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Short Communication

ABCB1 C3435T Polymorphism and the Risk of Drug Refractory Epilepsy (DRE)

Vivek Kumar Garg*

Demonstrator, Department of Biochemistry, Government Medical College and Hospital, Chandigarh, India

***Corresponding Author**: Vivek Kumar Garg, Demonstrator, Department of Biochemistry, Government Medical College and Hospital, Chandigarh, India.

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Drug refractory epilepsy takes place in one third of the newly treated epileptic patients. These patients become unresponsive to antiepileptic drugs (AEDs) [1]. It deteriorates their social life and also quality of life (QoL) same as occurs in traumatic brain injury [2,3]. Lack of sleep, smoking [4], omission of anticonvulsants are the main precipitating factors for epilepsy [5]. The most common reason for DRE is genetic polymorphisms in ATP-binding cassette subfamily B member 1 (ABCB1) gene. ABCB1 gene is a transporter gene which transports many drugs so known as multidrug transporter gene. It is believed that genetic variations are responsible for DRE [6]. Several studies are published in the past which contradicted each other toward the role of ABCB1 C3435T polymorphism. According to some authors this single nucleotide polymorphism (SNP) is main cause for efflux of AEDs like carbamazepine from the brain tissues so sufficient quantity of AEDs contributing to lesser amount in central nervous system (CNS). Several studies proved that CC genotype of this polymorphism is the major risk factor for the patients who develop pharmacoresistance [7-12]. But most of the studies contradict this statement. According to these positive studies there is no relationship between this SNP and pharmacoresistance [13-15]. In the past, there were many studies and meta-analysis has been done which proved that this polymorphism has no role in pharmacoresistance [6,16-18]. Such difference occurs because of many reasons like the definition of drug resistant and drug responsive patients used by the authors were different. Others reasons are the ethnicity and population difference among these studies. Sample size is also one of the reasons for this contradiction. Metabolic pathways of these AEDs are also responsible for this contradiction. In conclusion, we can't say whether ABCB1 C3435T polymorphism is linked to DRE or not.

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Conflict of Interest

None.

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