

# **The Interaction of Nature and Nurture and their Influence as Mechanisms of Change in Human Development**

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## **ABSTRACT**

*There has been a vigorous debate in recent time, about heredity versus environment and how they coalesce to create personalities, behaviours, and psychopathology that appeared unique to each person and influenced their developmental process. This study uses different methodological approaches to lifespan development to measure and analyze the interplay of genes and environments on human behaviour and underline how the interaction contributes to behavioural changes and continuity. The study focuses on the interaction between heritable and ecological factors in the path of behavioural growth and patently recognize etiological mechanism that supports the assumption that a particular hereditary or environmental condition ultimately lead to behavioural features and changes during development. The study used the accessible materials on lifespan development to understand the continuous source of person uniqueness in personality development. Finally, citing evidence from various multidisciplinary studies, the article concludes that shared environment significantly influences human behaviour but decline with age and that the strong interaction between the two factors formed the basis of who we are and how we behave.*

**Keywords:** *Environment, nature, nurture, personality, genetic, attitude, human development.*

## **INTRODUCTION**

Our orientation about life and the way we act to the situation around us is as results of interaction between different psychosocial factors such as genetics, social norms, core faith, and attitude. Bronfenbrenner (1979) and Lerner (2002) express that human behaviour cannot be fully understood without mentioning the changing relationship between human and the contextual environment. The milieu according to their studies is the inner biological levels while outer are the social and environmental levels. Nevertheless, both past and present literature emphasize the significant importance of social and contextual environment on specific genetic variants in human behavior and traits. Bronfenbrenner's Bioecological Theory

(Bronfenbrenner and Ceci, 1994) also emphasize the importance of interactions between people, families, peers, schools and society characteristics in explaining the concept of personality differences in development. Therefore, most reviewed studies on life span development highlights and improved our basic understanding of the mechanism involved in normal and abnormal behaviour.

***The Relationship between Heredity, Environment and Human Development:*** The introduction of heredity and environment to human development has received mixed reactions since its commencement in 1865. According to Galton (1865) the first research work on heredity and environment was issued earlier before Mendel's paper. This was developed increasingly till 1924, when the primary work on adoption and twin research on human development was reported (Merriman 1924; Theis 1924). However, ten years later the situation significantly affected the recorded progress made in the study. However, the rise of behaviourism during this period (Watson, 1930) further shows the barrier faced in the advancement of hereditary science, and have a massive bearing on the behavioural sciences despite its supposedly reassuring concept of ecological model grounded on a statement that we are who we are because of our inherited gene. Nevertheless, the work of genetic research on human behaviour continue to push for the recognition of the significance of heredities and milieu. Therefore the main research work on behavioural heredities was issued in 1960, and the study as reported in the textbook "Laid much emphasis on living animals" (Fuller and Thompson 1960). Thus, the reviewed literature reported that all aspects of psychology studied documented how genes and environment influenced twins and adoption studies (Plomin, DeFries, McClear and McGufin, 2001). Most studies on hereditary constantly reported genetic impact in numerous traditional parts of psychological studies like psychological disorder, behaviour, intellectual infirmities and skills, and substance use and misuse. Moreover, certain parts displaying the robust transmissible effect may be more shocking, like self-worth, interests, orientation, and educational accomplishment. The study further lends credence to the significance of genetic factors and the situation in the aetiology of a person uniqueness in behaviour. Additionally, recent situation shows that society also acknowledges the significant input of genetics. For instance, over 90% of caregivers and educators described heredities as the lowest contributor to mental disorder, behaviour, learning disabilities and intellect compared to the environment (Walker and Plomin 2005). However, before the swing of style changes from genetic toward the environment, it's worth mentioning that hereditary research offers the finest obtainable proof for the significance of the milieu. Thus, for the majority of these qualities, the parents and educators say it accurately: heredities and milieu each explains nearly half of the adjustment.

***Nature and Nurture:*** The assumption that race and individuality are the outcome of heredity, to say the least a biophysical phenomena, and that their interpretation is based or depended on the philosophy and process of biology has continued to gain ground and dominate debate in recent time among scholars and academician in the fields of social sciences. However, various reports show that there is a general assumption among scientists that support the causal correlation between environment, individuality and race. Consequently, research also shows that explanation of the concepts, that is, (environment and heredity) is based on their interpretation of the developing organism. On the other hand, genetic and environmental factors notably influence development of human behaviour.

Besides, reports show that human being is a creation of genetic and its past background, while present background offers the basis for a proper explanation of current behaviour. To support the argument, most researchers review diverse area under discussion to analyze human behaviour and psychopathology. This includes learning about person approaches to mental disorder (schizophrenia). It is obvious that these studies may offer answers to the debate concerning the influence of environment and genetics in human development. In addition, all efforts channeled towards establishing the relative contribution of genetic and environment on disparities observed in traits have not achieved any meaningful accomplishment. This assertion further supports the general opinion of both geneticists and psychologists which explain that human behaviour is grounded in the interaction of heredity and ecology (Haldane, 1938; Bjorklund and Pellegrini, 2002; Loevinger, 1943; Schwesinger, 1933; Woodworth, 1941; Chiappe and MacDonald, 2005). Based on that, it is assumed that the relative connection of heredity to differences in a specified trait, not stable, will definitely differ under different ecological conditions.

Socialization theories support the notion that some part of the environment like socio-economic status or couple's separation will variably or invariable create a situation whereby children raise in the same environment or under the same roof are similar to each other. Plomin and Daniels (1987) confirm that heritable sensitive strategies have reliably initiated a varied design, by indicating how the situation that influences behavioural growth operate by making children reared in the household to behave differently. This assumption is evident and quietly understood because children who did not share the same genetic factors, but reared in a similar adoptive household hardly take after or shared the same characteristics after the teenage years.

Though, siblings are a lot alike, nonetheless their resemblance is entrenched in their DNAs rather than in the shared situation. These situations are referred to as uninsured, and the reason for that is because they are not commonly experienced by children developing in a similar household. This assumption consistently raised

the question, why are the differences in behaviour among children developing in the same household? Little success has been reported with various situations displaying considerable but minor effects (Plomin, Asbury and Dunn, 2001; Turkheimer and Waldron, 2000). While, it was reported as very hard to deduce a particular non shared ecological issues responsible for huge amounts of difference, it's worth mentioning that uncommon situation is, in a broader sense, refer to how circumstances operate to impact on behaviour.

Previous efforts on the issue concentrated more on the household setting, nevertheless, it appears sensible to explain that, what is happening in the household environment, particularly, with peers and person's lifespan incidents can be a richer base of the non shared milieu (Harris, 1998). Literature also confirmed how unpremeditated factors add to the non shared milieu in a manner such as unsystematic noise, individual understandings, or elusive interaction of a concatenation of incidents. Though multiplied over time, minor changes in competence can definitely result to big variations in results. This paper explores various ways that describe the interaction between heritable and ecological factors in the path of behavioural growth and patently recognize etiological mechanism that support the assumption that a particular hereditary or environmental condition ultimately lead to behavioural feature and changes during development. Hence, the objectives of this essay are drawn from the literatures on human development and it looks at evidence that support the associations between environment and genetic factors and how this interaction lead to personal differences and changes across the life span. Therefore, the research paper aims at the following specific goals:

- i To analyze the interactions between nature and nurture as important components of behaviour
- ii To critically investigate the mediating role of genes and environment that related to risk and behaviour
- iii To explore different multidisciplinary studies that measure and focus on the interaction between environment and genetic factors at multiple levels of development and how this affects human behaviour.

## **APPROACHES**

To understand the processes involved in analyzing differences in behaviour, there is a need to look at multidisciplinary studies that measure and focus on the risk and resilience factors at many levels. The past decade has demonstrated a significant rise in studies that outline the link within a particular feature in the hereditary conditions of persons and their characteristic manners. This experience continues to show that there is a major shift from expressive and correlation strategies of the

past years to new purposeful challenges that validate illustrative hypotheses. Besides, evidence shows that categorizing differences in psychological traits have lent credence to the general beliefs about significant changes in a group features following situational adjustment. Therefore, there is an urgent need to explore various ways that explain the interaction between heritable and ecological factors in the course of behavioural growth and at the same time, clearly show etiological mechanism that supports various assumptions that any inherited or natural situation will eventually lead to a change in human trait. These assertions, though debatable, continue to dominate various research studies on human development particularly, the words, “how” of heredity and environment. In view of this argument, the following ideas and promising methodological approaches will answer the question “how” in this paper.

***The Genetic factors in Continuity and Change in Life Span Development:***

The use of genetically informative designs to analyze human behaviour has demonstrated and unravels importance of interindividual differences in trait scores that come as a result of inherited attributes (i.e., genetics) and environmental influences. Similarly, studies on genetically informative longitudinal designs has unravelled the genetic and environmental factors that measure occasions. These include the process that estimate and explain the intensity of the stability of each element (in this case, hereditary and environmental continuity). Besides the assumptions of genetic continuity, research shows that it is not ideal and faultless (i.e., reliability coefficients are constantly minus 1) from childhood to adolescence stage (De Fruyt et al., 2006; Gillespie, Evans, Wright and Martin, 2004; Spengler, Gottschling and Spinath, 2012). On the other hand, studies also show that during childhood and adolescence periods, fresh hereditary factors appeared and add to interindividual variation and sequential changes that exist in behaviour. Moreover, past and recent evidences on genetic influence show that similar to rank-order stability, genetic stability is enhanced from teenage years to middle age (Hopwood et al., 2011; Viken, Rose, Kaprio and Koskenvuo, 1994) pending middle and late adulthood when it becomes stabilized and perfect (Johnson, McGue and Krueger, 2005; Pedersen, 1993; Read, Vogler, Pedersen and Johansson, 2006).

**Deoxyribonucleic Acid (DNA):** The most significant event in hereditary study was the invention of the organization of DNA. This discovery led to the appreciation of the major structure that constitutes DNA (that is, heredity and DNA codes for protein). The future of the hereditary study of behaviour is a base for molecular heritable research of DNA that detect precise DNA variations accounted for the general effect of genetic factors in behavioural change. Detecting the DNA variations helped in solving problems or matters that continually generating debates, i.e. nature-nurture interaction, evolving, and multivariate instruments, with

higher accuracy and influence. Molecular inheritance significantly influence behavioural research and the studies do not need exceptional illustrations like in empirical hereditary research of twins and adoptee. Similarly, DNA can be acquired without much difficulty (from cheek wipes rather than blood), and this experience is also applicable to genotype of a DNA sign, which is also seen as economical. Besides, Butcher et al. (2004) established the importance of the technique called gene chips (microarrays) that can determine the genotype gene for hundred of thousand people within a period of three days. Thus, the research on genetic influence on behaviour emphasizes the fact that it is easier and cheaper to implore a recognized gene than looking for genes related with difficult personalities. This is because such practice brings an important heritable element to behavioural study (Plomin et al. 2003).

“Gene is the most single recognized threat feature for usual late-onset Alzheimer (LOAD) and that apolipo protein E (APOE), significantly engaged in transporting saturated fatty acid”. Studies confirmed that allele 4 genes definitely upsurge the danger fivefold for LOAD and documentation of the relationship amongst APOE allele 4 and LOAD were detailed ten years ago (Corder et al. 1993). Thus, it is worth mentioning that there is abundant study currently focusing on the dementia of genotype members for APOE that determine if it has different implication for persons with or devoid of this inherited threat features (Laurin et al. 2004; Mukamal et al. 2003; Podewils et al. 2005). Besides, studies maintain that genotyping APOE will turn out to be monotonous in a health setting if heritable threat influence is recognized as a predictor of differential reaction to medications or cures. Therefore, based on the recent development in the area, quite a lot of extensive behavioural works are presently procuring DNA on their models in hopes for the period when genetic factories recognized as applicable to their field of interest.

### ***The Environmental Factors in Continuity and Change in Human Development***

The results from environment studies on behaviour do without question, offers evidence that highlighted that these cured inherited gap that happened in a person’s life when he or she become 40years, is not related to the putrefaction of stability coefficients reported in older age. However, whether this assumption is convincing enough or merely suggestive is debatable. Johnson, Vernon and Feiler (2008) report how the main non genetic basis of a person’s dissimilarity in neuroticism and extroversion is unambiguous (that is to say, not distributed through relatives nurtured as one) and this according to their findings are referred to as *non shared environmental effects*. However, from a behavioural genetic perception, “environment” includes physiologically and biochemically intervened consequences, such as situational or ecologically triggered epigenetic influences. This illustrates

that of shared environmental factors demonstrate the disparity in permanence (that is, environmental continuity) in term of age analyzed. Gillespie et al. (2004) report that children who are twins and age between 12 years and 16 years, show significant low coefficients in ecological continuity for extraversion (that is to say, .17 and .18 was reported for males and females respectively) and neuroticism (.12 and .36 for male and females respectively).

Hopwood et al (2011) conducted a study on developmental changes commencing teenage years on the way to adulthood (17- 24 yrs) and report natural-continuity constants of .36 for negative emotive (neuroticism), .37 in support of common positive expressivity, and .39 for inherited constructive emotive in behaviour, while (the last two qualities represent parts of extraversion). Moreover, their study also investigates the continuity sequence within the ages of 24 and 29, and for this intermission, it reports larger ecological continuity, with constants varied between .56 - .60. However, Johnson et.al (2005) observe 5-year ecological-stability constants over .70 for middle adulthood. In addition, findings from Pedersen and Reynolds (1998); Read et al. (2006) studies show high decrease in environmental continuity in old age.

***The Genetic-Environmental Interaction:*** The evidence often cited in various research studies in support of genetics and environment interaction shows that there is a high correlation between life narratives, memories and experiences, and at the same time this may have cumulative effects across the life span. Accordingly, McCartney, Harris and Bernieri (1990) confirm that environment has significant consequences of interindividual disparities on neuroticism and extraversion and also enhance through age, hence, results to ecological variance. Nevertheless, an increase in interindividual difference as a result of ecological effects that rise with age, will lead to decrease in gaps that come from genetic effects. Therefore, it's worth noting that inherited assessed qualities decrease with age. Thus, on genetics that have measured heritability assess for diverse age cluster (Loehlin and Martin, 2001) reported no considerable disparities between age groups.

On the contrary, findings from studies combining cross-sectional information (on diverse age cohorts) and longitudinal information continue to show how heritability estimation lessening by age (Kandler et al., 2010; Viken et al., 1994). Besides, researchers have derived various methods that ascribe various personality changes to ecological factors like personal necessities in social roles (e.g., worker, partner, caregiver) associated capitals (Roberts and Wood, 2006), normative life expectancy stages (e.g., moving from one's paternal home, finishing education/a trade, leaving job due to old age), personal life situation that influence action that changes one's life (e.g., accidents, having a child or, marriage). To sum it up, personality growth and adjustment are significantly linked with age-graded social functions and correlated expertise (Roberts, Wood and Caspi, 2008).

Therefore to buttress this argument, similar evidence from another genetically revealing meta-analytic review study of fifteen diverse age groups showed high correlation between the amount of irritability of neuroticism and extraversion and age studied. This heritability of extraversion has a propensity to increase in people in their early 40s, and later declines continue, while for neuroticism, Johnson (2010) shows that heritability progressively decreases when people have reached adulthood. On the other hand, the model observed amid cognitive ability with age, shows differences in the genetic contributions to interindividual disparities in neuroticism and extraversion. However, Johnson's (2010) study on cognitive abilities continually show how genetics rise with age and this further confirmed the position that genetic material and environmental effects differentially contribute to human behaviour (i.e. Personality) and the level of their competence (i.e. abilities or skills). Therefore, a boost in the degree of behavioural trait inherent may replicate high consequence of dynamic and complex inheritable factor-environment correlations.

This assumption proofs that individuals actively manipulate and influence their surroundings (for example, by changing their acquaintances, abodes or professions) or exhibit social responses that relates to their heritable susceptible qualities. Similarly, environment and social reactions influence the individual's disposition. For instance, a hereditarily extroverted person may perceive life expectancy events as manageable and constructive. Nonetheless, understanding life expectancy as manageable and productive will definitely enhance that kind of person's strengths of extraversion (Kandler, Bleidorn, Riemann, Angleitner and Spinath, 2012). This shows that inherited effects to a degree build the ecological effects accounting for the mounting genetics components. Therefore, the procedures needed in inheritable factor-environment correlations determine the progression of reasoning abilities through the lifespan and the growth of extraversion in the earlier year.

***Belief and Culture:*** Another potential study about the environment and genetic influence on human development was sighted in the relative study of nurturing practices in diverse beliefs and cultures. One of the earlier researches on culture was reported by Whiting and Child (1953). In the study, they make use of data on 75 local people from the Cross-Cultural Files of the Yale Institute of Human Relations, to check the amount of assumptions concerning associations on nurturing practices and behavioural growth. This investigation was trailed by field remarks in five cultures, the effects of which up till now are not documented (Whiting, et al, 1954). However, evidence shows that similar studies conducted emphasized on various psychological situations offered by diverse social classes (Davis, et al., 1946). Nevertheless, one of the most interesting studies is the one conducted by Williams and Scott (1953) which focused on the relationship concerning socio-



economic level, broad-mindedness and motor-progression amongst Negro children, and Milner (1951) on the connection involving the reading willingness in first-grade youngsters and methods of parents-child relations. However, Milner (1951) reports disparity between the lower-class child and the middle-class child. Moreover, the study analyzed the disparity between the two classes of children as “a heartfelt progressive family situation or adult-engagement system which act or seen as a motivational requirement for adult-controlled learning.” However, the findings indicate that children from the lower-class see adults as mostly hostile. Furthermore, the study reports abroad prospect to relate orally to adult members in a household as a motivating factor (that is, attitude demonstrated by parents on chatting during meal time). However, the findings further show that parents from lower background tend to suppress and dampen down such discussion, on the other hand, parents from well to do family background will encourage such engagement in their home.

**Twin Studies:** It has been well established that the argument about nature and nurture influence on human behaviour cannot complete without exploring on twin studies. However, evidence shows that most well-liked research on twin studies focuses on evaluating the resemblance between MZ and DZ twins jointly raised in the same environment or on a variable of interest (Jang, 2005). Similarly, both identical twins, and monozygotic twins, are siblings with shared genotypes. Besides, it was established that study of identical twins serves as the best indicator that measure the significant influence of biology on traits and psychopathology in human development. For example, for a twin to have a dark hair or brown eyes, then the other twin will share the same characteristic of dark hair or brown eyes as well. Nevertheless, Plomin, DeFries, McClearn and Rutter, (1997) show that the concept of identical genes perfectly match the phenotypes that form the orientation and character of identical twins.

Besides, studies of identical twins or dizygotic twins show that they shared half of their genetic traits with each other. Moreover, despite sharing 50% genetic traits, the results show that they provide less interpretation like identical twins in interpreting the level of heritable effect. Nevertheless, they act as a commendable point of reference meant for evaluating identical twins. The study of fraternal twins shows the similarity that exists in first-degree family, except that they are not sharing the precise same age, like identical twins. Consequently, twin research typically depends on an illustration of monozygotic and dizygotic twins. However, in a situation where biological influence is much more than ecological influence, dizygotic twins would have or display psychopathology behaviour similar to each other compare to monozygotic twins (Plomin et al., 1997). In addition, this assertion further highlights the significant influence of heritability coefficient in human behaviour, that is, the estimate of how individual’s particular trait compares to

others with similar characteristic traits is related to genetic materials (Olson, Vernon, Harris, Aitken and Jang, 2001). Hence, the coefficient is significantly higher in identical twins compare to fraternal twins. Besides, identical twins often show diverse phenotypes (outside manifestation of genes) for similar genotypes (inherited composition). Thus, Hughes et al. (2005) explain that these attributes if noticed, represent non shared situations, despite identical twins sharing the same genetic composition. Hughes et al. (2005) show that they may experience different orientation all through their lives and this actually form the personality, behavior, and psychopathology that further express and uphold their uniqueness from one another.

**Attitudes:** Olson et al. (2001) conducted a study that defines the inherited of mindset and the innate features, such as cognitive which influence feelings and behaviour among pairs of twins. Their findings show that there is significant correlation between attitudes displayed by the participants and genetic factors. They also identify that assertiveness linked to self-reported perceptions or actions are frequently connected. The study for instance, asked the participants to grade the characteristic of their friendliness, and the findings show that the trait was related to 5 out of 6 behavioural features the participants displayed toward friendliness. Also the report shows high correlation between attitudes toward athleticism and self-reported athletic abilities. Evidence highlights that contributory model was particularly sustained in the results, for the reason that the physical ability (the mediator), is related to approaches towards athleticism.

However, despite the general assertion on this model, evidence shows that the model is not free of criticisms, for example Olson et al. (2001) report that it is difficult to believe that X is the source for the occurrence of Y in all circumstances. However, it is generally assumed that approach to governance seemed to be connected to soaring self-ratings of physical attraction, friendliness, and fierceness. Nevertheless, the implication of these various factors on behaviour, is not probable to correctly think of constant associations regarding inherited traits and approaches to life (Olson et al., 2001).

Additionally, evidence proofs that uninsured background experiences between duos of twins played significant impacts in determining the level of attitude variances and this overriding heritable predisposition and collective environment involvements (Olson et al., 2001). Moreover the term “non shared setting” refers to as the element within the environment that have direct influence on one of the twins while the other one remains constant (Van den Oord, Boomsma and Verhulst, 2000). Therefore some of the non shared environment experiences are highly related to feelings and self-assessment of physical features and cleverness (Olson et al. 2001).

**Theory of Mind:** Hughes et al. (2005) show that beliefs, intents and desires are very paramount to the development of human mental state. This assertion is well mentioned in various studies on human development as a theory of mind and clearly explained falsely interpretation or representation of the object and situation of a child at the age of four. However, based on these findings, research continues to search for answer on what really accounts for the variation in children false-belief grasp between biology and environment? Though, numerous evidences proved that children from extended families developed fast attainment of assumption of mind, yet hearing-impaired children born to families of hearing adults show slow accomplishment of theory of mind. This situation is due to cultural influences that come because of environmental influences.

Moreover, children with autism disorder also exhibit impaired theory of minds, these include girls diagnosed of chromosomal disorder (Turner's syndrome); the development is linked to heritable effects (Hughes et al., 2005). Hughes et al. (2005) conducted a research on identical and fraternal twin to test the implication of genetic and environment of theory of mind. The study explores social status, spoken ability, and the assumption of mind of each subject. The survey questions contain information that tested the aptitudes of participant to tie a phony conviction around a character presented the stories given, while the second phase of the survey package contains questions that measured the participants' skills to assume and abilities to deduce an untrue conviction about behaviour in a given story. However, findings show that the recorded variation that exists between the twin's theories of minds is due to uninsured environment and the proportional power in lessening direction were linked to common situations, vocal aptitudes, and heredity. Thus, a family with twins is associated with high sense of competitiveness. In addition, the more the people in a household talk about issues, the faster the concept of mindfulness. The study further confirmed that environmental influences are more significant than heritable influences in the growth of concept of awareness in children. However, this situation does not overshadow the reality of genetic materials and features completely (Hughes et al., 2005).

**Adoption Studies:** Interestingly, research on adoption and how it influences human behaviour and psychopathology is highly debated by researchers studying heredity and environment. Adoption studies are very vital to human development and contain factors that invariably responsible for the disparities in human behaviour: biological caregivers and environmental caregivers. Similarly, the significant relationship that is associated with biological ancestries and the adopted youngster is influenced or related to genetics. Likewise Plomin et al. (1997) also reveal that any association or links involving the adoptive parents, and the adopted youngster is typically ascribed to the contextual setting.

Additionally a review of literature consistently mentioned that the basis for research on adoption is to ascertain if the adopted offspring have the same characteristics like their blood relatives, which is a sign of heredity effects, and their common environmental effects which signify their connection with their adoptive families. Most of the work on adoption studies were carried out in Scandinavian countries, where the researchers are allowed to assess and use the national records to determine relatively vast and representative cohorts of adopted persons along with their adoptive and biological relatives. Similar to twin studies, evidence shows that most research work on adoption studies are exploring from empirical research, the majority of which supported the effectiveness of this method (Cadoret 1986, Plomin et al 1990b). However, an adoptive household will less represent those who are poor and the underprivileged people in the society thus limited the significance of ecological effects in adoption research. Therefore, ecological implications are related to the few fortunate or generally established middle classes.

***Intelligence:*** Research has continued to associate individual uniqueness in intelligence as a noticeable feature of human psychology, and as a strong predictor of individual life outcome. The source of individual differences in IQ is mostly discussed, and this discussion continuously ponders on whether differences noted in IQ are related to heredities or the environment, usually denoted as the “nature vs. nurture” discussion. The origin and heritability of intelligence continue to divide scholars’ opinion particularly how differences in human intelligence is attributed to genetics and not environmental. Recently, evidence shows that most of the argument is based on whether the elements of IQ instability alter with age or splitting ecological effects into common and uncommon elements. Thus absence of empirical foundation that supports both the natural and the ecological theories is confirmed by a huge chain of data collated in the past years.

In fact, reports evidently show that inconsistency in reasoning skills and behaviour in person is due to the interaction of inherited and ecological factors. Therefore common environment is seen as all ecological factors that make people who grew up or reared in the same household to behave in the same way. Scarr and Weinberg (1978), Teasdale and Owen (1984) in their studies engaged mostly 18 years older people, while Loehlin, Willerman and Horn (1997) used a longitudinal research and administered IQ tests on participated member one time at a median age of 8 and 10 years respectively. Their findings document high rise in the transmission of IQ through age at about 30% in infancy to 80% in adulthood (Spinath et al, 2006; Johnson et al, 2007; Jacobs et al, 2007; Edmonds et al, 2008; Deary et al, 2009). However, existing models of genetics-environment associations of human intellect assume that every age indicates particular genetic and environmental effects, and this resulted in variation of IQ in the same person

(Brant et al, 2009). This according to them is related to the inherent regulator on the morphological modifications that disturb brain in the course of development, with the galvanization of varied genetic factors at diverse ages (Deary et al, 2009). However, apart from the impact of the hereditary influence rise through growth, non-shared setting adds significantly to adjust through the ages, while common ecological effects decline, and unassertive in adulthood as related to infancy (Brant et al, 2009). Thus, non-common setting comprises elements expected of each person, and these practices are denoted by several ethnic and societal factors. Therefore, individual interest such as, reading, viewing TV and interests are deduced as non-shared settings and they play a significant role in determining reasoning abilities in later life. Interestingly, most evidence of adoption studies also buttressed the general assumption that significant effects of common environment on intelligence are limited to infancy and early teenage years.

However, this statement continues to raise serious debate because most of the adoptive families might not be a true representative of the whole population. This is because the adoptive parents are either chosen by the organizations or select themselves and apart from that evidence shows that they are well positioned, considerably older, educated and richer than natural parents. Thus, most of the adoptive parents took the decision to adopt because of their flair for raising children. This they do after serious consideration and planning and it's quite different from biological children that were a result of unintended gestations. To support the argument, in most cases, people who are unfortunate in life, particularly those who are poor, those with a history of drugs, alcohol, conduct disorders and those who have problems of having their own kids are mostly excluded from adopting children.

***Bipolar Disorder and Schizophrenia:*** A review studies on epidemiology and family studies advocate a number of resemblances between Schizophrenia and Bipolar disorders. On the other hand, family studies, mostly, propose a narrow overlapping threat for SA diagnoses and several affective disorders (Gershon et al 1988; Kendler et al 1993; Maier et al 1993). According to the study conducted by (Berrettini, 2000) family studies, including molecular genetic studies were carried out to determine the level of overlapping of genetic risk that exists for both disorders. This confirms the similarities that exist between bipolar disorder and schizophrenia from the normal age of inception to the courses of the illnesses. On the other hand, bipolar disorder shows that there is significant correlation between first-degree relatives of people with bipolar disorder and related mental disorders such as bipolar I disorder, bipolar II disorder, schizo affective disorder, and recurrent unipolar disorder. However, this does not show higher risk for schizophrenia itself (Berrettini, 2000). First-degree relatives of people who suffered from schizophrenia are prone to schizophrenia, schizo-affective disorder, and

recurrent unipolar disorder, but not for bipolar disorder. Additionally, first-degree relatives of people with bipolar disorder and schizophrenia will definitely have a higher risk for schizo-affective and recurrent unipolar disorders. These findings justified the significant relationship and overlapping between familial risk for bipolar disorder and schizophrenia (Berrettini, 2000). An earlier study on adoption shows that family environment has a less significant influence on child's mental disorder such as schizophrenia. However, in a study carried out on adopting offspring of biological mothers who have a medical condition of schizophrenia, and that of adopted offspring whom biological parents are free of mental disorder, Plomin et al. (1997) report that there is a significant relation between adopted children of schizophrenic mothers and schizophrenia, while adopted children of parents who didn't suffer schizophrenia show no sign of schizophrenia in life. Moreover, this supports the general assumption that stressed the point that no matter the theoretical perspectives, the particular environment that a child grew up did not influence risk for a disorder. For instance, if a child's parents have a medical condition of mental disorder, there is a high tendency that the child will experience the same risk disorder even if he or she is nurtured by biological or adoptive parents (Plomin et al., 1997).

Furthermore, evidence from Plomin et al. (1997) on adoption study shows high proportion and significant correlation between adoptees of birth parents with schizophrenia, and flaunted schizophrenic-like behaviours. Therefore, Plomin et al. (1997) findings continue to support the assumption of genotype-environment interaction theory, and further lay credence to the general expression that genotype is related to the environmental background. Additionally, the adoptees whom his/her natural caregiver experienced schizophrenia will show greater chances of schizophrenia or related associated syndromes if the adoptive relatives have low functioning. Despite the success recorded in research about genotype-environment interaction; evidence continues to indicate the difficulties encounter in answering what openly causes schizophrenia, and why it is not highly noticed amongst adopted children. Consequently, one of the main concerns recorded in literature is the lack of information of a genetic material that bears the syndrome schizophrenia. Hence, it is new to figure out whether such genetic factor exists in human beings, and if so, it is doubly difficult to comprehend at what level does this potential gene control these forms of adoption studies (Loehlin, Willerman and Horn, 1988).

***Infant Shyness:*** Daniels and Plomin (1985) examines the genetic-environment interaction in adoptive study was conducted to unravel the reasons why there is a major difference in the way infants respond to attention. Some infants are openly responsive to attention; some are slow, while others are afraid and reserved. Moreover, it is difficult to deduced whether babies are shy because their mothers do not seldom take them out, or because the mothers transfer the genetic shyness

trait to their child. Consequently, the general procedures highlighted in this study further confirmed the significant association between the infants, adoptive and biological parents' shyness, parental sociability, and parental introversion-extraversion (Daniels and Plomin, 1985). Self-reported ratings of the genes were carried out before giving birth to the infants, and the rating of the infants' shyness was achieved by the adoptive parents when the babies reached the age of two. Reports on no adoptive families showed that there is significant correlation between parents who reported high rates of shyness, low rates of sociability, and high rates of introversion and shy infants. A similar report was also recorded in adoptive families whose parents rated in the same way and this further indicates the interplay between home environment and genetics on cognitive and social development (Daniels and Plomin, 1985). Moreover, the report confirms that biological mothers are rated high in introversion and this also applicable to their adopted-away babies. Therefore, the significant importance of a genetic link over family environment stressed the need for further research on the issue (Daniels and Plomin, 1985).

***Antisocial Personality Disorder:*** Substantial evidence documented from twin and adoption studies highlight the significant contribution of inherited and shared ecological features in the development of antisocial behaviour (Maes et al, 2007). Literature continues to support the assertion that genetic predisposition contributes significantly to various behavioural disturbances such as antisocial and violent behaviour in human beings. Recent evidence of assessment and meta-analysis of 24 heritably explanatory researches about violent behaviour established that genetic explanation is responsible for almost 50% of the change (Miles and Carey, 1997; Rhee and Waldman, 2002). Besides, no genetic factor responsively impacts on behaviour, being the inherited effect on behaviour interceded the action of vital regulators, such as neurotransmitters (Popova, 2008).

Additionally, various attempts are taken to determine any correlation between children who are at risk for antisocial personality disorder and development of symptoms in an adoptive family environment, or whether such environment will save from developing disorder's symptoms. However, Roth and Finley (1998) confirm that there is high tendency that antisocial personality disorder is likely to develop in adoptees with biological risk factors (at least one biological parent had a background of criminality or antisocial personality disorder). Similarly, adoptees with no history of developmental disorder will not have the symptom, despite living in an adoptive environment.

Therefore the interaction between adoptive family environment and the preexisting biological risk make antisocial personality disorder relatively common among adoptees (Roth and Finley, 1998). Hence, adoptees will develop higher possibility for antisocial personality disorder if their biological and adoptive parents are both from criminal backgrounds. However, despite this assumption, many

factors need to be considered before arriving at that conclusion. For example, there is a need to affirm or illuminate on whether this disorder is as a result of a characteristics of the biological mother, or father. Nevertheless, evidence shows that most of these studies were conducted using only information generated from the biological mother, without involving the other important segment, the biological father. Moreover, there is no general consensus about the use of criminal background as an immediate checkmark for analyzing antisocial personality disorder in biological and adoptive parents. Although it is commonly ascribed that the presence of a biological parents criminal background means that such parent has a medical condition of antisocial personality disorder, at the same time he or she has transferred it down to the adopted-away offspring. These results confirm that such problem cannot be proved and that lack of a criminal background means lack of the disorder itself (Roth and Finley, 1998).

**Family Studies:** Family studies continue to act as a benchmark and imperative factor that discover and determine the level of menace of relatives developing mental disorders that are common and affect other family members. Thus to support this assertion, case-control family studies are employed, and these include total number of relative risk and population relative risk of a mental illness. Moreover, Jang (2005) reports that the relative risk correlate with the possibility that a family member of a person with a mental will develop a disorder than someone from a family with no history of mental disorder. As a result, the report shows that population relative risk calculates roughly the level of risk that affect a person from families with mental health condition as opposed to families of a person with no history of any mental illness (Jang, 2005). It was long-established that family studies have often served as a benchmark that determines the menace of transferring mental disorders to children within families.

However, this does not totally explain the significant influence and contribution of outside factors, such as family environment and culture. These studies are performed using molecular genetic studies, and the DNA is taken out from participants' blood samples, which further explain that the correlation between the DNA and the observed behaviour is projected. Therefore, the most common molecular genetics study is called linkage analysis. This kind of study tries to locate a specific gene on a chromosome in the human body. Therefore, if a gene for a particular mental illness is being investigated, researchers need to identify an already-known gene on the chromosome and make it with a marker. Therefore, the location of the markers and that of the actual diseased gene is very vital. However, the closer the two are, the higher the possibility that the disease and marker genes are related or connected together (Jang, 2005).



**Attention Deficit Hyperactivity Disorder:** Research on molecular genetics on ADHD has come out with several credible candidate inheritable factors (Dopamine D4 receptor gene (DRD4), Dopamine D5 receptor gene (DRD5), Dopamine transporter (DAT 1) gene and Catechol o-methyl transferase gene (COMT). Thus, evidence from relationship studies documented limited results on effects of inheritable factor variants (Faraone et al, 2005), and the relationship results by a number of signs are changing across various research that is, DAT 1 (Banaschewski et al., 2009, Coghill and Banaschewski, 2009). Therefore, this change might be as a result of the control of heritable influences by ecological effects that vary among models.

Thapar et al. (2007) highlight that phenotypic intricacy, and variation in the continuousness and variations in medical display in ADHD will together swayed by the interaction amongst pre-and perinatal along with psychosocial, ecological as well as inherited threat issues. The effects of ecological issues, for instance, intrauterine coverage for diverse medications (prenatal smoking experience (Becker et al., 2008, Khan et al., 2003), liquor intake in the course of pregnancy (Brookes et al, 2006, Langley et al., 2008), psychosocial hardship (Laucht *et al.*, 2007) mothers' voiced feeling (EE) (Rookes et al, 2006, Psychogiou et al., 2008) stern early withdrawal (Sonuga-Barke and Rubia, 2008, Stevens et al., 2009), or low birth weight (Langley et al., 2008, Thapar et al., 2005), were all documented in G 9 E research. Apart from emphasizing the impacts of the milieu in regulating inherited effects, report shows that a number of these research offers proof of hereditary support to continuousness of the condition (El-Faddagh et al, 2004) and growth of comorbid conduct disorder (Langley et al., 2008; Thapar et al., 2005).

**Obesity:** The debate on obesity has continued to generate interest in recent time. One of the main arguments is about what step can be taken to tackle the epidemic. Literature on the topic shows that the discussion will take many ways. Some of these question include, whether the fault is individual or shared environment? Is it biological or behavioural? Is it hereditary or ecological? For the purpose of this paper, is it "nature" or "nurture"? Basically, this argument is not limited to academic discourse because the discussions also define economic obligation, distributions of maintenance, right to programmes, covered by health scheme, and administrative compensation. This paper establishes that each one of these opinions is a "straw man" for the causal path physiologic procedures at work that incite this fatness epidemic. Obesity is seen as a multi-factorial attributes that arise as a result of multifaceted interaction concerning genetic factor and milieu (Loos and Bouchard, 2008). The increase in the incidence of obesity happened in a little time signifying that ecological and behavioural orientation have significant influence (Agurs-Collins and Bouchard, 2008). G 9 E is widely accepted owing to the huge person's

changes in reactions to the obesogenic situation. Persons with a heritable tendency to grow obesity will display the utmost increase in heaviness, while persons with inherited 'immune' to obesity achieve little weight, if at all (Agurs-Collins and Bouchard, 2008). Similarly, ecological feature's impact behaviour or way of life that later decide vitality consumption or vitality spending (Bouchard, 2008).

However, the variations in person's reactions to deterrence and handling tactics, including undesirable vitality equilibrium owing to augmented energy spending and reduced energy consumption, also appear to be swayed by persons' inherited upbringing (Bray, 2008). Interestingly, many efforts were taken to integrate hereditary and/or genetic factor-setting into obesity mediation and deterrence (Bray, 2008). However, the documented evidence on the topic shows that selected genetic factors are linked to weight reduction after mediation (e.g., way of life change, pharmacological/nutritional mediations, and workout) (summary (Bray, 2008). For example, one polymorphism (rs9939609) in the overweight mass and obesity related genetic factor (FTO) is reported to have a significant influence on the body mass index (BMI), and this is duplicated in another huge example (Loos and Bouchard, 2008).

Persons homozygous with threat A-allele weigh on average around 3–4 kg extra and have a 1.6-fold augmented danger of obesity as related to individuals that did not genetically received a risk allele (Loos and Bouchard, 2008). Besides, it is also recorded that an important FTO genotype 9 physical act association, where the physically quiet homozygous transmitters of the danger A-allele had an upsurge in BMI as related to homozygous transmitters of the T-allele (Andreasen, et al., 2008). Furthermore, other FTO changes indicate a vital relationship with physical activity (Rampersaud, et al, 2008). Nevertheless, concerning this G 9 E with FTO differences and physical activity the results in diverse research are changing and not reliable. This can be clarified amid usage of the diverse dimensions of physical activity (Andreasen and Andersen, 2009).

**Gender:** Despite the general assumption and credit among gender scholars that sex variances and resemblances in behaviour echo both environment and heredity, most hypothetical studies highlight one or some other set of reasons to support their claims. It's worth saying that at the moment only limited concepts give considerable attention to the entangled support of genetic and socio-cultural effects. Naturally, the school's emphasis on a single form of the possible underlying device without charting a strong reason for employing other method. Thus, the research on inherited and hormonal effects gives a little contribution to the environment that promotes this situation or role, while evidence from social creation models frequently do not identify the ecology that is being interpreted. Moreover, the notion that supported the partition of the environment and hereditary sensitiveness is inbuilt in the idea of gender versus sex breakup that began from

the discussions of women's liberation in the 1970s (Unger, 1979). On the other hand, if the characteristics of men folk and womenfolk shoot from entangled environment and hereditary causality, this difference is at best systematically difficult. Therefore the process that perceived variances among womenfolk and the men folk are labeled *sex differences*, irrespective of their sources. The word *sexual category or gender* can best explain as the connotations that were ascribed to males and females by persons and societies (gender stereotypes). However, thinking away from dualistic gender/sex viewpoint, allow psychologists the opportunity to view both society orientation and biology not as distinct effects, but as interrelating mechanisms of hereditary and environmental. One of the main problems in creating particular collaborative concepts is the multifaceted nature of hereditary and environment. Assumed that biology is a multifaceted collection of issues like socio-cultural situations, every collaborative concept of sex variances models simply a quota of possible biosocial collaborations.

To explain such methods, it is imperative to mention various exertions that hypothesize specific collaborations that offer limited charting of entangled nature and nurture. Lickliter and Honeycutt (2003) indicated that genetic factor does not work as condensed elements of inheritance, but as reactionary organizations that are extremely reliant on ecological contribution. Therefore, the inherited sex variance does not just package men and women to behave in diverse manners. According to Fisher (2006), genetic factor does not state behaviors or reasoning processes; they create supervisory features, signaling molecules, receptors, enzymes, etc., that interrelate in exceptionally multifaceted systems, moderated by ecological effects, so as to form and sustain the brain. The effectiveness of menarche in teenage girls demonstrates this type of interdependency philosophy (Allison and Hyde, 2013). Thus, the period of commencement is controlled biologically by the growth of the adrenal glands and the regulatory impacts of the hypothalamic–pituitary–gonadal axis. Therefore, to justify for the different effects on menarche, Ellis (2004); James, Ellis, Schlomer and Garber (2012) suggest an evolutionary-developmental concept that identified the negative significant correlation between father absenteeism, maternal misery and the quality of family relations. They also confirm that poorer household value speed up pubertal growth, gestured by prior menarche and younger sensual action with girls. Due to the related impacts of low socio-economic position, these impacts are expected to be intervened in girls by the discharge of strain hormones (cortisol and epinephrine) via the hypothalamic–pituitary–adrenal axis that also discharges adrenal androgens which enable the early stage of pubertal growth and later arouse the change of the gonads and secondary sexual features.

## CONCLUSION

It worth noting that research on genetic has contributed significantly in recent time about the environment, particular non shared environment and the function of genetic in human behaviour. The general agreement in the field of human development is that any interindividual changes that occur in human behaviour as a result of ecological contact are described by the hereditary control of such outcomes. Therefore, the assertion further that genetic factor–ecological interaction gives a better explanation for persons’ susceptibility and resilience to ecological dangers in the growth and manifestation of behaviour. It is obvious and evidently established in various methodological approaches on nature and nurture interact that human behaviour and personality is highly influenced by genetic and environmental factors.

At the same time reviewed literature on twin, adoption, and family studies offered clear evidence on how heredity and environment contributes to human behaviour, personality, and psychopathology. On the other hand, various reports and analysis of twin, adoption, and family studies raised a large array of topics that support the assertion that bedrock for each human being varied in structure. For example, in some instance, the study shows that genetics seem to dominate; while in other instances, environment elucidated all. However, in most situations evidence shows that the strong interaction between the two factors formed the basis of who we are and how we behave.

Though recent evidence continue to emphasize various problems associated with heredity–environment issues, nevertheless, feasibility of these problems is certain by the ongoing argument about, “Which one?” and “How much?” and this replaced by more fundamental and suitable question, “How?” genetic influences— as well as environmental issues of an organic environment— differ along a “continuum of implicitness.” Therefore it is long –established that the more circuitous their relationship with behaviour, the wider will be the range of disparity of possible effects.

For example, one powerful instance of a range of indirectness is clarified by analyzing metal deficiency that comes as a result of brain damage; moreover other examples illustrate the physical quality linked with social stereotypes. In addition studies show that factors such as deafness, physical diseases, and motor disorders deteriorating at middle points. Furthermore , reports confirmed that ecological factors which directly influence behaviour is well-organized along a continuum of the breadth or stability of effect, as demonstrated by being part of a social class, level of education attained , speech handicap, and acquainted with specific test items. Thus, the evidence shows that recent research studies give clear facts and method that look at the modus operandi of hereditary and

environmental factors. However, the most mentioned among them includes: the explorations of, (a) inherited conditions that explain or trigger differences in behaviour among selectively bred groups of animals; (b) associations between physiological variables and personality differences, particularly in the case of pathological deviations; (c) function of prenatal physiological issues in behaviour development. Moreover, other methods such as, early experience upon final behavioural features; cultural differences in child-rearing tradition to intellectual and emotional development; mechanism of somato psychological relationships; and psychological development of twins from childhood to adulthood, in concert with observations of their social environment all explain heredity and environmental interaction.

In addition, evidence revealed that such approaches are particularly different amid viewing the subjects used, types of psychological functions studied, and particular investigational measures followed. But it is just such heterogeneity of tactic that required by a broad variety of ways in which genetics and environmental factors relate to behaviour development. Besides, evidence shows that genetically and environmentally informative research is greatly required. At this point, it is very imperative to mention that with age hereditary factors become stable, hence enhance rank-order stability of neuroticism and extraversion and to personality development transversely childhood, adolescence, and young adulthood. At the same time, the evidence confirmed that environmental bases add to this rising continuity and declining stability that develop in old age. Therefore, environmental influences become visible and cumulate across the life span and lead to the decline of the inherited contribution to differences in neuroticism and extraversion with age, a guide indicating enduring changes in personality owing to ecological effects.

## REFERENCES

- Agurs-Collins T. and Bouchard C.** (2008). Gene-nutrition and gene- physical activity interactions in the etiology of obesity. *Introduction: Obesity* (Silver Spring) 16(Suppl 3):S2-S4
- Allison, C. M. and Hyde, J. S.** (2013). Early menarche: Confluence of biological and contextual Factors. *Sex Roles*, 68, 55-64.
- Andreasen C. H. and Andersen G.** (2009). Gene-environment interactions and obesity further aspects of genomewide association studies. *Nutrition*, 25:998-1003
- Andreasen C.H, Stender-Petersen K.L, Mogensen M.S, Torekov S.S, Wegner L, Andersen G, et al** (2008). Low physical activity accentuates the effect of the FTO rs9939609 polymorphism on body fat accumulation. *Diabetes*, 57, 95-101
- Banaschewski T., Becker K., Scherag S., Franke B. and Coghill D.** (2009). Molecular genetics in attention-deficit/hyperactivity disorders: An overview. *European Child Adolesc Psychiatry*

- Becker K., El-Faddagh M., Schmidt M. H., Esser G. and Laucht M.** (2008). Interaction of dopamine transporter genotype with prenatal smoke exposure on ADHD symptoms. *J Pediatr*, 152, 263–269
- Berrettini, W. H.** (2000). Are schizophrenic and biological disorders related? A review of Family and molecular studies. *Biological Psychiatry*, 48, 531-538.
- Bjorklund, D. and Pelligrini, A.** (2002). *The origins of human nature*. Washington, DC: American Psychological Association.
- Bouchard C.** (2008). Gene–environment interactions in the etiology of obesity: Defining the Fundamentals. *Obesity (Silver Spring)* 16(Suppl 3): S5–S10
- Brant A. M., Haberstick B.C, Corley R.P, Wadsworth S.J, DeFries J. C. and Hewitt J.K.** (2009). The developmental etiology of high IQ. *Behavioural Genetic*, 39, 393-405,
- Bray M. S.** (2008). Implications of gene–behaviour interactions: prevention and intervention for Obesity. *Obesity (Silver Spring)* 16(Suppl 3): S72–S78
- Bronfenbrenner, U.** (1979). *The Ecology of human development: Experiments by nature and design*. Cambridge, MA: Harvard University Press.
- Bronfenbrenner, U. and Ceci, S. J.** (1994). Nature-nurture reconceptualized in developmental perspective: A bioecological model. *Psychol Rev*, 101(4), 568-586.
- Brookes K. J., Mill J., Guindalini C., Curran S., Xu X., Knight J. et al** (2006). A common haplotype of the dopamine transporter gene associated with attention-deficit/hyperactivity disorder and interacting with maternal use of alcohol during pregnancy. *Arch Gen Psychiatry*, 63, 74–81
- Butcher, L. M., Me burn, E., Liu, L. et al** (2004). Genotyping pooled DNA on micro arrays: a systematic genome screen of thousands of SNPs in large samples to detect QTLs for complex traits. *Behavior Genetics*, 34, 549-555
- Cadore, R. J.** (1986). Adoption studies: historical and methodological critique. *Psychiatr. Dev.* 1, 45–64
- Chiappe, D. and MacDonald, K.** (2005). The evolution of domain-general mechanisms in intelligence and learning. *Journal of General Psychology*, 132 (1), 5–40.
- Coghill, D. and Banaschewski, T.** (2009). The genetics of attention deficit/ hyperactivity disorder. *Expert Rev Neurother*, 9, 1547– 1565
- Corder, E. H., A. M. Saunders, W. J. Strittmatter, D. E. Schmechel, P. C. Gaskell, G.W. Small, A. D. et al** (1993). Gene dose of apolipo protein E type 4 allele and the risk of Alzheimer’s disease in late onset families. *Science* 261:921-23.
- Daniels, D. and Plomin, R.** (1985). Origins of individual differences in infant shyness. *Developmental Psychology*, 21, 118-121.
- Davis, Allison, John Dollard and American Youth Commission** (1946). *Children of Bondage: The Personality Development of Negro Youth in the Urban South*. Washington, D.C.: American Council on Education.
- Deary I. J., Corley J., Gow A. J., Harris S. E., Houlihan L. M., Marioni R. E., et al** (2009). Age-associated cognitive decline. *British Medical Bulletin*, 92, 135-152,
- De Fruyt, F., Bartels, M., Van Leeuwen, K. G., De Clercq, B., Decuyper, M. and Mervielde, I.** (2006). Five types of personality continuity in childhood and adolescence. *Journal of Personality and Social Psychology*, 91, 538–552.
- Edmonds C. J, Isaacs E. B., Visscher P. M., Rogers M., Lanigan, J., Singhal A., et al** (2008). Inspection time and cognitive abilities in twins aged 7 to 17years: development, heritability and genetic covariance. *Intelligence*, 36: 210-225,
- Ellis, B. J.** (2004). Timing of pubertal maturation in girls: An integrated life history approach. *Psychological Bulletin*, 130, 920–958.

- El-Faddagh M., Laucht M., Maras A., Vohringer L. and Schmidt M. H.** (2004). Association of Dopamine D4 receptor (DRD4) gene with attention-deficit/hyperactivity disorder (ADHD) in a high-risk community sample: a longitudinal study from birth to 11 years of Age. *J Neural Transm*, 111:883–889
- Faraone S. V., Perlis R.H., Doyle A.E., Smoller J.W., Goralnick J.J., Holmgren M.A. and Sklar P.** (2005). Molecular genetics of attention deficit/ hyperactivity disorder. *Biol Psychiatry*, 57:1313–1323
- Fisher, S. E.** (2006), Tangled webs: Tracing the connections between genes and cognition. *Cognition*, 101, 270–297
- Fuller, J. L. and W. R. Thompson** (1960). *Behaviour genetics*. New York: Wiley.
- Galton, F.** (1865). Heredity talent and character. *Macmillan's Magazine*, 12:157-66, 318-27.
- Gershon E.S., DeLisi L.E., Hamovit J., Nurnberger J. I. Jr, Maxwell M. E., Schreiber J. et al** (1988). A controlled family study of chronic psychoses. *Arch Gen Psychiatry*, 45:328–336.
- Gillespie, N. A., Evans, D. E., Wright, M. M. and Martin, N. G.** (2004). Genetic simplex modelling of Eysenck's dimensions of personality in a sample of young Australian twins. *Twin Research*, 7, 637–648.
- Haldane, J. B. S.** (1938). *Heredity and politics*. New York: Norton.
- Harris, J. R.** (1998). *The nurture assumption: Why children mm out the way they do*. New York: Free Press.
- Hopwood, C. J., Donnellan, M. B., Krueger, R. F., McGue, M., Iacono, W. G., Blonigen, D. M. and Burt, S. A.** (2011). Genetic and environmental influences on personality trait stability and growth during the transition to adulthood: A three-wave longitudinal study. *Journal of Personality and Social Psychology*, 100, 545–556.
- Hughes C., Happé F., Taylor A., Jaffee S. R., Caspi A. and Moffitt T. E.** (2005). Origins of individual differences in theory of mind: From nature to nurture. *Child Development*, 76, 356-370.
- Jacobs H. J, Williams A. G, Cahill H. and Nathans J.** (2007). Emergence of Novel Colour Vision in Mice Engineered to Express a Human Cone Photopigment. *Science*, 315, 1723-1725
- James, J., Ellis, B. J., Schlomer, G. L. and Garber, J.** (2012). Sex specific pathways to early puberty, sexual debut, and sexual risk taking: Tests of an integrated evolutionary–developmental model. *Developmental Psychology*, 48, 687– 702.
- Jang, K. L.** (2005). *The behavioural genetics of psychopathology: A clinical guide*. Mahwah, NJ: Erlbaum.
- Johnson, W.** (2010). Understanding the genetics of intelligence: Can height help? Can corn oil? *Current Directions in Psychological Science*, 19, 177–182.
- Johnson, A. M., Vernon, P. A. & Feiler, A. R.** (2008). *Behavioural genetic studies of personality: An introduction and review of the results of 50+ years of research*. In G. J. Boyle, G. Matthews & D. H. Saklofske (Eds.), *The SAGE handbook of personality theory and assessment: personality theories and models* (Vol. 1, pp. 145–173). London, England: Sage.
- Johnson D. S., Mortazavi A., Myers R.M. and Wold B.** (2007). Genome-Wide Mapping of in Vivo Protein-DNA Interactions. *Science*, 316: 1497-1502
- Johnson, W., McGue, M. and Krueger, R. F.** (2005). Personality stability in late adulthood: A behavioural genetic analysis. *Journal of Personality*, 73, 523–551.

- Kandler C., Bleidorn W., Riemann R., Angleitner A. and Spinath F. M.** (2012). Life events as environmental states and genetic traits and the role of personality: A longitudinal twin study. *Behavior Genetics*, 42, 57–72.
- Kandler C., Bleidorn W., Riemann R., Spinath F. M., Thiel W. & Angleitner A.** (2010). Sources of cumulative continuity in personality: A longitudinal multiple-rater twin study. *Journal of Personality and Social Psychology*, 98, 995–1008.
- Kendler K.S., McGuire M., Gruenberg A.M., O'Hare A., Spellman M. and Walsh D.** (1993). The Roscommon family study. *Arch Gen Psychiatry*, 50:527–540.
- Kahn R.S., Khoury J., Nichols W.C. and Lanphear B.P.** (2003). Role of dopamine transporter genotype and maternal prenatal smoking in childhood hyperactive-impulsive, inattentive, and oppositional behaviours. *J Pediatr* 143:104–110
- Langley K., Turic D., Rice F., Holmans P., van den Bree M.B., Craddock N., et al** (2008). Testing for gene x environment interaction effects in attention deficit hyperactivity disorder and associated antisocial behaviour. *Am J Med Genet B Neuropsychiatr Genet*, 147B:49–53
- Laucht M., Skowronek M.H., Becker K., Schmidt M.H., Esser G., Schulze T.G. and Rietschel M.** (2007). Interacting effects of the dopamine transporter gene and psychosocial adversity on attention-deficit/hyperactivity disorder symptoms among 15- year-olds from a high-risk community sample. *Arch Gen Psychiatry*, 64:585–590
- Laurin D., Masaki K. H. Foley D. J. White L. R. and Launer L. J.** (2004). Midlife dietary intake of antioxidants and risk of late-life incident dementia: The Honolulu-Asia Aging Study. *American Journal of Epidemiology*, 159:959-67.
- Lerner, R.** (2002). *Concepts and theories of human development*. Mahwah, NJ: Lawrence Erlbaum Associates.
- Lickliter, R. and Honeycutt, H.** (2003). Developmental dynamics: Toward a biologically plausible evolutionary psychology. *Psychological Bulletin*, 129, 819–835.
- Loehlin, J. C. and Martin, N. G.** (2001). Age changes in personality traits and their heritabilities during the adult years: Evidence from Australian Twin Registry samples. *Personality and Individual Differences*, 30, 1147–1160.
- Loehlin J. C., Horn J. M and Willerman L.** (1997): Heredity, environment and IQ in the Texas adoption study. See Sternberg & Grigorenko 1997, pp. 105–25
- Loehlin, J. C., Willerman, L. and Horn, J. M.** (1988). Human behaviour genetics. *Annual Review of Psychology*, 39, 101-134.
- Loevinger, J.** (1943). On the proportional contributions of differences in nature and in nurturing to differences in intelligence. *Psychol. Bull.* 40, 725-756.
- Loos R.J., & Bouchard C.** (2008). FTO: the first gene contributing to common forms of human Obesity. *Obes Rev* 9:246–250
- Maes H.H., Silberg J.L., Neale M.C. and Eaves L.J.** (2007). Genetic and cultural transmission of antisocial behaviour: an extended twin parent model. *Twin Research Human Genetics*, 10: 36-150,
- Maier W., Lichtermann D., Minges J., Hallmayer J., Heun R. & Benkert O.** (1993). Continuity and discontinuity of affective disorders and schizophrenia. Results of a controlled family study. *Arch Gen Psychiatry*, 50, 871– 883.
- McCartney, K., Harris, M. J. and Bernieri, F.** (1990). Growing up and growing apart: A developmental meta-analysis of twin studies. *Psychological Bulletin*, 107, 226–237.



- Merriman, C.** (1924). The intellectual resemblance of twins. *Psychological Monographs*, 33:1-58.
- Miles D.R. and Carey G.** (1997). Genetic and environmental architecture of human aggression. *Journal of Personality and Social Psychology*, 72: 207–217
- Milner, A.** (1951). A study of the relationships between reading readiness in grade one school children and patterns of parent-child interaction. *Child Development*, 22, 95-112.
- Mukamal, K. J., Kuller L. H., Fitzpatrick A. L., Longstreth Jr. W. T., Mittleman M. A. and Siscovick D. S.** (2003). Prospective study of alcohol consumption and risk of dementia in older adults. *JAMA* 289:1405-13.
- Olson J. M., Vernon P. A., Harris J. A., and Jang K. L.** (2001). The heritability of attitudes: A study of twins. *Journal of Personality and Social Psychology*, 80, 845-860.
- Pedersen, N. L. and Reynolds, C. A.** (1998). Stability and change in adult personality: Genetic and environmental component. *European Journal of Personality*, 12, 365–386.
- Pedersen, N. L.** (1993). *Genetic and environmental continuity and change in personality*. In T. J. Bouchard Jr. & P. Propping (Eds.), *Twins as a tool of behavioural genetics* (pp. 147– 162). New York, NY: Wiley.
- Plomin R. and Daniels D.** (1987). Why are children in the same family so different from one another? *Behaviour Brain Sci.* 10:1–60
- Plomin R, DeFries J. C. and McClearn G.E.** (1990b). *Behavioural Genetics: A Primer*. New York: Freeman. 455 pp. 2nd ed.
- Plomin, R., DeFries, J. C., McClearn, G. E. and Rutter, M.** (1997). *Behavioural genetics* (3rd. ed.). New York: Freeman.
- Plomin, R., Defries, J. C., McClearn, G. E. and McGuffin, P.** (2001). *Behavioral genetics* (4th ed.). New York: Worth
- Plomin, R., K. Asbury and J. Dunn** (2001). Why are children in the same family so different? Nonshared environment a decade later. *Canadian Journal of Psychiatry*, 46:225-33.
- Plomin, R., DeFries, J. C., Craig, I. W. and McGuffin, P.** (2003). *Behavioural genetics*. In R. Plomin, J. C. DeFries, I. W. Craig, and P. McGuffin (eds.), *Behavioral genetics in the post genomic era*. Washington, DC: American Psychological Association, (pp. 3–15).
- Podewils L. J., Guallar E., Kuller L. H., Fried L. P., Lopez O. L., Carlson M. and Lyketsos C. G.** (2005). Physical activity, APOE genotype, and dementia risk: Findings from The Cardiovascular Health Cognition Study. *American Journal of Epidemiology*, 161:639-51.
- Popova N. K.** (2008). From gene to aggressive behaviour: the role of brain serotonin. *Neuroscience and Behaviour Physiology*, 38: 471- 475
- Psychogiou L., Daley D.M., Thompson M.J. & Sonuga-Barke E.J.** (2008). Do maternal attention-deficit/hyperactivity disorder symptoms exacerbate or ameliorate the negative effect of child attention-deficit/hyperactivity disorder symptoms on parenting? *Dev Psychopathol* 20:121–137
- Rampersaud E., Mitchell B.D., Pollin T.I., Fu M., Shen H., O’Connell J.R., et al** (2008). Physical activity and the Association of common FTO gene variants with body mass index and obesity. *Arch Intern Med* 168:1791–1797

- Read S., Vogler G. P., Pedersen N. L. and Johansson B.** (2006). Stability and change in genetic and environmental components of personality in old age. *Personality and Individual Differences*, 40, 1637–1647
- Rhee S. H & Waldman I. D.** (2002). Genetic and environmental influences on antisocial behaviour: a meta-analysis of twin and adoption studies. *Psychology Bulletin* 128: 490–529
- Roberts, B. W. and Wood, D.** (2006). *Personality development in the context of the Neo-Socioanalytic Model of personality*. In D. Mroczek & T. Little (Eds.), *Handbook of personality development* (pp. 11–39). Mahwah, NJ: Erlbaum.
- Roberts, B. W., Wood, D. and Caspi, A.** (2008). *The development of personality traits in adulthood*. In O. P. John, R. W. Robins, & L. A. Pervin (Eds.), *Handbook of personality: Theory and research* (3rd ed., pp. 375–398). New York, NY: Guilford.
- Roth, W. E. and Finley, G. E.** (1998). Adoption and antisocial personality: Environmental factors associated with antisocial outcomes. *Child and Adolescent Social Work Journal*, 15, 133–150.
- Scarr S. and Weinberg R. A.** (1978). The influence of family background on intellectual attainment. *Am. Soc. Rev.* 43:674–92
- Schwesinger, G. C.** (1933). *Heredity and environment*. New York: Macmillan.
- Sonuga-Barke E.J. and Rubia K.** (2008). Inattentive/overactive children with histories of profound institutional deprivation compared with standard ADHD cases: A brief report. *Child Care Health Dev* 34:596–602
- Spengler, M., Gottschling, J. and Spinath, F. M.** (2012). Personality in childhood: A longitudinal behaviour genetic approach. *Personality and Individual Differences*, 53, 411–416.
- Spinath B., Spinath F.M., Harlaar N. and Plomin R.** (2006). Predicting school achievement from cognitive ability, self perceived ability, and intrinsic value. *Intelligence*, 34: 363–374,
- Stevens S.E., Kumsta R., Kreppner J.M., Brookes K.J., Rutter M. and Sonuga-Barke E.J.** (2009). Dopamine transporter gene polymorphism moderates the effects of severe deprivation on ADHD symptoms: developmental continuities in gene–environment interplay. *Am J Med Genet B Neuropsychiatr Genet* 150B:753–761
- Teasdale T. W. and Owen D. R.** (1984). Heritability and familial environment in intelligence and educational level—a sibling study. *Nature*, 309:620–22
- Thapar A., Langley K., Asherson P. and Gill M.** (2007). Gene–environment interplay in attention-deficit hyperactivity disorder and the importance of a developmental perspective. *Br J Psychiatry* 190:1–3
- Thapar A., Langley K., Fowler T., Rice F., Turic D., Whittinger N., et al** (2005). Catechol O-methyltransferase gene variant and birth Weight predict early-onset antisocial behaviour in children with attention- deficit/hyperactivity disorder. *Arch Gen Psychiatry* 62: 1275–1278
- Theis, S. V. S.** (1924). *How foster children turn out* (Vols. Publication No.165). New York: State Charities Aid Association.
- Turkheimer, E. and Waldron, M.** (2000). Nonshared environment: A theoretical, methodological, and quantitative review. *Psychological Bulletin*, 126, 78–108.
- Unger, R. K.** (1979). Toward a redefinition of sex and gender. *American Psychologist*, 34, 1085–1094.

- Van den Oord, E. J. C. G., Boomsma, D. I. and Verhulst, F. C.** (2000). A study of genetic and environmental effects on the co-occurrence of problem behaviours in three-year-old twins. *Journal of Abnormal Psychology*, 109, 360-372.
- Viken, R. J., Rose, R. J., Kaprio, J. and Koskenvuo, M.** (1994). A developmental genetic analysis of adult personality: Extraversion and neuroticism from 18 to 59 years of age. *Journal of Personality and Social Psychology*, 66, 722-730.
- Walker, S. O. and Plomin, R.** (2005). The nature-nurture question: Teachers' perceptions of how genes and the environment influence educationally relevant behaviour. *Educational Psychology: An International Journal of Experimental Educational Psychology*, 25:5, 509-516.
- Watson, J. B.** (1930). *Behaviourism*. New York: Norton.
- Whiting, J. W. M. and Child, I. L.** (1953). *Child Training and Personality*. Yale University Press, New Haven, CT.
- Whiting, J. W. M., et al.** (1954). *Field guide for a study of socialization in five societies*. Cambridge, Mass.: Harvard University (mimeo.).
- Williams, J. R. and Scott, R. B.** (1953). Growth and development of Negro infants IV. Motor development and its relationship to child rearing practices in two groups of Negro infants. *Child Development*, 24, 103-121.
- Woodworth, R. S.** (1941). Heredity and environment: A critical survey of recently published material on twins and foster children. *Social Science Research Council Bulletin*, No. 47.