Chapter 1 : Love of Shopping is Not a Gene : Dagg :

One can read not only about the "shopping gene," but also about the "reading gene," the "humility gene" and the "coaching gene." Pop science, fostered by Darwinian psychology, run amok.

The first instance of debate occurred between Ronald Fisher and Lancelot Hogben. Fisher sought to eliminate interaction from statistical studies as it was a phenomenon that could be removed using a variation in scale. Hogben believed that the interaction should be investigated instead of eliminated as it provided information on the causation of certain elements of development. A similar argument faced multiple scientists in the s. Lewontin and Layzer argued that in order to conclude causal mechanisms, the geneâ€”environment interaction could not be ignored in the context of the study while Jensen defended that interaction was purely a statistical phenomenon and not related to development. Rothman supported the use of a statistical definition for interaction while researchers Kupper and Hogan believed the definition and existence of interaction was dependent on the model being used. In contrast to previous debates, Moffitt and Caspi were now using the statistical analysis to prove that interaction existed and could be used to uncover the mechanisms of a vulnerability trait. Contention came from Zammit, Owen and Lewis who reiterated the concerns of Fisher in that the statistical effect was not related to the developmental process and would not be replicable with a difference of scale. Tabery [11] has labeled them biometric and developmental interaction, while Sesardic [12] uses the terms statistical and commonsense interaction. The biometric or statistical conception has its origins in research programs that seek to measure the relative proportions of genetic and environmental contributions to phenotypic variation within populations. Biometric geneâ€”environment interaction has particular currency in population genetics and behavioral genetics. Biometric interaction is relevant in the context of research on individual differences rather than in the context of the development of a particular organism. Developmental interaction is not seen merely as a statistical phenomenon. Model A describes a genotype that increases the level of expression of a risk factor but does not cause the disease itself. For example, the PKU gene results in higher levels of phenylalanine than normal which in turn causes mental retardation. The risk factor in Model B in contrast has a direct effect on disease susceptibility which is amplified by the genetic susceptibility. Model C depicts the inverse, where the genetic susceptibility directly effects disease while the risk factor amplifies this effect. In each independent situation, the factor directly effecting the disease can cause disease by itself. Model D differs as neither factor in this situation can effect disease risk, however, when both genetic susceptibility and risk factor are present the risk is increased. For example, the G6PD deficiency gene when combined with fava bean consumption results in hemolytic anemia. This disease does not arise in individuals that eat fava beans and lack G6PD deficiency nor in G6PD-deficient people who do not eat fava beans. Lastly, Model E depicts a scenario where the environmental risk factor and genetic susceptibility can individually both influence disease risk. When combined, however, the effect on disease risk differs. The models are limited by the fact that the variables are binary and so do not consider polygenic or continuous scale variable scenarios. Additionally, adopted individuals are compared to their adoptive family due to the difference in genes but shared environment. For example, an adoption study showed that Swedish men with disadvantaged adoptive environments and a genetic predisposition were more likely to abuse alcohol. Later studies leverage biometrical modelling techniques to include the comparisons of dizygotic twins to ultimately determine the different levels of gene expression in different environments. For example, a Danish study on high-risk children with schizophrenic mothers depicted that children without a stable caregiver were associated with an increased risk of schizophrenia. Candidate studies such as these require strong biological hypotheses which are currently difficult to select given the little understanding of biological mechanisms that lead to higher risk. These studies are also often difficult to replicate commonly due to small sample sizes which typically results in disputed results. The polygenic nature of complex phenotypes suggests single candidate studies could be ineffective in determining the various smaller scale effects from the large number of influencing gene variants. A polygenic score is generated using the alleles associated with a trait and their respective weights based on effect and examined in combination with environmental exposure. Though this method of research is still

early, it is consistent with psychiatric disorders. As a result of the overlap of endophenotypes amongst disorders this suggests that the outcomes of geneâ€"environment interactions are applicable across various diagnoses. An effective approach to this all-encompassing study occurs in two-steps where the genome is first filtered using gene-level tests and pathway based gene set analyses. Specifically complex traits studies have come under scrutiny for producing results that cannot be replicated. For example, studies of the 5-HTTLPR gene and stress resulting in modified risk of depression have had conflicting results. Studies are suggested to produce inaccurate results due to the investigation of multiple phenotypes and environmental factors in individual experiments. There is disagreement on which scale should be used. Under these analyses, if the combined variables fit either model then there is no interaction. The combined effects must either be greater for synergistic or less than for an antagonistic outcome. The additive model measures risk differences while the multiplicative model uses ratios to measure effects. The additive model has been suggested to be a better fit for predicting disease risk in a population while a multiplicative model is more appropriate for disease etiology. For example, a child with a poor quality environment would be more sensitive to a poor environment as an adult which ultimately led to higher psychological distress scores. This depicts a three way interaction Gene x Environment x Environment. The same study suggests taking a life course approach to determining genetic sensitivity to environmental influences within the scope of mental illnesses. Some people carry genetic factors that confer susceptibility or resistance to a certain disorder in a particular environment. The interaction between the genetic factors and environmental stimulus is what results in the disease phenotype. This would allow doctors to more precisely select a certain drug and dosage to achieve therapeutic response in a patient while minimizing side effects and adverse drug reactions. The diet, for example, is modifiable and has significant impact on a host of cardiometabolic diseases, including cardiovascular disease, coronary artery disease, coronary heart disease, type 2 diabetes , hypertension , stroke , myocardial infarction , and non-alcoholic fatty liver disease. Geneâ€"environment interactions can modulate the adverse effects of an allele that confers increased risk of disease, or can exacerbate the genotypeâ€"phenotype relationship and increase risk, in a manner often referred to as nutrigenetics. A classic example of geneâ€"environment interaction was performed on Drosophila by Gupta and Lewontin in In their experiment they demonstrated that the mean bristle number on Drosophila could vary with changing temperatures. As seen in the graph to the right, different genotypes reacted differently to the changing environment. Each line represents a given genotype, and the slope of the line reflects the changing phenotype bristle number with changing temperature. Some individuals had an increase in bristle number with increasing temperature while others had a sharp decrease in bristle number with increasing temperature. This showed that the norms of reaction were not parallel for these flies, proving that geneâ€"environment interactions exist. Seven genetically distinct yarrow plants were collected and three cuttings taken from each plant. One cutting of each genotype was planted at low, medium, and high elevations, respectively. When the plants matured, no one genotype grew best at all altitudes, and at each altitude the seven genotypes fared differently. For example, one genotype grew the tallest at the medium elevation but attained only middling height at the other two elevations. The best growers at low and high elevation grew poorly at medium elevation. The medium altitude produced the worst overall results, but still yielded one tall and two medium-tall samples. Altitude had an effect on each genotype, but not to the same degree nor in the same way. A group of genotypes requires similar growing degree-day GDD to flower across all environments, while another group of genotypes need less GDD in certain environments, but higher GDD in different environments to flower. The complex flowering time patterns is attributed to the interaction of major flowering time genes Ma1, [27] Ma6, [28] FT, ELF3 and an explicit environmental factor, photothermal time PTT capturing the interaction between temperature and photoperiod. In the absence of this enzyme, an amino acid known as phenylalanine does not get converted into the next amino acid in a biochemical pathway , and therefore too much phenylalanine passes into the blood and other tissues. This disturbs brain development leading to mental retardation and other problems. PKU affects approximately 1 out of every 15, infants in the U. However, most affected infants do not grow up impaired because of a standard screening program used in the U. Newborns found to have high levels of phenylalanine in their blood can be put on a special, phenylalanine-free diet. If they are put on this diet right away and stay on it, these children

avoid the severe effects of PKU. Low MAOA activity is a significant risk factor for aggressive and antisocial behavior in adults who report victimization as children. Persons who were abused as children but have a genotype conferring high levels of MAOA expression are less likely to develop symptoms of antisocial behavior. Egg Development Time by Temperature Contrary to the aforementioned examples, length of egg development in Drosophila as a function of temperature demonstrates the lack of geneâ€"environment interactions. The attached graph shows parallel reaction norms for a variety of individual Drosophila flies, showing that there is not a geneâ€"environment interaction present between the two variables. In other words, each genotype responds similarly to the changing environment producing similar phenotypes. For all individual genotypes, average egg development time decreases with increasing temperature. The environment is influencing each of the genotypes in the same predictable manner.

Chapter 2 : I Have Never Been to Ireland, But I Do Love a Limerick

Anne Innis Dagg's "Love of Shopping" is Not a Gene is a scathing, entertaining and extremely accessible geneticist's critique of "Darwinian Psychology" -- that is, the "science" of ascribing human.

Share via Email Genes are far from the whole story. But the scientific evidence for a biological basis for sexual orientation seems strong. Scientific evidence says otherwise. It points strongly to a biological origin for our sexualities. I would argue that understanding our fundamental biological nature should make us more vigorous in promoting LGB rights. Three gene finding studies showed that gay brothers share genetic markers on the X chromosome; the most recent study also found shared markers on chromosome 8. This latest research overcomes the problems of three prior studies which did not find the same results. Panel Discussion - Born that way: Is there a gay gene and should it matter? Read more Gene finding efforts have issues, as Copland argues, but these are technical and not catastrophic errors in the science. But each of these genes has a small effect on the trait so do not reach traditional levels of statistical significance. In other words, lots of genes which do influence sexual orientation may fall under the radar. But scientific techniques will eventually catch up. In fact there are more pressing problems that I would like to see addressed, such as the inadequate research on female sexuality. Genes are far from the whole story. Sex hormones in prenatal life play a role. For example, girls born with congenital adrenal hyperplasia CAH , which results in naturally increased levels of male sex hormones, show relatively high rates of same-sex attractions as adults. Further evidence comes from genetic males who, through accidents, or being born without penises , were subjected to sex change and raised as girls. As adults these men are typically attracted to women. The fact that you cannot make a genetic male sexually attracted to another male by raising him as a girl makes any social theory of sexuality very weak. Genes could themselves nudge one towards a particular sexual orientation or genes may simply interact with other environmental factors such as sex hormones in the womb environment to influence later sexual orientation. The brains of gay and heterosexual people also appear to be organised differently. For example patterns of brain organisation appear similar between gay men and heterosexual women and between lesbian women and heterosexual men. Differences in brain organisation mean differences in psychology and study after study show differences in cognition between heterosexual and gay people. Thus gay differences are not just about who you fancy. They are reflected in our psychology and the ways we relate to others. The influence of biology runs throughout our sexual and gendered lives and those differences, that diversity, is surely to be celebrated. Some writers tend to wave off the scientific evidence by urging us to look to the history of sexuality or claim that homosexuality is a social construction cue Michel Foucault and the like. But these accounts are mere descriptions at best and not scientific theories. Social constructionist accounts generate no hypotheses about sexual orientation and are not subject to systematic testing. So why should we take their claims seriously? Social constructionism and postmodernist theory question the very validity of empirical science in the first place. That makes it no better than climate science denial. Change is widely used to argue against biological explanations. This is false because it is our biology that allows us to learn, respond to socialisation, and helps generate our culture. So showing evidence of change is not an argument against biology. There is indeed some fluidity in sexuality over time, predominantly among women. People may change the identity labels they use and who they have sex with but sexual attractions seem stable over time. Remember, sexual orientation is a pattern of desire, not of behaviour or sexual acts per se. It is not a simple act of will or a performance. We fall in love with men or women because we have gay, straight, or bisexual orientations and not because of choice. They worry more about this than the consequences of choice or environmental explanations, which are not without risk either. But clearly none of the direst predictions have materialised. Sexual minority identities have not been medicalised nor has there been any genetic testing. So should the causes of sexuality influence how we view sexual minority identities? The causes of a trait should not influence how we see it. But the science shows us that sexuality has a biological basis: The biology of sexuality diversity tells the world to deal with it. We are who we are, and our sexualities are part of human nature. It reminds me of something Noam Chomsky alluded to: He studies the biology of sexual orientation

and the implications for mental health and is the co-author of Born Gay? The Psychobiology of Sex Orientation Topics.

Chapter 3 : Love Of Shopping Is Not A Gene, Dagg

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Chapter 4 : gene - Dictionary Definition : racedaydvl.com

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Chapter 5 : "Love Of Shopping" Is Not A Gene: Problems With Darwinian Psychology | Black Rose Books

Curious if the people who reviewed the book negatively actually read it. The way you do science is propose a falsifiable and testable theory. When you are attempting to explain human behaviour now in terms of the evolutionary past, this is not possible.

Chapter 6 : NPR Choice page

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Chapter 7 : Geneâ€“environment interaction - Wikipedia

The book Love Of Shopping Is Not A Gene, Anne Dagg is published by Black Rose Books.

Chapter 8 : Love Of Shopping Is Not A Gene by Anne Innis Dagg

Focusing on the problems present in Darwinian psychological research, Innis Dagg, an eminent and outspoken critic of this ideology, first presents an overview of the theory and its popularity both among professionals and lay people.