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IRON EXCESS AND INSULIN RESISTANCE IN CANCER DEVELOPMENT AND PROGRESSION

Juan Ariel Jara Guerrero

Complutense University of Madrid, Spain

ron is physiologically essential for life but biochemically dangerous. Chronic accumulation of iron causes pantropic organ damage and excess body iron play an important role in carcinogenesis, coronary artery disease, neurodegenerative disease, stroke and inflammatory disorders. Iron is very slowly excreted from humans once it is absorbed into the body. The significance of iron excess has been markedly underestimated, despite the fact that iron overloading disorders are as common place in the US white population. Iron-overload and catalytic iron promotes activation of oxidative responsive transcription factors and pro-inflammatory cytokines that increase cancer extension and aggravate them. There is accumulative evidence for iron as a carcinogenic metal in epidemiological, clinical, animal, and cell culture studies. The role of iron in various cancers, such as colorectal and liver cancer was demonstrated. Recent advancements on the molecular mechanisms of iron carcinogenesis evolved the insulin-resistance generation and promotion, fisiopatologia condition that is not only permissive, but may be generated cancer and promoting it. Unlike other nutritional metals, iron is highly conserved: toxicity due to excess iron can occur either acutely after a single dose or chronically due to excessive accumulation in the body from diet. In vivo studies have demonstrated that an iron deficiency induced by either feeding a low iron diet or injecting the iron chelators deferoxamine mesylate decreases tumor growth. Iron supplementation has at times proven ineffective and even detrimental to health. Thus, iron excess may mediate the increased cancer risk associated with insulin resistance and heme-rich diets, and subjects who are insulin resistant can minimize any health risk associated with iron overload by avoiding heme-rich flesh foods and donating blood regularly. The energy that sustains cancer cells derived preferentially from glycolysis depends on the gene p53 deficiencyiron induced.

poetalobo60@gmail.com