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Thesis Title	**Association Studies Between Myokines and Molecular Genotypes of Alcohol Dehydrogenase in Iraqi Alcoholism Academic Years 2011 – 2014**			
Year	**2014**			
Abstract	**involving a complex gene-with –gene and gene with environment interaction.Alcohol affects every body systems causing wide range of health problems ,including Poor nutrition ,memory disorders ,difficulty with balance and walking ,liver disease (including Cirrhosis and hepatitis),nonsustained high blood pressure,anemia clotting disorder ,reduce immunity to Alcoholism belived to be a multifactorial ,polygenic disorder infection ,low blood sugar ,high blood fat content, gastrointestinal inflammatory and muscle weakness (including heart).** **Nearly half of high doses chronic alcohol consumers develop alcoholic skeletal myopathy .The pathogenic mechanismes are:-** **1)chronic alcohol abuse causes reduction in muscle mass specially type II fiber atrophy ,by decreased the number of ribosomes and inhibited of the intiation complex in the translation processes .** **2) Oxidative stress, increased free radical production or decreased activity of antioxidants, impaired free fatty acids and increased lipid peroxidation , muscular membrane alteration ,and alterations in structural proteins DNA, RNA and protein synthesis.** **3)the role of acetaldehyde ,which interacted with proteins ,DNAand form adducts prevent protein secretion and cause enlargement of liver (hepatomegaly).** **Some myokines like Inter leukine-15(IL-15) and Tumer necrosis factor-alpha(TNF-α) were investigated in serum of 140 Iraqi men (70) alcoholic and (70) non alcoholic, living in Baghdad city, by enzyme linked immunosorbent assay (ELISA). Level of IL-15 was higher in alcoholic(59.01±11.09**			

	pg/ml) than in non alcoholic (34.92±9.03 pg/ml).IL-15 was highly expressed in skelatel muscle due to high physical activity of alcoholic compared with nonalcoholic .IL-15 exerts anabolic effect, increased protein synthesis and decreased protein breakdown, it opposite the effect of TNF-α, but it does not overcome the effects of alcohol and its toxic metabolites. **TNF-α, a pro-inflammatory cytokine was higher in alcoholic(83.42 ±10.12 pg/ml) than nonalcoholic (41.68±15.74 pg/ml) due to liver damage causing by ethanol and toxic metabolite acetaldehyde, this cytokine promotes apoptosis and necrosis process .** **There was a significant correlations between IL-15, TNF-α and the quantity of alcohol consumed (g/day) ,duration of intake(by year) ,AST/ALT ratio(medical marker of alcoholic liver disease),GGT, and CK.** **Alcohol metabolism is one of the biological determinants, that can significantly influence drinking behavior and development of alcoholism ,most alcohol elimination occurs in liver by oxidation to acetaldehyde and acetate, catalyzed by alcohol dehydrogenase and aldehyde dehydrogenase both of them exhibit genetic polymorphism and ethnic variation.** **The variants alleles ADH2*2 and ADH3*1 ,which encoded high activity ADH isoforms protect against alcoholism in East asian(orient people),to investigate the type of variant in Iraqi people ,and their role in alcoholism, genotype of samples show that ADH3*1/3*2 was the most common in alcoholic(43%) and nonalcoholic(37%) and the ADH2*2was (33%) in alcoholic and(50%) in nonalcoholic, ADH3*1,was (24%) in alcoholic and (37%) in nonalcoholic ,while ADH2*1 was(67%) in alcoholic and(50%) innonalcoholic, ADH3*2,the low active alleles, were higher in alcoholic(33%) than nonalcoholic(26%) .** **In conclusion the ADH2*2 the active allele in ADH2 genotype plays a major role in the risk of alcoholism , while ADH3*1 also the active allele in ADH3 plays a minor role.**