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**Correction****Correction: The potential of lipid soluble thiamine in the treatment of cancer****Derrick Lonsdale<sup>1</sup> and Chandler Marrs<sup>2,\*</sup>**<sup>1</sup> Emeritus Cleveland Clinic, Westlake, OH, USA<sup>2</sup> Lucine Health Sciences, Henderson, NV, USA**\* Correspondence:** drchandlermarrs@gmail.com; Tel: +7022814685.**A correction on**

Derrick Lonsdale, Chandler Marrs. The potential of lipid soluble thiamine in the treatment of cancer, by D. Lonsdale, C. Marrs. AIMS Biophysics, 2020, 7(1): 17–26. DOI: 10.3934/biophy.2020002. The original article was submitted and published with an incorrect abstract taken from another source. When the error was identified, it was corrected immediately.

**Abstract:** In the 1920s Otto Heinrich Warburg and his group concluded that deprivation of glucose and oxygen in tumor cells led to a lack of energy, resulting in cell death. He believed that this was the cause of cancer. Later research pointed towards environmental or genetic influences but modern researchers concluded that the Warburg effect was secondary to that of oncogenes. Thus, the prevailing research for many years has been directed almost exclusively towards the study of genetics. The pendulum appears to be swinging back, however, to a more metabolically driven etiology. Arguably, the impairment of oxidative phosphorylation, leading as it does to a decrease of ATP concentration, gives rise to a compensatory massive glucose uptake and anaerobic glycolysis; hallmarks of cancer that are the consequence of the Warburg effect and subsequent mitochondrial damage. Recent mitochondrial research confirms this. When highly metastatic cells are transplanted to media with healthy mitochondria, tumorigenesis is suppressed. Conversely, when healthy cells are paired with unhealthy mitochondria, oncogenesis is induced. This suggests that disturbed oxidative metabolism is a key component of oncogenesis and that rectifying oxidative metabolism may be a promising goal for cancer therapy. As the rate limiting nutrient in oxidative phosphorylation, a fundamental co-factor for amino acid and fatty acid metabolism, thiamine is central to effective

energy management, and as such, it is also indispensable to the energy consuming methylation cycle. Additionally, thiamine connects other common patterns observed in oncogenesis; namely, hypoxia, low glutathione, and elevated or altered ROS reactions forcing the shift towards the telltale anaerobic metabolism common in cancer. Thiamine, thus, is a critical nutrient to assess and address with cancer. This paper discusses the role of thiamine in metabolism, mitochondrial function, epigenetics, and oncogenesis and suggests a role for a lipid soluble form of thiamine in the treatment of cancer.

### **Conflict of interest**

Clinical studies of TTFD have been performed by Dr. Lonsdale since 1973 under independent investigator license IND 11019. Dr. Marrs declares no conflicts of interest.



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