The insulin resistance (IR) syndrome is an impairment of humoral regulation of fatty acid (FA) and glucose (GLU) metabolisms at phylogenetically different levels in vivo: at paracrine cell communities and at the total organism. We distinguish between exogenous and endogenous IR syndromes. Exogenous IR syndrome involves physiological function of INS when it is “hampered” by impaired biological function of trophics, i.e., palmitic metabolism of fatty acids which are substrates for mitochondrial oxidation. Endogenous IR is incompliance of bioregulatory functions at the levels of a) the entire organism upon realization of the locomotion function and b) paracrine communities upon realization of the biological function of adaptation (maintenance of “purity” of the intercellular medium), biological reactions of inflammation and homeostasis. There are two types of endogenous IR syndrome. The first results from aphysiologically high dietary intake of exogenous palmitic saturated FA and the second is associated with dietary excess of carbohydrates and oleic metabolism of FA. The IR syndrome is an energy issue in vivo. Insulin primarily regulates FA metabolism and then metabolic conversions of GLU. Low-density lipoproteins are phylogenetically different and not functionally associated systems of cellular transport and intake of different FA.

V.N. Titov
Russian Cardiology Research-and-Production Center, Ministry of Health, Russia

Etiology and pathogenesis of endogenous and exogenous insulin resistance syndromes. Atherosclerosis, oleic and palmitic varieties of fatty acid metabolism.