

## Decavanadate Contribution to Vanadium Cellular Biochemistry: Muscle Contraction and Oxidative Stress

Manuel Aureliano ([maalves@ualg.pt](mailto:maalves@ualg.pt))

*CBME, Dpt. Chemistry and Biochemistry, FCT, University of Algarve, 8005-139 Faro, Portugal*

Although decameric vanadate ( $V_{10}$ ) is not normally considered in biological studies, due to its instability at physiologic pH, it is suggested that it may not completely fall apart into other vanadate oligomers before interacting, *in vitro*, with high-affinity with many proteins particularly those involved in muscle contraction regulation such as sarcoplasmic reticulum calcium pump and myosin (1,2). Moreover, very few *in vivo* animal studies involving vanadium consider that decavanadate might induce changes in cell homeostasis, namely in several stress markers (3). These recent findings, that are now summarized, point out the decameric vanadate species contributions to *in vivo* and *in vitro* vanadium biological effects that may be useful to gain a deeper knowledge of vanadium cellular biochemistry (4).

It is suggested that decavanadate affects myosin-actin interaction and calcium translocation by the calcium pump, therefore disturbing muscle contraction regulation. Besides, an acute exposure to “decavanadate” promotes different effects than other vanadate oligomers in catalase activity, glutathione content, lipid peroxidation, mitochondrial superoxide anion production and vanadium accumulation, whereas both solutions seem to equally depress reactive oxygen species (ROS) production as well as total intracellular reducing power. Questions that remain to be addressed include for instance: i) Can the calcium pump be modulated by  $V_{10}$ ?; ii) How can  $V_{10}$  bind tightly to myosin and prevent actomyosin dissociation?; iii) How is  $V_{10}$  taken up into cells?; iv) Can it be formed in intracellular medium?; v) Is oxidative stress induced by decavanadate due to its decomposition?; vi) How is decavanadate accumulated by mitochondria?; vii) Which are the molecular and subcellular targets of  $V_{10}$  with physiological relevance?

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