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Genetics Beyond Diabetes and Baldness

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Introduction

Previously the concept of genetics was applied to a limited number of diseases with obvious phenotypes where simple people use to observe and relate. It was never easy to establish a clear cut of heritability range between genetic variation and a risk of suffering from a complex disorder due to many reasons including but not limited to; gene variations, non-linear interactions between genetic variance and phenotype severity, complex gene-gene interactions and many others [1]. However, every day, scientists in a research lab somewhere discover a genetic linkage to a disease or a syndrome. This has gone far beyond simple imagination where traits or syndromes used to be related almost exclusively to environment are now genetically linked. The difficulty of identification of patterns among the affected people and the involvement of multiple genes and environmental effects were all behind the difficulty in understanding the link of some of these syndromes/diseases or desired traits to genetics. Advances in genetics/genomics and molecular biology have enabled geneticists to decipher some of the genes that are believed to be linked to some diseases. Recently genetic links has been established to many entities some of which were never thought of as linked with genetics while some were linked to genetics but with no understanding of their patterns. Obesity, neurological disorders, intelligence, asthma, cardiovascular diseases, periodontal diseases, longevity, deafness, cataracts, cancer are just few of the entities that are positively linked to genetics. This editorial will focus mainly on neuropsychiatric genetics including substance addiction, smoking and the genetics of anger.

Addiction or substance use disorders (SUD) have been related to some genetic building blocks that might modulate or predispose to the addiction in individuals carrying such genes. In general, all substance abuse exerts its effect by increasing dopamine (DA) in the central node in the brain reward pathway [1,2]. Therefore, variation in genes related to dopamine signaling may contribute to heterogeneity in addiction phenotypes among different individuals [2]. Certain markers or SNPs that are related to the DA pathway are targeted among the individuals and their multi-locus genetic profile (MLGP) is estimated for each subject [3]. Davis and Loxton [3] also have found that brain reward processes exert their influence by their development of stable personality traits that are positively associated with addictive behaviors. Further, it has been reported that more than 1500 risk genes were found to be involved in different types of addiction behaviors. Interestingly, first degree relatives of SUD experience 4-8 fold increase in the risk of developing the disorder [4]. Nevertheless, the manifestations of SUD might not be solely genetics-related, as the environment sure will have its own effect with more environmental influence during the adolescent period [3]. In fact, genetics along with drug exposure and environmental context are required for the genetic variability to manifest [5]. For instance, variants of delta opioid receptor gene (OPRD1) were identified in subjects with opioid dependence and were intriguingly different from those subjects without opioid dependence [5].

Addiction to Smoking is believed to have a genetic component. Gabrielsen et al. [6] have reported a novel association between rs16969968 polymorphism in CHRNA5 gene and the addicted use of snus, a type of smoke used to help smokers to cease cigarette smoking. It was shown that the individuals who have variant A allele of this SNP have a reduced receptor activity for nicotine and thus require larger amount of the nicotine (smoking more) to a satisfactory level of DA release compared to those with the different allele where they have more of the nicotine receptors. This lead to the conclusion that SNP rs16969968 has an effect on smoking behavior linked to nicotine dependence. Therefore, individuals who have the A allele will have more difficulty in quitting smoking and consequently more risk of developing lung cancer. Further, genome wide studies identified a gene located on chromosome 15 that encodes some nicotine receptor subunits with heritability for smoking initiation to be at 37% in males while heritability for persistence to be 59% in males with similar ratios for females [7].

Anger trait is defined as the frequency of an individual experiencing the emotional state of anger over time in response to a stimulant that might not provoke anger in otherwise normal people. Aggression, hostility and anger-related dispositions are partially heritable as indicated by twin studies [8]. These traits are associated with medical and psychiatric diseases that might lead to increased risk of premature mortality [9]. Variation in serotonin activity or its biosynthesis might be related to aggression related traits in males particularly [8]. Polymorphisms has been identified in some of the serotonin pathways particularly tryptophan hydroxylase (TPH) gene which is located on chromosome 11 and have two alleles U and L. It was found that men who are homozygous for allele A218C L were scored more on assaultiveness, irritability and unprovoked anger than those homozygous or heterozygous for the alternate allele U. In addition, the L allele was more common in criminal offenders than among the none offenders. Other genes might be involved in anger were also discovered. CREB1 is a protein that is found in the hippocampus and is involved in antidepressant response. Exposure to chronic or repeating stress decreases the expression of this protein or decreasing its phosphorylation and thus predisposing individuals to be more vulnerable to stress and anger management. Individuals homozygous for CREB1 rs4675690 polymorphism (CC) with previous history of sexual abuse display higher tendency to express unprovoked anger loudly than individuals homozygous or heterozygous for the alternate allele TT [10]. Hakulinen et al. [9] reported that individuals homozygous for serotonin receptor 1B (HTR1B) rs6296 allele (CC) had higher level of childhood aggressiveness and hostility compared

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to individuals homozygous or heterozygous for the alternate genotype. Interestingly, those who had the childhood aggressiveness with CC alleles had also a higher level of adult aggressiveness and hostility. Rebollo and Boomsma [11] reported that additive genetic effect explain 23% of the anger variance among males and 34% among females.

In conclusion with better understanding of the genetic influence on such phenotypes, it might be possible to better understand the environmental effects on these phenotypes and modify the environment in such a way that minimize or even prevent the onset of these syndromes or bad habits. In addition, better understanding the genetics lying behind these traits might lead to personalized treatments or personalized prevention strategies for individuals at special risk.

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