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Metabolic syndrome

Metabolic syndrome, sometimes known by other names, is a clustering of at least three of the five following medical conditions (giving a total of 16 possible combinations giving the syndrome):

abdominal (central) obesity (cf. TOFI)

elevated blood pressure

elevated fasting plasma glucose

high serum triglycerides

low high-density lipoprotein (HDL) levels

Metabolic syndrome is associated with the risk of developing cardiovascular disease and type 2 diabetes.

In the USA, about a quarter of the adult population have metabolic syndrome, and the prevalence increases with age, with racial ethnic minorities being particularly affected.

Insulin resistance, metabolic syndrome, and prediabetes are closely related to one another and have overlapping aspects.

The syndrome is thought to be caused by an underlying disorder of energy utilization and storage. The cause of the syndrome is an area of ongoing medical research.

He et al. conducted Mendelian randomization (MR) to clarify whether or not the genetically predicted MetS and its components are causally associated with stroke and its subtypes. Genetic instruments of MetS and its components and outcome data sets for stroke and its subtypes came from the gene-wide association study in the UK Biobank and MEGASTROKE consortium, respectively. Inverse variance weighting was utilized as the main method. Genetically predicted MetS, waist circumference (WC), and hypertension increase the risk of stroke. WC and hypertension are related to increased risk of ischemic stroke. MetS, WC, hypertension, and triglycerides (TG) are causally associated with the increasing of large artery stroke. Hypertension increased the risk of cardioembolic stroke. Hypertension and TG lead to 77.43- and 1.19-fold increases, respectively, in small vessel stroke (SVS) risk. The protective role of high-density lipoprotein cholesterol on SVS is identified. Results of the reverse MR analyses show that stroke is related to hypertension risk. From the genetical variants perspective, our study provides novel evidence that early management of MetS and its components are effective strategies to decrease the risk of stroke and its subtypes ¹⁾.

Everyday discrimination contributes to poorer metabolic health in midlife women in the U.S. These findings have clinical implications for the development of MetS, and ultimately cardiovascular disease and diabetes, and intervention strategies to reduce these outcomes ²⁾.

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Patients treated for nonfunctioning pituitary macroadenoma (NFMA) with suprasellar extension show disturbed sleep characteristics, possibly related to hypothalamic dysfunction. In addition to hypopituitarism, both structural hypothalamic damage and sleep restriction per se are associated with the metabolic syndrome, mainly due to decreased HDL-cholesterol and increased triglycerides. Risk factors included hypopituitarism and preoperative visual field defects. Hypothalamic dysfunction may explain the metabolic abnormalities, in addition to intrinsic imperfections of hormone replacement therapy. Additional research is required to explore the relation between derangements in circadian rhythmicity and metabolic syndrome in these patients ³⁾.

1)

He Q, Wang W, Li H, Xiong Y, Tao C, Ma L, You C. Genetic insights into the risk of metabolic syndrome and its components on stroke and its subtypes: Bidirectional Mendelian randomization. J Cereb Blood Flow Metab. 2023 May 18:271678×231169838. doi: 10.1177/0271678×231169838. Epub ahead of print. PMID: 37198928.

2)

Beatty Moody DL, Chang YF, Brown C, Bromberger JT, Matthews KA. Everyday Discrimination and Metabolic Syndrome Incidence in a Racially/Ethnically Diverse Sample: Study of Women's Health Across the Nation (SWAN). Psychosom Med. 2017 Aug 5. doi: 10.1097/PSY.000000000000516. [Epub ahead of print] PubMed PMID: 28787363.

3)

Joustra SD, Claessen KM, Dekkers OM, van Beek AP, Wolffenbuttel BH, Pereira AM, Biermasz NR. High prevalence of metabolic syndrome features in patients previously treated for nonfunctioning pituitary macroadenoma. PLoS One. 2014 Mar 7;9(3):e90602. doi: 10.1371/journal.pone.0090602. eCollection 2014. PubMed PMID: 24608862; PubMed Central PMCID: PMC3946551.

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