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Letter to the Editor

Does the most potent lung surfactant dipalmitoylphosphatidylcholine pose a risk for decompression illness in diving mammals?

In the search for the hypothesized gas micronuclei from which bubbles evolve during decompression after diving, we succeeded in establishing the chain of events. The lung surfactant dipalmitoylphosphatidylcholine (DPPC) most probably leaks into the blood stream. Leaving the plasma, the DPPC settles on the luminal aspect of blood vessels (Arieli et al., 2019) to create an oligolamellar lining of phospholipids (Hills 1992). We named this site an "active hydrophobic spot" (AHS) (Arieli, 2017). During the dive, the nanobubbles formed at the AHS from dissolved gas, an occurrence unrelated to diving activity, become the gas micronuclei from which bubbles evolve upon decompression (Arieli, 2017). Because the existence of AHS is the main threat of decompression illness (DCI), it is expected that reduction of AHS would be an important adaptation in deep-diving marine mammals. This could be achieved by the reduction of DPPC in lung surfactants, reduced leakage from the lung to the blood or by reduced settling at the luminal aspect at the blood vessels. DPPC is the most potent of surfactants and comprises about 40 % of the total surfactants in the human lung and in other terrestrial mammals. Because of the near-perpendicular alignment of its two palmitic acids, it can be packed in a dense layer. Diving mammals, diving to depths which cause lung collapse, experience many collapsing and expanding of the lung and therefore their surfactants should be adapted to such swings. Lung surfactants were recently studied in diving mammals (Gutierrez et al., 2015; Spragg et al., 2004). Both studies report an increase in phosphatidylcholine (PC) which increases the fluidity and anti-adhesion characteristics of the surfactant. According to both studies, adaptation to diving in marine mammals was attributed to an increase in fluidity and anti-adhesion in the lung PC, enabling the re-expansion of collapsed alveoli upon ascent from a deep dive.

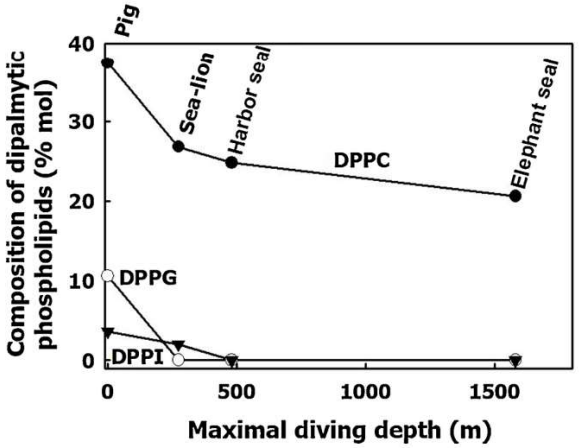

Fig. 1. Composition of DPPC, DPPG and DPPI in mol% of the total lung surfactant phospholipids plotted against maximal diving depth. Data were taken from Spragg et al. (2004) and Eguchi and Harvey (2006).

Spragg et al. (2004) reported a surprising finding: The deep diving elephant seal, has a high minimal surface tension in comparison with other mammals: Elephant seal (11.2), Sea lion (0), Harbor seal (0) and human (0) mN/m. The elephant seal also has a lower concentration of DPPC with respect to all surfactant phospholipids than the others. In Fig. 1, DPPC and other Dipalmitoyl phospholipids: Dipalmitoylphosphatidylglycerine – DPPG and Dipalmitoylphosphatidylinositol - DPPI are plotted as a function of maximal diving depth (Spragg et al., 2004; Eguchi and Harvey, 2006). All dipalmitoylphospholipids are reduced as a function of diving depth. It seems that dipalmytic phospholipids became less favorable with the increase in diving depth. If DPPC is the main surfactant in the active hydrophobic spots (AHS) in the blood vessels, its existence in the lung poses a risk for decompression Illness (DCI). However, DPPC has the highest tension reduction effect as surfactant. Therefore, it is possible that the balance between a beneficial effect on surface tension and a negative effect of DCI is compromised. Thus the deep diving Elephant seal pays the price of a poorer ability to lower surface tension in exchange for reducing the risk of DCI.

Declaration of Competing Interest

The author declares that he has no conflict of interest.

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