Muscle deterioration in patients with lung disease seen connected to CO2

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This is Prof. Yosef Gruenbaum from the Hebrew University of Jerusalem. Credit: The Hebrew University of Jerusalem

Muscle deterioration in patients with lung diseases might be a direct consequence of high carbon dioxide levels in their blood, an international team of researchers headed by Prof. Yosef Gruenbaum of the Hebrew University of Jerusalem has found.

The incidence of lung diseases continues to increase in the world's populations. For example, one in seven persons in the UK is affected by some form of chronic lung disease, most commonly chronic obstructive pulmonary disease (COPD) and asthma.

Many of these diseases also cause, in the worst cases, muscle deterioration as well as elevated levels of carbon dioxide (hypercapnia) in the bloodstream. In a normal situation, the lungs allow for a proper

balance of oxygen from the atmosphere reaching the bloodstream and carbon dioxide from the bloodstream being transferred to the atmosphere.

It is still a matter of some controversy whether the high carbon dioxide levels in patients with chronic lung disease actually cause damage to those patients and specifically whether the loss of muscle is a consequence of those heightened levels.

Prof. Gruenbaum and his Ph.D. student Kfir Sharabi from the Department of Genetics at The Hebrew University, in collaboration with the groups of Dr. Amos J. Simon and Dr. Gideon Rechavi from the Sheba Medical Center and Tel Aviv University, and Dr. Jacob I. Sznajder, Dr. Richard I. Morimoto and Dr. Greg J. Beitel from Northwestern University in the U.S., teamed together to answer these questions. The results of their research appeared in a recent study published in the Proceedings of the National Academy of Sciences (PNAS) in the US.

They used the worm *C. elegans*, in which many basic processes are discovered, to study its response to induced elevated carbon dioxide levels. They found that levels exceeding 9% (normal level in living beings is around 5%) reduced the worm's spontaneous movement capability, which was accompanied by deterioration of body muscle.

(These results suggest re-evaluating the consequences of the procedure of permissive hypercapnia, also known as therapeutic hypercapnia, in which patients with acute lung injury are treated with increased levels of carbon dioxide.)

In addition, there were signs that the experimental animals showed slower development, were less fertile, but, surprisingly, had an increased lifespan. Another unexpected result was the large number of genes that

showed specific and dynamic changes after only one hour of exposure to the heightened carbon dioxide levels.

The researchers noted also that physiological and molecular response to hypercapnia appeared to be different from responses to heat shock or to low oxygen levels.

Source: The Hebrew University of Jerusalem