal of Psychiatry, 121*, 241–258.

Burston, D. (1998). *The wing of madness: The life and work of R. D. Laing.* Harvard University Press.

Chaua, S.E., & McKenna, P.J. (199 Schizophrenia – a brain disease? A critical review of structural and functional cerebral abnormality in the disorder. *British Journal of Psychiatry, 166*, 563–582.

Cohen, D. & Crabtree, B. (2006) 'Qualitative Research Guidelines Project.' July 2006. hqualres.org/HomeCrit-3518.html.

Collier, A. (1977) *R.D. Laing: The philosophy and politics of psychotherapy.* London: The Harvester Press Limited.

Gottesman, I. (1991) *Schizophrenia genesis: The origins of madness.* New York: Freeman.

Joyala, C.C., Laaksoc, M.P., Tiihonenb, J., Syvälahtig, E., Vilkmang, H., Laaksog, A., Alakareh, B., Räkköläineni, V., Salokangasj, R.K.R. & Hietalaj, J. (2003) The amygdala and schizophrenia: a volumetric magnetic resonance imaging study in first-episode, neurolepticnaive patients. *Biological Psychiatry, 54*:11, (1), 1302–1304.

Kavanagh, D.J. (1992) Recent developments in expressed emotion and schizophrenia. *British Journal of Psychiatry, 160* (5), 601–620.

Lehrer, D.S., Christian, B.T., Mantil, J., Murray, A.C., Buchsbaum, B.R., Oakes, T.R., Byne, W., Kemether, E.M. & Buchsbaum, M.S. (2005) Thalamic and prefrontal FDG uptake in never medicated patients with schizophrenia. *American Journal of Psychiatry, 162*(5), 931–938. dx.doi.org/10.1176/appi.ajp.162.5.931

Lidz, T. & Fleck, S. (1985) *Schizophrenia and the family.* Second Edition. New York: International Universities.

McCreadie, R.G., Thara, R., Kamath, S., Padmavathy, R., Latha, S., Mathrubootham, N., & Menon, M.S. (1996) Abnormal movements in never-medicated Indian patients with schizophrenia. *British Journal of Psychiatry, 168* (2), 221–226.

Meltzer, H.Y & McGurk, S.R. (1999) Psychopharmacological approaches: the effects of clozapine, risperidone, and olanzapine on cognitive function in schizophrenia. *Schizophrenia Bulletin, 25*(2), 233–256.

Miklowitz, D.J. (1994) Family risk indicators in schizophrenia. *Schizophrenia Bulletin, 20,* 137–149.

Narr, K.L., Toga, A.W., Szeszko, P., Thompson, P.M., Woods, R.P., Robinson, D., Sevy, S. Wang, Y.P., Schrock, K. & Bilder, R.M. (2005) Cortical thinning in cingulate and occipital cortices in first episode schizophrenia. *Biological Psychiatry, 1*, 58:1, 32–40.

Olson, D.H. (1972) Empirically unbinding the double bind: A review of research and conceptual formulations. F*amily Process, 11*, 69–94.

Schuham, A. I. (1967) The double bind hypothesis a decade later. *Psychological Bulletin, 68*(6), 409–416.

Siebert, A. (1999) Brain disease hypothesis for schizophrenia disconfirmed by all evidence. *Journal of Ethical Human Sciences and Services, 1*, 179–182.

Spinks, R., Nopoulosa, P., Ward, J., Fullerd, R., Magnotta, V. A. & Andreasen, N.C. (2005) Globus pallidus volume is related to symptom severity in neuroleptic naive patients with schizophrenia. *Schizophrenia Research, 73*:2–3, 1, 229–233.

Zipursky, R.B., Reilly, T.J., & Murra, R.M. (2012) The myth of schizophrenia as a progressive brain disease. *Schizophrenia Bulletin 39*(6), 1363–1372