

## Why are we a Sensitive Population to COVID-19?

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For many years, the Mexican population has modified their eating habits and lifestyle. Modernization favored spectacular things with electrification, the introduction of television in each home, the intubation and distribution of drinking water, introduction in various hygiene and health measures, national vaccination increased the longevity of the Mexican population, we went from 65 years to 75 years olds in the 70's, and currently we reach an average longevity of 80 years of age. In contrast to these advances, we are currently a highly consuming population in sugary drinks and high carbohydrate food, with the repercussion of high rates of overweight, obesity and unfortunately very high figures for type 2 diabetes (DT2) and hypertension. The unfortunate increase in sedentary lifestyle, little physical activity, and poor nutrition currently shows its effects.

In Mexico, the prevalence of obesity and overweight from 1988 to 2006, it went from 34.5% to 69.3% tripled in just 18 years, unfortunately the high percentage of obesity and DT2 places the country in the second world place after the US. The increase in DT2 [1] is recognized as a national public health problem, in addition to the high costs in the health sector due to the comorbidities to which it is linked. In the child population, 1 in four children (26%) is overweight and obese, increasing in adolescents, 1 in 3 has the problem (31%).

The problem is multifactorial where part of the causes that explain these problems include economic, political, social, and cultural factors, where poverty and the distribution of resources modified habits in the social environments of the population, coupled with genetic factors. Fat or adipose tissue in animals and humans has various functions, among which are recognized: energy balance, lipid and glucose metabolism, thermoregulation, hormonal

and reproductive modulation, modulation of blood pressure, as well as regulation in blood coagulation processes [2].

Overweight and obesity have been recognized as physiological states of chronic inflammation, where elements of the immune response are present. In a state of normal weight and health, hematological components such as macrophages maintain a balanced response together with a low concentration of Leptins and a higher concentration of adiponectin, which together maintain an anti-inflammatory state. On the contrary, when overweight and obesity are present, in fatty tissue several processes are interrupted, in addition to changes in adipocytes; under these conditions, several type monocytes 1 (M1) are confined with adipocytes inside the fatty tissue, generating stages of hypoxia, high load of monocyte chemoattractant proteins (MCP-1). These changes cause an inverse effect in leptin concentration (up) and adiponectin (low), favoring the intervention of other pro-inflammatory elements with the increase of TNF- $\alpha$  (Tumor Necrosis Factor), IL-6 (Interleukin 6), contrasting with the reduction in AMPK (complex enzymatic protein quinase), insulin sensitivity, NF- $\kappa$ B (kappa chain B-cell necrosis factor), with increased secretion of fatty acids. All this generates insulin resistance [1], metabolic syndrome, cardiovascular problems, inflammation among other conditions in target organs [3].

Hypoxia in different areas of fatty tissue leads to the death of adipocytes with an increased inflammatory response. The chronic inflammation associated with obesity has multiple metabolic repercussions with the intervention of pro-inflammatory adipokines, as well as the persistence or presence of type 1 (M1) or pro-inflammatory macrophages over M2 (anti-inflammatory), thus, Investigating how to resolve or reduce the effects of chronic inflammation in individuals with obesity and DT2 could reduce part of the problem [4].

The current epidemic of COVID-19, in its worldwide increasing numbers day by day, never as now it is observed that the stage of persistent inflammation in patients with DT2, overweight, obesity and hypertension are limited in their immune response to the new viral infection. The medical care of patients infected with COVID-19 and these chronic diseases, show that patients where the inflammatory process is treated, the response against viral infection is favored. Perhaps, for the population with these chronic diseases, before the use of vaccines, the population's alternative is to reduce the inflammatory processes that allow the immune response to be restored in these individuals [5].

### Bibliography

1. Saad MJA., *et al.* "Linking gut microbiota and inflammation to obesity and insulin resistance". *Physiology* 31 (2016): 283-293.
2. Izaola O., *et al.* "Inflamación y Obesidad (lipoinflamación)". *Nutrición Hospitalaria* 31.6 (2015): 2352-2358.
3. Kreuter R., *et al.* "The role of obesity in inflammatory bowel disease". *BBA- Molecular Basis of Disease* 1865 (2019): 63-72.
4. Macedo RM and Calder Pc. "Obesity, inflammation, Toll-like receptor 4 and fatty acids". *Nutrients* 10 (2018): 432.2-19.
5. México. "Acuerdo Nacional para la Salud alimentaria. Estrategia contra el sobrepeso y la obesidad". Bicentenario Independencia de México (2010).

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