

Short Communication

Can Genetic Testing Coupled with Enhanced Dopaminergic Activation Reduce Recidivism Rates in the Workers Compensation Legacy Cases?

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An overwhelming segment of the world's population possesses certain genetic variations that increase risk for genetic predispositions to substance seeking and even behavioral addictions (e.g. gambling, internet gaming, multiple sex partners etc.) that preclude them from reaching their optimum health potential, contribute to impaired health, and/or can cause involuntary indulgence in detrimental and self-destructive behaviors. This is especially true when this genetic reward deficiency (RDS) [1] problem leads to not only compulsions and excessive cravings but also impaired decision making. This brain hard wiring will ultimately lead to unwanted narcotic addictions; anti-social behavior; crime and unnecessary medical procedures that burden society [2].

It is our notion that the real genesis of all behavior, whether so-called normal or abnormal behavior, derives from an individual's genetic makeup at birth involving gene variations that make up about 3% of the human genome. While people are not doomed because of carrying these so called variations (called alleles) this risk predisposition, due to multiple gene combinations and polymorphisms (gene variations), is expressed differently based on numerous environmental (epigenetic) elements. So carrying these known gene variants especially in those genes known to control what is called the "Brain Reward Cascade (BRC) influencing how the brain programs feelings of well-being, mediate the actual expression of these important genes [3].

It is well-established that psychiatric disorders are complex multifactorial illnesses involving chronic alterations in the brain reward circuitry. While it is agreed that genetic factors are important in the etiology of disorders such as RDS, relatively high rates of discordance among identical twins suggest the importance of other mechanisms. Certainly, environmental factors such as stress or prior drug exposure play a role in the onset of these illnesses. There is evidence that sustained abnormalities are maintained by epigenetic modifications in specific brain regions. Addictive behavior can be modeled in animals by inducing disease-like states through environmental manipulations (e.g., chronic stress, drug administration) [3].

As David Smith the founder of the Haight-Ashbury Clinic stated "Love Needs Care" and that is what positive caring has in common with powerful epigenetic effects. We firmly embrace this "love and care" concept as part of any treatment goal with the knowledge that through epigenetic effects either methylation and or deacetylation gene expression will lead to positive dopamine release at the reward centers of the brain [4]. Understanding the importance of epigenetics and its effects on chromatin structure will lead to new therapeutic targets to combat for example drug seeking behavior. In fact, we promote the concept that the core of predisposition to these behaviors is a set of genes (called candidate genes), which mediate a feeling of well-being via chemical messenger (i.e. neurotransmitters) interaction at the "reward site" of the brain (N. Accumbens), leading to normal dopamine release and influencing dopamine receptor density (the actual number of dopamine receptors). A low number of Dopamine receptors suggest a hypodopaminergic function as manifested in all addictive disorders. When there are a low number of dopamine receptors, the person will be more prone to seek any substance or behavior that stimulates the dopaminergic system (a sort of "dopamine fix"). In fact, Nora Volkow the current director of the National Institutes of Drug Abuse (NIDA) stated that "all roads lead to dopamine" further described by Blum et al. [5].

To understand generalized craving behavior, due to hypodopaminergic function (a deficiency of reward responsiveness [blunted], individuals self-medicate through biochemical (illicit or nonillicit) attempts to alleviate or compensate for the low dopaminergic brain activity via drug-receptor activation (alcohol, heroin, cocaine, glucose, etc.) [6]. This will substitute for the lack of reward and yield a temporary compensatory sense of well-being. It is this low Dopamine genetic variant that sets these so called "Legacy Case" workers (see below) up for a predisposition for addiction. A very high percentage of these workers are involved in the "injury-treatment-medication-injury" revolving door cycle and may even carry genetic variants (serotonergic and dopaminergic) that result in a higher incident of accidents (e.g. driver accident tendentiousness) for these prone individuals [7].

In order to help explain this so called pseudo self-healing process, we are cognizant of known dysfunctional diagnosis known as "Legacy Cases." These cases are a major problem within the Workers

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