Chapter 15
Childhood Antecedents of Personality Disorders

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Abstract Early life influences have for long been suspected to play a crucial role in shaping adult life. A significant body of literature now supports the effect of early life influences on adult medical conditions such as obesity and heart disease. In psychiatry too, evidence now suggests a similar phenomenon. This is especially true for personality disorders which are now seen as a continuity of the patterns of tendency to feel and behave observed in childhood. A number of factors ranging from psychological to socio-economic and biological have been postulated as causal factors, mediating factors or simply associated factors. The present chapter attempts to discuss some of the basic theoretical foundations, the commonly cited factors involved, and the utility and implications for clinical practice.

Keywords Childhood antecedents · Personality disorders · Conduct disorder · Attention deficit hyperactivity disorder · Life span approach

Abbreviations

ADHD	Attention deficit hyperactivity disorder
APA	American Psychiatric Association
COMT	Catechol-O-methyl transferase
DOHaD	Developmental origins of health and disease
MAO A	Monoamine oxidase
WHO	World Health Organization

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15.1 Introduction

Early life influences and its effects have always been an area of great interest in psychiatry. The classical psychoanalysts referred to the formative years of life as having great significance in the development of personality and causation of a range of psychiatric disorders. From a more biological standpoint too, it appeals to reason that minor 'defects' produced in early life or prenatally, can produce potentially devastating consequences in later life. Over the years though, the emphasis seems to have shifted away from this line of thinking for a variety of reasons. One potential reason could be the way the diagnostic criteria for personality disorders are structured in the current classificatory systems (World Health Organization 1992). The current classificatory systems seem to give an impression of personality being relevant or assessable only after middle to late adolescence (Salekin 2016). This could have potentially hindered research in this area. However, in the recent years there is an acknowledgement of a lifespan approach to personality pathology, best exemplified by many of the structural changes proposed in the appendix of DSM-5 for further study (American Psychiatric Association 2013).

Traditionally, personality disorders were discussed as categorical entities; and were broadly categorized into three 'clusters': Cluster A or the 'eccentric' cluster, cluster 'B' or the 'dramatic' cluster and cluster 'C' or the 'fearful' cluster. Categorical arrangements do seem to have some validity. While cluster analytic studies failed to replicate this three cluster model; many environmental factors influencing personality clusters fit into a three factor solution roughly coinciding with the clusters A, B, and C described above (Mervielde et al. 2005).

However, issues related to comorbidity; overlap between personality disorders and normal personality as well as between personality disorders that led to a plethora of not otherwise specified diagnosis; and the similarities between structure of personality and personality disorders led to the consideration of dimensional conceptualization of personality disorders. In the debate on whether psychiatric disorders are categorical or dimensional, the broader consensus is that both apply to differing extent and also depending upon the disorder in question. However, personality disorders as a group is more often considered as dimensional rather than categorical (Zimmerman and Spitzer 2009). In view of the limitations in the diagnostic criteria in current classificatory systems, several researchers have attempted to study childhood presentations using various modifications of the existing criteria. However, the validity of such modified criteria has often been questioned. Among the other ways of assessment, the one that has the highest empirical support is the five-factor model (Mervielde et al. 2005). The five factors are extraversion, conscientiousness, agreeableness, neuroticism, and openness. Minor variations of this model have been widely used. The terms personality, temperament, and character have been used somewhat interchangeably by many but are conceptually somewhat different (Cloninger and Svrakic 2009). Allport (1963) defined personality as the 'dynamic organization of one's psychophysical symptoms determining his/her adjustment to the environment.' Temperament refers to 'body's innate biases in conditioned behavioral response to stimuli'

whereas character refers to 'what one voluntarily makes of oneself.' Cloninger has proposed four types of temperament—novelty seeking, reward dependence, persistence and harm avoidance and three dimensions of character—self directed, cooperative and self-transcendent (Cloninger and Svrakic 2009).

15.2 Conceptual Framework for the Role of Antecedents

15.2.1 The Developmental Origins of Health and Disease (DOHaD) Hypothesis

Developmental Origins of Health and Disease (DOHaD) hypothesis is an interesting hypothesis and has gained much ground in recent years (Kubota et al. 2015). In simple terms, it refers to the observation that less than optimal environmental condition during early life may permanently change the structure and function of various body parts, such that, the individual gets predisposed to various chronic conditions in later life. Such an alteration is believed to occur via various mechanisms, for instance, alteration of feedback mechanisms and altered maladaptive stress responses. From a homeostatic standpoint, such a reaction serves to salvage the organism in its early life, which may have evolutionary significance. However, such short term gains occur at the cost of long term losses. The multi-factorial causation of various medical conditions such as cardiovascular disorders and diabetes are believed to be in keeping with such a hypothesis (Moore 2016). Among psychiatric disorders, schizophrenia has been the best studied disorder within the DOHaD framework. Meta-analytic studies (Cannon et al. 2002) have verified that indicators of suboptimal early life environment such as low birth weight and smaller head circumference predict risk of schizophrenia. A number of factors have been proposed as possible explanations, including altered complement activity and excessive oxidative and nitrosative stress (Mhillaj et al. 2015). Personality disorders being chronic conditions of adult life automatically attract research attention in this regard. Indeed, there is now a considerable body of evidence supporting such a view, though, as has been previously mentioned, some inconsistencies exist. Some of these inconsistencies could be attributed to the potentially nonlinear relationships between markers of early life adversity and adult conditions. The classic example of such a phenomenon is birth weight. While low birth weight predisposes to various adult disorders, weight beyond 4 kg also increases risk for such disorders.

15.3 Childhood Antecedents of Personality Disorders

A number of antecedents have been described in relation to personality disorders; some of them specific to certain clusters or disorders, while others posing a general risk to personality disorders as a group while still others increasing risk

for several psychiatric disorders. Some of the better researched antecedents are described below.

15.3.1 Parity and Maternal Age

Children born to mothers in their teenage or third or more in the birth order have been reported to demonstrate an increased evidence of personality disorders (Fazel et al. 2012). Other studies have demonstrated that this increased risk may be especially for the cluster B (dramatic cluster) of personality disorders (Kuja-Halkola et al. 2012; Mitrou et al. 2010). But as in case of previously discussed antecedents, this finding too is not without inconsistency. Some longitudinal studies have found no effect of teenage births on future risk of personality disorders (Kantojärvi et al. 2008). Similarly, parity beyond the sixth pregnancy did not increase risk of personality disorders but did so for other psychiatric disorders (Kemppainen et al. 2000). This again highlights the nonlinear nature of risk factor-outcome relationship.

15.3.2 Growth in Early Life

Pre- and post-natal growth (growth in early life) is another important antecedent that is often linked to personality disorders (Samuel and Widiger 2008). Studies have looked into several different temperamental traits in relation to this characteristic. Negative affectivity and harm avoidance have been found in several studies to be linked to the measures of poor early growth such as 'thinness' (Pesonen et al. 2006) and lower birth weight, length, and head circumference (Pesonen et al. 2009). However, other studies have demonstrated that very low birth weight babies went on to develop higher levels of conscientiousness, agreeableness (Pesonen et al. 2008) and behavioral inhibition (Pyhälä et al. 2009) and lower levels of openness to experience. There is some evidence to suggest that sex may have a modifying role to play but evidence on the exact nature of this is conflicting and inconclusive (Moilanen et al. 2010; Lahti et al. 2008).

15.3.3 Temperament

Temperament refers to the organism's innate bias in responding to the external stimuli. They include its spontaneous and characteristic ways of reacting to stimuli such as danger, novelty, challenges and reward (Basaglu et al. 2011). Temperamental traits are generally believed to have a strong genetic component

and heritability and also to be precursors of adult personality. A number of studies including several longitudinal studies have shown a 'continuity of pattern' in behavior from early childhood to adulthood. For instance, measures such as irritability, attention span, lability and negativism measured from as early as 6 months of age to five years predicted borderline personality symptoms at age twelve and beyond (Belsky et al. 2012). Similarly, poor behavioral control at 3 years of age predicted antisocial personality at 21 years of age (Caspi et al. 1996). Along similar lines, low inhibition and fearfulness at 3 years of age also predicted psychopathic behavior in adulthood in another study. Persistent overestimation of self confidence after age 10 is a predictor of narcissistic personality. In the same vein, persistence of illogical thinking and loose associations beyond age 7, poor attention and executive functioning, and, disorganized verbal responses predict schizotypal disorder-related communication impairments in adulthood. Reduced involvement in extracurricular activities and less popularity in school reflects temperamental inclination towards adult avoidant personality. On the contrary, several studies have shown that many temperamental features are not stable and may change over an individual's life span. It has been suggested that certain 'core' features do not change whereas 'secondary' features may change over a period of time. However, as can be seen, most of the work till date is focused on the dramatic cluster, that too mainly borderline and antisocial personality disorders and there is a dearth of research related to other personality disorders.

15.3.4 Parenting Style and Neighborhood

Maladaptive parenting style is another important antecedent of personality disorders. Again, most studies have focused on borderline and antisocial personality disorders. Measures of poor parenting style such as hitting, shouting, hostility, resentment, physical, and emotional abuse have all been associated with increased subsequent development of personality disorder symptoms, especially borderline (Waylen et al. 2008; Johnson et al. 2006). Other features such as low closeness to parents, maternal over-control and disorganized attachment patterns have also been noted to have similar associations. A wide oscillation from hostile over-control to passive aloofness is often seen in mothers with borderline personality pathology and this raises the risk of personality disorders for their children (Stepp et al. 2011).

Neighborhood also seems to play an important role in shaping personality and this is best exemplified by studies related to antisocial personality disorder. Presence of delinquent peers in the neighborhood along with an environment of socio-economic adversity, crime and violence has been reported to promote the continuation of conduct and antisocial personality symptoms (Piehler and Dishion 2008). Similar observations have been made in case of borderline personality, especially with regard to self-injurious behaviors (Prinstein et al. 2000).

15.3.5 Parental Psychopathology

History of substance use in parents and presence of parental psychopathology are also known antecedents for personality disorders, usually of the borderline type. As shall be described in a subsequent section, presence of personality disorders in parents, especially of the dramatic cluster, tend to increase risk of developing the same in their children (Stepp et al. 2011). Presence of schizophrenia in parents increases risk for development of the eccentric cluster of personality disorders in their children. As is the case for many of the other previously discussed antecedents, the possibility of confounding exists here too. Presence of substance use and psychopathology in parent is often linked to separation, divorce, lower socioeconomic status, and poor parenting style; all known antecedents of personality disorders.

15.3.6 Psychiatric Disorder in Childhood

Early development of many psychiatric disorders increases the risk for further development of personality disorders. In some cases, this represents a continuity of the same symptoms with age appropriate changes into adulthood. In others, the development of personality problems may be viewed as reactive. A third possibility is that of shared risk between the said psychiatric disorders and personality disorders. Finally, the possibility of a flawed diagnostic system artificially dividing a single disorder into multiple parts, giving an impression of one disorder being the antecedent of the other is also possible. Some of the better described associations are discussed below.

15.3.6.1 Conduct Disorder

Conduct disorder or symptoms represent one of the most well studied antecedents of personality disorders, specifically for the antisocial type (Hill 2003). A number of parameters are available to identify those who are likely to have greater problems within this group. Some of these factors include earlier development of antisocial behavior, greater number of conduct symptoms, greater severity of conduct symptoms and longer persistence of such symptoms. It was conventionally believed that children with conduct symptoms who remitted actually 'recovered,' i.e., did not retain the risk of developing antisocial personality; however, this may not be entirely true. Some evidence suggests that although the risk of developing a syndromal antisocial personality disorder is definitely reduced, such children do develop a number of problems in adulthood. Such problems typically included poor educational and occupational performance, social isolation, depression and anxiety-related problems (Hill 2003). As is the case with other antecedents, it is

only logical to assume numerous confounders in relation to presence of conduct disorder, e.g., lower socio-economic status, substance use, parental separation, separation and so on. However, there is some evidence to suggest that the risk attributable to conduct disorder is over and above, and independent of such factors (Hill 2003).

15.3.6.2 Attention Deficit Hyperactivity Disorder (ADHD)

The association between ADHD and future personality disorders is somewhat controversial. ADHD is often closely associated with conduct or oppositional defiant disorder leading to the confusion. However, careful longitudinal studies have shown that ADHD in itself does not elevate risk for criminality—in other words, the rates of criminality are similar in those with conduct disorder alone versus those with both conduct disorder and ADHD (Mordre et al. 2011).

15.3.6.3 Other Disorders

Depressive disorders in childhood have been found to increase the risk of personality disorders across all three clusters (Cohen et al. 2005). Similarly, childhood anxiety disorders impart an increased risk of adult paranoid and anankastic personality disorders (Kasen et al. 2001). Childhood anxiety disorders also increased risk of suicidal attempts across various personality disorders across clusters (Rudd et al. 2004).

15.3.7 Socio-economic Adversity

A number of studies have looked into the effects of socio-economic status in early childhood on development of personality disorders in adulthood. The findings are somewhat inconclusive. A longitudinal study found increased risk for borderline and antisocial personality disorders in children exposed to economic adversities (Crawford et al. 2009; Lahey et al. 2005). Widom et al. (2009) on the other hand, did not find any such association. Such discrepancies may be attributed to several factors such as small effect of the adversity on future personality making differences of power between studies important. Socio-economic status may act as a confounding factor influencing many other variables; for instance weight and other anthropometric disadvantages. Similarly, family conflict, abuse, separation may all be influenced by socio-economic status. Further, socio-economic status is known to modify effects of separation from parents (Tieman et al. 2005). Other methodological differences include varying definitions of socio-economic strata and varying impact of a particular status in different countries, regions or cultures.

15.3.8 Parental Separation

A major limitation of studies on parental separation is that they are of a retrospective nature. Available evidence suggests that a number of personality disorders of varying severity occur in relation to parental separation, especially of the dramatic cluster (Lahti et al. 2008). Parental separation could be of various types depending upon the extent and severity of separation. Being separated from both parents versus living with a single parent can have different impacts on the child. Similarly, some studies suggest that the duration of separation and the age at separation can potentially impact development of personality pathology.

Presence of a single parent or separation by the age of 14 years was found to considerably increase the risk of Cluster B (dramatic) personality disorders (Mäkikyrö et al. 1998; Kantojärvi et al. 2008). The risk was particularly increased for borderline and antisocial personality disorders. Moreover, there is some evidence to suggest that separation before the age of 5 years predisposes to borderline personality disorder (Crawford et al. 2009) whereas even earlier separation (before 3 years of age) may lead to 'psychopathic' personality (Gao et al. 2010b) in later life. Long periods of separation in the first 2 years of life have been found in some studies to heighten risk of schizotypal disorder (Anglin et al. 2008).

15.3.9 Biological Variables

A number of biological factors have been postulated as indicative of specific personality disorders. Again, they are best studied for borderline and antisocial personality disorders. Many of the variables described below are either actually or potentially involved in mediating the role of various antecedents discussed so far. As shall be seen, these biological variables play crucial roles in various aspects of the functioning of the developing brain, including how various environmental stimuli are perceived and handled, and, what impact such stimuli have on modeling future cognitions and behaviors, and eventually the personality itself.

15.3.9.1 Monoamine Oxidase A (MAO-A)

The plausibility of the role for enzymes degrading the major brain neurotransmitters such as serotonin, dopamine, nor-adrenaline and acetylcholine in various psychiatric conditions was always evident to researchers. One such enzyme, the MAO-A, has been studied in relation to antisocial personality disorders (Reti et al. 2011). Evidence suggests that among Caucasians, the high activity allele of MAO-A was protective against antisocial personality disorder amongst those without a history of physical abuse (Reti et al. 2011).

15.3.9.2 Serotonin Transporter

The serotonin transporter gene is among the most widely studied genes in all of psychiatry. Its regulation of synaptic serotonin levels implies obvious relevance to numerous psychiatric disorders. There is preliminary evidence to suggest that the serotonin transporter gene mediates the effect of various previously mentioned antecedents on the risk of development of antisocial personality disorder (Douglas et al. 2011). African-American women, with homozygous low activity alleles of the serotonin transporter gene were found to have higher risk of developing antisocial personality disorder (Douglas et al. 2011).

15.3.9.3 Catechol-O-Methyl Transferase (COMT)

As is well known, the COMT enzyme is a key enzyme in the metabolism of various monoamine neurotransmitters. Thus, alteration in its genetic structure has been studied in various psychiatric disorders. The replacement of valine for methionine results in a version of the enzyme that is more active. This plausibly could cause lowered dopamine levels in the brain. A study found that antisocial personality traits were much higher in those carrying the valine substituted COMT gene than in controls (Thapar et al. 2005). Also, preliminary evidence for a gene-environment interaction of the COMT gene and low birth weight was found in males (Thapar et al. 2005). It is being speculated that the counterpart of the same in females is borderline personality disorder (Beauchaine et al. 2009).

15.3.9.4 Amygdalar Structure and Fear Conditioning

The amygdale is a crucial structure in the brain related to various aspects of fear recognition and response. Since, consequence of a given action is an important variable that determines future repetition (or absence of) of the act, fear has been considered to be an important factor in shaping behavior and personality. In well adjusted individuals, it is believed that antisocial acts during childhood result in consequences that induce fear, which in turn, results in learning that such behaviors are not desirable and are not to be performed. In individuals with various constitutional and environmental factors favoring eventual development of antisocial personality, fear conditioning is poor and leads to a failure in modifying the antisocial behaviors. In a landmark study (Gao et al. 2010a), investigators measured fear conditioning at 3 years of age. Twenty years later, the relationship of fear conditioning with adult criminal behavior was assessed. Poor to no fear conditioning was found in them, in keeping with the aforementioned hypothesis. Low levels of amygdalar activation and an inability to recognize cues signaling threat as well as fearful facial expressions were also seen, giving clues to potential mechanisms of antisocial behavior. However, it must be remembered that these findings are at the group level and cannot be extrapolated to individuals. Criminal behavior of an

individual is a combined effect of a multitude of factors. Indeed, in certain studies, excessive activation of the amygdale have been linked to antisocial personality questioning the previously mentioned hypothesis, or alternatively, suggesting that different mechanisms may be at work in different groups of individuals (Meyer-Lindenberg et al. 2006). One such explanation is that the increased activation represents heightened anxiety related to pathological aggression.

15.3.9.5 Epigenetics

It is known that many psychiatric disorders including personality disorders have a strong 'genetic' basis. Such a basis is typically polygenic, meaning that a number of genes, each with a small effect act together to account for the variation. Indeed, such an assumption has been made for many other psychiatric disorders as well. This has often been interpreted as fixed genes present at birth that pose a certain vulnerability, which when combined with other influences such as environmental, result in the disorder in question. However, more recent research suggests that this is partly true and partly false. Although born with a certain chromosomal quota, the non DNA elements can be altered to influence the downstream effects of genes—a phenomenon that epigenetics deals with. The best known of such mechanisms are the acetylation or methylation of histones. It was earlier believed that such epigenetic changes are possible only in early infancy. However, now it is known to occur at much older ages too (Champagne 2008). This potentially represents one of the mechanisms by which the environment influences development of a psychiatric disorder, including personality disorders. It has also become increasingly clear over the years that such epigenetic influences may be much more than initially imagined. In fact the current consensus is that most behavior traits are shaped epigenetically by way of gene-environment interactions. Thus, an individual begins with certain broad dispositions which progressively branch out to generate more specific and subtle variations defining individual personality traits (Templeton 2006). Thus, effects of a 'bad environment' may be undone by the effects of a 'good environment.' Such epigenetic changes may be partially transmitted to further generations, or altered by counteracting environmental influences; all without any change in the DNA sequence. Such changes are not detectable by traditional quantitative genetics, explaining its elusive nature (Templeton 2006). This is an area of active research interest with the potential to identify newer ways of understanding human behavior.

15.3.9.6 Others

Several other genes have been postulated in the etiology of personality disorders for which clear and direct evidence are lacking. These genes have been typically speculated to increase risk for some of the key traits common to several personality disorders. For instance, dopamine receptor D4 (DRD4), dopamine transporter (DAT1) and dopamine receptor D2 (DRD2) have been considered as influencing

impulsivity and in turn increasing risk of borderline and antisocial personality disorders, besides other psychiatric disorders such as ADHD (Beauchaine et al. 2009).

15.4 Future Directions

Further studies need to assess interactions between genetic and environmental factors contributing to development of personality disorders. The exact neurobiological mechanisms through which these factors work also need to be explored. Prospective studies collecting data on genome and epigenome, HPA axis, and different early life stressors will help in identifying the possible etiological roles played by each of these factors and the interactions among them. Moderating and mediating roles of different etiological factors should also be simultaneously assessed. Functional brain imaging methods could help in identifying the abnormalities in brain functioning associated with these disorders characterized by potentially reduced brain growth in prenatal life. Above all, use of this knowledge to design early intervention programs is also the need of the hour. Some work has already been done in this area, particularly for borderline personality disorder. Its close association with mood disorders has encouraged an integrated early intervention program based on clinical staging (Chanen et al. 2016).

15.5 Conclusions

Traditionally, there has been some reluctance in acknowledging that personality disorders have antecedents in childhood and need to be described and understood since the early years of life. However, over the past few years, this view has garnered wider acceptance and some of the traditional views have become untenable. The DSM-5 too, has now considered personality from a life cycle perspective. In keeping with the DOHaD hypothesis, a range of adult conditions are now being viewed from a developmental perspective and search is on to find out their antecedents, or even better, their etiological factors. Such a search has also instilled some hope that such disorders may not necessarily have a chronic and relentless course. Future studies must actively look into the various antecedents with the eventual goal of building capabilities to intervene early enough to delay, retard, or completely prevent these disorders.

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