Editorials

Hydrophobicity: the link between bubbles, bubblers and autoimmunity?

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Some data show that divers can be divided into two groups: 'bubblers' and 'non-bubblers'; no hypothesis has proposed a generally accepted explanation for such a phenomenon.

Hills demonstrated on electron microscopy an oligolamellar lipid lining on the luminal aspect of ovine blood vessels.[1] He also provided evidence of hydrophobicity, using the measured angle to a small (5µl) drop of water. The hydrophobicity was reduced by rinsing these vessels with chloroform, which led to this lining being identified as phospholipids. Hills suggested that the deposition of lung surfactants created this hydrophobic lining. Arieli and Marmur demonstrated clearly defined areas on the surface of blood vessels that fit the suggestion of hydrophobic spots at which bubbles nucleate and grow after decompression from higher pressure.[2] Tiny, flat gas nanobubbles measuring 5–100 nm form spontaneously when a smooth hydrophobic surface is submerged in water containing dissolved gas.[3] One might suggest, therefore, that a permanent layer of nanobubbles covers these hydrophobic intra-vascular spots.

Protein interactions

The chain of amino acids in a protein may include hydrophobic acids, and the α-helices are also the most common structural elements of the protein to cross biological membranes. Because hydrophobicity is high for a gaseous phase, the hydrophobic regions in proteins will react with the gaseous phase. In contact with a gas phase, the configuration of a protein will be altered and the denatured protein will change its immunochemical properties. This process occurs with bubbles in the blood.[4]

Surfactants act against proteins and cause autoimmune diseases

Large protein molecules are probably carried in different quantities and with different timing in the blood. When a large molecule containing a hydrophobic domain encounters the strong hydrophobicity of the nanobubble layer at a surfactant spot, it will adhere to the spot and its altered configuration will be recognized as a foreign molecule, setting in motion an autoimmune response. Autoimmunity increases with age,[5] which is itself considered a risk factor for decompression sickness (DCS) in human divers. If the hydrophobically active spots increase in area and number with age, when added surfactants are deposited, this may explain the concomitant increase in the risk of DCS and in autoimmune diseases.

A large variability in the prevalence of hydrophobic spots in humans may explain differences in sensitivity to autoimmune diseases and to decompression stress (bubblers vs. non-bubblers). The presence of hydrophobic spots and the availability of certain proteins may determine the timing of onset of the autoimmune disease. This might explain the early appearance of Type I diabetes and the later onset of other diseases.

This explanation of the bubblers and non-bubblers division is consistent with the 'de-nucleation' processes as applied to humans before diving and explains why mechanical energy such as vibration, oxygen breathing (at a time interval before diving that is incompatible with desaturation), thermal 'energy' and possibly exercise before diving all appear to be protective for bubble production.[6–9] Once again, science and research in diving show increasingly wider applications and connections with other physiological and pathological processes.

References

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