

# [Is there a genetic basis for criminality criminology essay](https://assignbuster.com/is-there-a-genetic-basis-for-criminality-criminology-essay/)

“ We are what we repeatedly do” is a quote by one of the greatest revolutionary minds of all time, Aristotle. Though it’s millennia since he lived, his logic isn’t too outdated, if you frequently donate to charity, then you’re benevolent. If you repeatedly break the law, you’re a criminal. The modern question is where these behaviours stem from; your genetics or your environment? There is an increasing amount of empirical evidence on the contribution of genetic factors to individual differences in criminal and antisocial behaviours (Turkheimer et al. 1995). A recent meta-analysis of over 400 studies suggests that about 41% of human behaviour is genetically influenced with greatest heritability for antisocial behaviour and smoking (Malouff et al. 2008). It is a topic relevant both in a scientific and a social context. Should the discovery of a genetic basis for antisocial behaviour change the way it is treated and viewed by society? Or lead to a change in its definition, if you’re a criminal due to a genetic predisposition, is it different to part-taking in criminal behaviour without a found genetic component?

It is necessary to begin by asking, what is criminal and antisocial behaviour? It isn’t simply disobedience, some forms of disobedience are acts designed to change the law or common practice such as the suffragette movement (CIBA Foundation Symposium. 1996). Other acts break the law, but are entirely acceptable within subgroups of society such as the recreational taking of cannabis. Some behaviour also breaks the law purely because the offender is below a legally defined age such as with alcohol and sex. One definition of antisocial behaviour is behaviour that lacks consideration for others and that may cause damage to society, whether intentionally or through negligence (Berger, 2003). Criminality falls under this definition of antisocial behaviour. This definition will be adopted here but it must be noted that antisocial behaviour cannot be restricted to that which is disapproved in all societies. In accord with the opening quote, repetition is important. In Farringtons 1995 study of males from inner-city London, 96% admitted to having committed one or more of ten common crimes such as theft, violence, vandalism. Yet, we don’t view 96% of the human population as criminals.

Over the past decades, there has been a paradigm shift in the way human behaviour is approached. The social learning model has been majorly replaced by a balanced view emphasising the importance of genetic and environmental factors on human behaviour. There is now a wealth of evidence supporting that both genetics and environmental factors play an important role in accounting for individual differences in antisocial and criminal behaviour.

There are two basic approaches to researching behavioural genetics. The first is classical genetics designs; a traditional approach involving no DNA examination. It analyses individual differences in a given trait by examining patterns of resemblance among individuals who are related genetically, environmentally or both (Baker et al, 2006), by studying twins, nuclear families and adoptive families. Classical approaches broadly determine whether genes are important and estimate the extent of the genetic influence. The second method studies Quantitative Trait Loci (QTL), genes which exert small but significant influence on complex traits. A QTL approach is considered more molecular than the classical designs because it narrowly specifies DNA sequences that increase risk for antisocial behaviour. This molecular aspect is a much more recent and developing approach than the classical studies, however, it is an important technique and studies are emerging indicating specific gene associations.

To date, the classical approach has provided the scientific community with abundant evidence for both genetic and environmental influences on antisocial and criminal behaviour. The studies vary in the type of antisocial behaviour (juvenile delinquency, aggression, adult criminal behaviour etc), the definition of antisocial behaviour and the way its measured (official and school records, behavioural ratings by parents or teachers, self-reporting). It has been studied across the human lifespan and males are more extensively studied than females. Due to the inconsistency between each study, reviews of these studies are important to draw well-informed conclusions. A review by Rhee and Waldman in 2002 looked at 51 distinct studies which focused on some aspect of antisocial behaviour. The results of the studies were combined to estimate the relative effect of genetic and environmental influences. There a significant effects of additive genetic influence (0. 32), non-additive genetic influences (0. 09), shared (0. 19) and non-shared environment (0. 43), which clearly demonstrates the effect of heredity and environment on antisocial behaviour. The review also found that non-additive genetic effects appear most strongly for criminality compared to other forms of antisocial behaviour, and that there was a strong and decreasing importance of shared environment factors from childhood to adulthood. Genetics seems to be more strongly correlated with criminality and antisocial behaviour in adulthood than in childhood or adolescence.

A wide range of behaviours are considered antisocial, however, it may be beneficial, especially in a legal context, to study illegal, criminal behaviours. Several large scale twin studies have been conducted in various countries such as USA (Cadoret et al. 1995) and Denmark (Hutchings & Mednicks. 1975). They found that there is higher agreement between monozygotic (MZ) twins, who are genetically identical, than dizygotic (DZ) twins, who on average share 50% of their genes, for property crimes such as theft and vandalism (Cloninger & Gottesman. 2000). This indicates a genetic component which is further backed up by evidence that property crime convictions among adopted individuals significantly increased when the biological parent was convicted but showed little or no increase when adopted children were raised by adoptive parents with such convictions (Baker et al. 1989). In the case of committing violent crimes, there seems to be no increased risk when studied as a function of adoptive or biological parents.

In contrast to the large genetic influence on adult criminality, childhood behaviours such as minor rule breaking, theft, truancy, vandalism, can apparently be explained by environmental factors. Small genetic influence was only found in theft and minor rule breaking (Baker et al. 2006).

Antisocial behaviour and aggression play key roles in the diagnosis of three mental disorders. Antisocial personality disorder in adults often involves aggressive, impulsive, and irresponsible behaviour. Significant genetic influences have been consistently found in twin samples and adoption studies. Conduct disorder is a childhood behavioural problem indicated by aggression, destructive behaviours, theft, dishonesty and truancy. It is thought to be a severe and clinical form of antisocial behaviour. It is associated with negative outcomes such as drug abuse, depression and, as adults, antisocial personality disorder. Multiple studies have shown that children with conduct disorder have a largely increased risk of developing antisocial personality disorder as an adult (Loeber. 1991). A history of antisocial personality disorder in a parent is the strongest predictor of persistence of conduct disorder from childhood into adolescence (Lahey et al. 2000). Like in antisocial personality disorder, significant genetic effects have been found in twin samples and adoption studies (Eaves et al. 1997). Recent studies have shown that conduct disorder is significantly heritable, with estimates ranging from 27% to 78% (Baker et al. 2006). It seems there’s a wealth of evidence indicating a strong link between genetics and conduct disorder and antisocial personality disorder.

The third mental disorder is oppositional defiant disorder which is characterised by a repeated pattern of negative, aggressive and defiant behaviour in children. Twin studies have also investigated the heritability of oppositional defiant disorder. Several found significant genetic influences in oppositional defiant disorder symptoms, with heritability estimates ranging from 14% to 65% (Eaves et al. 1997).

All these studies provide direct evidence for genetic influences on antisocial behaviour. However, there are numerous studies that provide indirect evidence by examining the genetics of traits that correlate with antisocial behaviour. Understanding the genetic influences on correlated traits can give us important information on the genetic mechanisms underlying antisocial behaviour. One of the most relevant traits is impulsivity. Individuals are differently susceptible to antisocial behaviour due to variation in traits such as impulsivity which are heritable according to data from large twin and adoption studies. Heritability estimates range from 20% to 72% (Coccaro et al. 1993). It is thought that genes modulate behaviours such as impulsivity, which can lead to other disorders such conduct disorders, antisocial personality disorder and ADHD. ADHD in childhood has been linked to antisocial behaviour in two ways. Research has shown that, children with ADHD are more likely than those without it to show antisocial behaviour as an adult (Hetchman et al. 1984). It appears that children who have symptoms of ADHD have a more persistent form of conduct disorder. Other studies have shown that youths exhibiting both ADHD and antisocial behaviour manifest severe forms of antisocial behaviour such as extreme physical aggression. An adoption study has shown a high genetic component for attention problems which are a key diagnostic feature of ADHD (Van Den Oord et al. 1994) and heritability estimates range from 39% to 91% (Thapar et al. 1999).

As shown, there is an abundance of classical genetic studies showing the importance of genetic predispositions as well as environmental factors. However, this research contributes little to the exact biological mechanisms underlying the genetic effects. They represent “ black boxes” in our understanding of antisocial behaviour. Some recent approaches have been adopted to tackle this gap in our knowledge. One such approach is “ a measured risk factor” which investigates traits and behaviours known to correlate with the risk of antisocial behaviour. Multivariate genetic models are used to explain sources of genetic covariance underlying the correlation between a trait and antisocial behaviour. This may be applied to the traits discussed earlier such as impulsivity, attention deficit but also to biological risk factors such as hormones, neurotransmitters etc. However, little research using this method has been done to date.

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A second approach is by using QTL designs which identifies specific genes as having associations with or functional significance in antisocial behaviour. It could be called a measured gene approach. Knowing the genes involved in behavioural disorders such as those discussed, including antisocial behaviour, brings with it a better understanding of the genetic mechanisms. Identifying particular genes associated with different disorders that regulate, say, neurotransmitter activity may allow adjustment of their levels by pharmacological methods. Genes associated with neurotransmitters have been identified for antisocial behaviour, in particular serotonin (5-hydroxytryptamine or 5-HT). Low levels of serotonin have been shown to increase impulsivity and repress sensible behaviour, both of which are linked to antisocial behaviour (Moore et al. 2002). A serotonergic gene with a relationship to antisocial behaviour is the gene coding for the serotonin receptor. A variant polymorphism of this gene, Serotonic2A Receptor gene (HTR2A), was found to be associated with drug abuse, shoplifting, hostility, vandalism and rape (Comings. 2000). Another serotonergic gene associated with antisocial behaviour is the HTR1DA gene which is suspected to play a role in serotonin metabolism. The C variant of this gene was found to be significantly associated with antisocial personality disorder in adults and conduct disorder in children (Comings. 2000).

In particular, studies on aggression, a form of antisocial behaviour, have made advances in this field. A detailed analysis of 24 studies of the genetics of aggression indicated that heritability accounts for around 50% of the variance (Rhee & Waldman, 2002). It was also noted that heritability for aggression was more important in adulthood than childhood and was higher in males than in females. Studies have shown that the Y chromosome is important for aggression in males, through its role in male determination but also through other Y-linked loci. When the male determining gene (Sry) was deleted in mice, it was shown that aggression was independent of the Sry locus implying other genes on the X and Y chromosomes are involved (Gatewood et al. 2006). One candidate gene related to sexual differences is the gene coding for the androgen receptor (AR), two studies have found a significant link for males with shorter trinucleotide repeat motif with verbal aggression (Jonsson at al. 2001) or violent criminal activity such as murder and rape (Rajender et al. 2008).

A wealth of research indicates the key role of serotonin in aggression. Monoamine oxidase A (MAOA) is an enzyme which oxidases biogenic amines such as serotonin (5-HT), noradrenalin and adrenalin. Null mutations in the MAOA locus which cause an imbalance in serotonin metabolism are correlated with aggression in humans (Brunner et al. 1993). Later studies have centred on detecting behavioural associations with VNTR variants in MAOA. Significant associations have been found between longer base pair alleles of the MAOA gene and behaviour disorders including ADHD, conduct disorder and substance abuse (Comings. 2000). Other genes thought to be involved in the association between aggression and serotonin include the genes coding for tryptophan hydroxylase enzyme, which catalyses the rate limiting step in serotonin production and the genes coding for the serotonin receptors. There are further strong genetic associations between stress and aggression with candidate genes including those coding for dopamine-beta-hydroxylase and adrenaline receptors.

Genetic influences on behaviour are not simple to understand. Gene expression for antisocial behaviour frequently depends on a number of other factors such as the effects of other genes or environmental influences. Classical genetic studies indicate strong interactions between genes and environment. The conclusion from candidate gene studies is that there are few, if any loci with a large effect; it is becoming increasingly obvious that the impact of genes is multifactorial; including other genes and the environment (Craig & Halton. 2009). Studies of antisocial behaviour now require a comprehensive view of biological and social risk factors as well as their interaction. The extent to which genetic influence varies as a function of environmental effects is known as a gene X environment (GxE) interaction. A significant GxE interaction has been found in all major adoption studies of criminal convictions, such that the genetic predispositions, indicated by biological-parent antisocial behaviour, presents the greatest risk to the adopted offspring in the presence of adverse environmental conditions, indicated by adoptive-parent antisocial behaviour (Baker et al. 2006). Negative environmental factors due to being raised by antisocial parents may have the largest impact on individuals who are genetically predisposed towards antisocial behaviour (Cloninger & Gottesman. 2000).

GxE is also seen at the specific gene level. A deleterious MAOB gene linked to aggression in human and mice has been demonstrated to have the greatest influence on antisocial behaviour in individuals who have experienced severe mistreatment during childhood (Caspi et al. 2002). The discovery of GxE interactions in antisocial behaviour highlights the sheer complexity of the gene-behaviour relationship.

Until the past decade, there was very little research done to investigate how different risk factors interact in predisposing individuals to antisocial behaviour. Recently, there is a renewed interest in biosocial interaction effects and a number of possible interactive processes for biological and social risk factors have been outlined in the Biosocial Model. It acknowledges that biological risk factors may directly result in antisocial behaviour independent of social risk factors and vice-versa; this is the assumption of the majority of research to date. The essence of the biosocial model is that biological and social risk factors interact to produce antisocial behaviour. It also states that protective factors can disrupt all three pathways; biological, social and biosocial. A key understanding in the model is that there are antisocial subtypes and risk factors give rise to both a general predisposition to antisocial behaviour and different antisocial subtypes with potentially different etiological bases, such as psychopathy or violence.

Going back to the original question, is there a genetic basis for criminality and antisocial behaviour? The answer would have to be yes; there is a genetic basis for criminality and antisocial behaviour, and for most human behaviours. The evidence of its existence is out there in overwhelming quantity and multiple forms.

Taking the answer as yes, there is a magnitude of other things that must also be understood. Attempting to understand genetic involvement in any behaviour is complicated, no gene has a monopoly control over any behaviour and its workings can only be understood in the light of interaction with other genes, environmental influences and protective factors. Future genetic studies of human behaviour require a comprehensive view of the interaction of biological and social risk factors; the adoption of an interactive predisposition approach.

“ In behavioural sciences, the three essential guidelines are probability, probability, and probability.” this is an essential quote by a leading psychologist Sandra Scarr. This is the second point of understanding; no aspect of human behaviour is so black and white that the presence of a gene means the behaviour is inevitable. Genes work in a probabilistic manner. There are no genes for long legs, but “ all things being equal”, having gene a will give you an increased likelihood of having longer legs. In “ all things being equal”, the all things are the genetic environment; the influence of other genes, the social environment; shared and unshared influences, and the interaction of these things. When these are equal in two individuals, the possession of gene a in individual 1 makes it more probable that his legs are longer than the legs of individual 2 who possesses gene b. In the same way, just because an individual has a shorter trinucleotide repeat in their androgen receptor does not mean they deterministically will murder and rape; it just increases the probability of them doing so. This probabilistic view, as opposed to a deterministic view, of genetic influence is essential to the appropriate social understanding of genetic predisposition to antisocial behaviour and criminality.

This paradigm shift in how human behaviour is viewed, away from a social learning model and towards an interactive predisposition, will be reflected in society. One implication of a genetically based disposition to antisocial behaviour is the possible absolution of an individual’s responsibility for such behaviour. As much as the advances are huge advances in the field of genetics in relation to antisocial behaviour, the broader picture is not complete. In most cases, genetic predisposition alone is currently too little understood to be a tool in so serious a matter as criminal blame. It is evident that there is a large genetic influence on these behaviours but only a few genes have been pinpointed with confidence in their effects. A more complete understanding of environmental influences is also needed, if genetic predisposition can be used to free or reduce a person of guilt, then so can early environment which also predisposes an individual to antisocial behaviours. This gets into an area where influences may be harder to prove. Also, it has been highlighted that the relationship between genotype and phenotype for antisocial behaviour is not linear, there are other influences such epistasis; this must be researched more fully so it is clearly understood before appropriate changes in society and the legal system can come about.

In the future, when genetic predisposition is more adequately understood behavioural genetics could potentially be used in almost every aspect of the criminal justice system. Genetic behavioural forensic profiling might be used in law enforcement to predict the defendant’s personality and behavioural traits. Behavioural genetic information could be presented at a bail hearing by the prosecutor advising no bail to be granted due to the genetic predisposition to impulsivity or violence. It may also be used to support an insanity defence or as an independent basis for acquittal or used at parole hearings, to deny parole or as a basis for release (Rothstein. 2005).

However, it is possible, even with this lack of understanding, to apply genetic predisposition to the legal system in personality disorders. As discussed, individuals who repeatedly show antisocial behaviour have a genetic predisposition. Personality disorders are often characterized by a manifest of persistent antisocial behaviour. Should a personality disorder be permitted to nullify or reduce responsibility for a criminal act?

The modern answer seems to be yes. Apart from genetic predisposition, the main reasoning is that personality disorders are highly similar to other disorders that remain eligible for consideration in an insanity defence and a rule excluding them from forming the basis of a not guilty defence is scientifically indefensible and jurisprudentially hazardous (Kinscherff. 2010). This argument is the groundwork for the foundation of a criminal responsibility defence. A verdict of “ not guilty by reason of insanity” does not mean the individual returns to the community. They are usually committed to a state psychiatric facility for a time, often longer than they would have spent in prison and this must be applied to a not guilty plea on the grounds of a personality disorder. If a defendant successfully uses impairments arising from a personality disorder to nullify criminal responsibility then the defendant should be assumed dangerous and to protect public safety, they should be subjected to intervention to achieve the goal of incapacitation, potentially with lifetime supervision. Further research is needed as currently there is no demonstrated effective intervention for some personality disorders, especially antisocial personality disorder. The alternate proposition is that a personality disorder should not nullify responsibility for a criminal act. This implies that there is some conceptual “ bright line” distinguishing personality disorders and all other mental disorders in terms of genetic heritability, etiology, neuro-cognitive functioning or other relevant dimension; however, this implication is not supported by science (Kinscherff. 2010).

Another implication of a genetically based disposition to antisocial and criminal behaviour is the treatment of these people in society. In the late nineteenth and early twentieth centuries, researchers believed that genes were fully responsible for criminal activity and that criminals could be identified by their physiological features. This information, combined with ideas of a eugenics movement led to acts of sterilisation to rid society of “ criminals, idiots, imbeciles, and rapists” (Joseph, 2001). This belief induced a period of widespread inhumane treatment in Western Europe and many USA states. Genetic research into human behaviour was also misused to support Nazi claims of racial superiority, which directly affected millions in World War 2 (Rothstein. 1999). To prevent this, society must be educated in the probabilistic view of genetic influence, along with the importance of environmental factors and their interaction. This is where the media plays a crucial role; the genetics of human behaviour have always been popular with the public. However, the descriptions of scientific research in the popular media are not always controlled. There is an increasing number of reports advocating that violence, impulsivity, aggression and other behaviours are ‘ hard-wired’ rather than caused by many factors (Clark et al. 2000). Misunderstanding and over-estimation of the role of genes in human behaviour is of great concern. It is to be expected that individuals and institutions may act on these misconceptions. Many entities with financial interest in the behaviour of particular individuals might seek to use genetic information to predict behaviour; insurers might be liable for injuries caused by an impulsive or aggressive individual. Employers may be interested in a potential employee’s behavioural disposition. The wrongful use of behavioural genetics in employment could lead to the exclusion of individuals from important opportunities. Laws may be needed to prevent generic discrimination in employment and to protect the confidentiality of genetic information. Behavioural genetic information is an ethical and societal issue; it brings to light individual and social ideas of equality of opportunity, discrimination and personal responsibility.

A final implication is the great need to try and identify those individuals, especially children, who may become susceptible to certain disorders or personality traits that can lead into antisocial, delinquent, or criminal behavior, in confidence. Society needs to focus on the treatment and rehabilitation of those individuals in need. Certain educational, environment enrichment programs have been shown to have a lasting effect on children if given by a certain age (Raine et al. 2003). If more of these programs could be developed, society could help prevent the future antisocial or criminal behavior of children, minimizing genetic factors in the role of human behaviour. One misapplication of the need to identify predisposed individuals is in fetal screening. Is the utilization of genetic information to determine who should and should not be born a reversion to the negative eugenic practices of the early twentieth century (Paul. 1994)? Prospective parents might test embryos for a genetic marker before implantation to avoid giving birth to a child with potential for criminality. The use of genetic information in order to select against potential humans with undesirable traits certainly smacks of eugenic motivations (Duster. 1990). Society needs a deep understanding of interactive predisposition before it is capable of correctly dealing with important ethical debates such as this one.

In light of recent research, it is impossible to deny the role of genetics in behaviour. As the topic catches the media’s eye more and more, the idea of behavioural genetic determinism becomes ever more popular and widespread, there is a potential for misuse and misunderstanding of behavioural genetic information. As its application in everyday life is likely to increase, this misunderstanding is a critical societal challenge which needs to be tackled immediately. Unless this concern is addressed, there is a risk that the legal and commercial applications of behavioural genetics will outpace the science to our detriment. It must be widely understood that having a genetic predisposition for criminal behavior does not determine the actions of an individual, but if they are exposed to the right environment, then their chances are greater for engaging in criminal or antisocial behavior. As much as there is a need for further genetic research, social science needs to research the effect of genetic explanations for behaviour on individuals and society.