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SINGLE NUCLEOTIDE GENE POLYMORPHISM OF INTERLEUKIN-1B GENE AT POSITION –3962 IN TYPE 1 DIABETES OF IRAQI PATIENTS

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ABSTRACT : The aim of this study was to evaluate the frequency of polymorphism of interleukin-1-B gene (IL1B) at position $_{3962}$ SNP) in T1D and in healthy controls subjects39 of Iraqi patients, (12 males & 27 females;1 5.65 ± 1.79 years) and 21 controls. 7 males & 14 female; 14.66 ± 3.43 years were enrolled in this study the polymorphism of IL1-B$_{-3962}$ was data waved by polymerase chain reaction-specific sequence primer (PCR-SSP) assay. Results revealed that comparing *IL1*B$_{-3962}$*genotypes* and *alleles* between T1D patients and controls frequencies of TT genotype and *T allele* (47.93 *vs*. 69.23%; P =0.589, respectively) were significantly rise in patients contrast to controls, (38.32 *vs*. 61.90%; P =0.425) and the related RR rates were 19.2 and 19.2, respectively. And the associated EF values were 1.56 and 1.38. In contrast, CC genotype and *C allele* (9.47 *vs*. 30.77%, P =0.729 respectively) frequencies were significantly decreased in patients, compared to controls (3.05 *vs*. 38.10%; P =0.425) Similar observations were made in TC genotype and associated PF values were 0.77 and 0.72, respectively. These findings suggest that IL1B$_{-3962}$ SNP might have a role in the etiopathogenic mechanism of T1D in the samples of Iraqi patients.

Key words : SNP IL-1B diabetes.

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INTRODUCTION

Diabetes is a chronic disease which influences over 3 million people in the UK –about 10% Type 1 diabetes and residual 90% have Type 2 diabetes (Ruggenenti *et al*, 2010; Bonifacio, 2015). T1D is a serious autoimmune disease affecting millions of people worldwide. This information is for adults with T1D and parents of children with this condition (Bonifacio, 2015). T1D usually starts in childhood, adolescence, or early adulthood, but it may also start later in adult life (Ozougwu *et al*, 2013; Soren and Grey, 2015). Everyone needs a hormone insulin to keep their blood glucose at a normal level. But with T1D, the pancreatic gland not synthesis insulin or make very little of it (Chisholm *et al*, 2010; Ruggenenti *et al*, 2010). T1D is an autoimmune disease characterized by the destruction of the insulin-producing islet β cells. Cytokines act as pleiotropic polypeptides regulating inflammatory and immune responses through actions on cells. They provide important signals in the pathophysiology of a range of diseases, including T1D (Francisco *et al*, 2004; Gonzalez and Fernandez, 2008). There is increasing evidence showing that polymorphisms in cytokine genes may play an important role in modulate the immune response. Numerous cytokines have been shown to participate in the pathogenesis of T1D (Hata *et al*, 2010). Cytokines IL-1β, TNF-α and IFN-γ that are secreted by macrophages and T cells have abroaderrole in the development of T1DM than previously thought (Vincenz *et al*, 2011). As gene polymorphisms can influence in cytokine production or function, they may potentially contributed to genetic predisposition to the disease, as at TGF-B1, TNF-α and IL-6 (Sainz *et al*, 2008;Van de Veerdonk and Netea, 2013). Mediators of inflammation such as TNF-α, IL-1β, the IL-6 family of cytokines, IL-18, and certain chemokine's have proposed to be involved in the events result in both forms of diabetes (Ludwiczek *et al*, 2004; Arend *et al*, 2008; Ortis *et al*, 2012). Further supply for inflammation to contribute to diabetes comes from researchers to examined the role of inflammatory cytokines in diabetic such as IL-1 was first implicated in the development of diabetic (Gonzalez and Fernandez,