Deutenomics in peer reviewed medical literature

Below is a partial list of papers, as they appeared to provide scientific evidence regarding deutenomics, in the peer reviewed clinical and translational medical literature:

1)
Metabolomics 12, 58 (2016)
Temporal characterization of serum metabolite signatures in lung cancer patients undergoing treatment

Hao, D. et al.

https://link.springer.com/article/10.1007/s11306-016-0961-5

Deutenomics quotation(s) from this paper:

"one possible explanation for the segregation of metabolite pools between those prognostic for survival and those indicative of progression may lie in compartmentalization of biochemical processing and possible mitochondrial dysfunction"

"metabolites from our study related to survival … are long chain fatty alcohols which may result from oxidative processing in peroxisomes or from dietary sources
these are ketogenic substrates which are lower in deuterium content …"

2)
International Journal of Molecular Sciences 20(20), 4984 (2019)
Personalized Treatment Response Assessment for Rare Childhood Tumors Using Microcalorimetry–Exemplified by Use of Carbonic Anhydrase IX and Aquaporin 1 Inhibitors

Gros, S. J. et al.

https://doi.org/10.3390/ijms20204984

Deutenomics quotation(s) from this paper:

"Boros et al. have proposed that mechanisms similar to the inhibition of carbonic anhydrase can inhibit growth of tumor cells by limiting uptake of deuterated water into cells"

"these authors further suggest that these mechanisms might result in metabolic changes of translational impact"

3)
Scientific Reports 10, 5955 (2020)
Network-based metabolic characterization of renal cell carcinoma

Pandey, N. et al.

https://doi.org/10.1038/s41598-020-62853-8

Deutenomics quotation(s) from this paper:

"Renal cell carcinoma is caused by defective mitochondria that can impose tumor transformation by deuterium oncoisotope accumulation. Therefore, deuterium depletion (deupletion) upon deuterium depleted substrate oxidation can provide a low deuterium metabolic water as therapeutic adjuvant. Such metabolic intervention can be initiated and maintained via diet and potables in integrative therapeutic settings"

4)
Molecular Cancer Research 18(6), 883–890 (2020)
Metabolic Profiling of Formalin-Fixed Paraffin-Embedded Tissues Discriminates Normal Colon from Colorectal Cancer

Arima, K., et al.

https://doi.org/10.1158/1541-7786.MCR-19-1091

Deutenomics quotation(s) from this paper:

"Because these essential ketogenic BCAAs are degraded directly into acetyl-CoA and succinyl-CoA in human mitochondria, their accumulation reiterates mitochondrial dysfunction in tumor cells to completely oxidize ketogenic substrates into carbon dioxide that is accompanied by inherent transfer of protons with less deuterium from nutrients to metabolic matrix water (36, 37). The fundamental mitochondrial function of curtailing deuterium oncoisotope accumulation in intermediary metabolites and nucleotides to prevent cell transformation has been argued as a surrogate marker for predicting response in personalized treatment and as a prognostic marker for patient survival in several clinical cancer studies (38, 39). Our study thus ingeminates again the clinical importance of insufficient mitochondrial deuterium depletion with resulting oncogenic transformation that can be unearthed from paraffin embedded tissue samples via metabolic profiling."

5)
Scientific Reports 11, 3250 (2021)
Indication of high lipid content in epithelial-mesenchymal transitions of breast tissues

Sabtu, S. N., et al.

https://doi.org/10.1038/s41598-021-81426-x

Deutenomics quotation(s) from this paper:

"Attention is drawn to potential links to several original contributions within which deupleting metabolic markers have been demonstrated clinically. These involve lung[76], rare childhood cancers[77], renal cell cancers[78] and colorectal cancers[79], until now lacking evidence in breast cancer. The growing field of what is referred to as deutenomics has consistently shown the increased significance of deuterium depletion via natural cellular ketogenic substrate oxidation. The underlying medical biochemistry mechanisms, described by Boros et al.[80,81], have considered how defective mitochondria with diminished low deuterium ketogenic fatty acid substrate oxidation can hamper recycling of deuterium depleted metabolic water. This is seen to be performed by tricarboxylic acid cycle (TCA cycle) hydratase reactions[82]. Such mechanism could preserve normal epithelial cellular mesenchymal phenotype in breast to prevent or reverse cancer formation."

6)
Vaccines 10(5), 790 (2022)
Targeting Membrane Trafficking as a Strategy for Cancer Treatment

Tejeda-Muñoz, N., et al.

https://doi.org/10.3390/vaccines10050790

Deutenomics quotation(s) from this paper:

"Vacuolar-ATPase (V-ATPase), initially identified in Saccharomyces cerevisiae and plant vacuoles, is an 830 kDa multi-subunit transmembrane complex. V-ATPases have a similar structure and mechanism of action to mitochondrial F-ATPase (F-type), and several of their subunits evolved from common ancestors. V-ATPase serves to pump protons into the lumen of different endosomal compartments and contribute to endosomal acidification [30] via deuterium discrimination [31] (Figure 3), whereas F-ATPase synthesizes most of the ATP and deuterium-depleted metabolic water in the matrix of mitochondria using an electrochemical proton gradient and oxygen in complex IV [32]."

7)
Scientific Reports 13, 12136 (2023)
Identification of metabolic pathways contributing to ER+ breast cancer disparities using a machine-learning pipeline

Santaliz-Casiano, A., et al.

https://doi.org/10.1038/s41598-023-39215-1

Deutenomics quotation(s) from this paper:

"Consistently, other studies also reported a role for free fatty acids in other cancer types including lung64 childhood tumors65, and colon cancer66. Recent studies using biological deuterium fractionation and discrimination points out diet as the main source of increased fatty acid pool in plasma, which is delivered to cells via circulation. Thus, fatty acids act as the intermediate proton carrying carbon source for mitochondrial respiration. Further, generation of ketones using these deuterium-depleted fatty acids might explain benefit of ketogenic diets. Food insecurity and inequality in food quality and availability resulting in metabolic inefficiency might contribute to differential fatty acid profiles in AA vs. NHW women and breast cancer disparities."

8)

Nutrients 15(20), 4360 (2023)
Regulation of Tumor Apoptosis of Poriae cutis-Derived Lanostane Triterpenes by AKT/PI3K and MAPK Signaling Pathways In Vitro

Yue, S., et al.

https://doi.org/10.3390/nu15204360

Deutenomics quotation(s) from this paper:

"Moreover, the triterpenoids consist of six isoprenoid (2-methyl-1,3-butadiene) ketone units, whose highly saturated carbon skeleton provides a large number of protons for the production of deuterium-depleted metabolic water by mitochondria. Natural triterpenoids can be used as ketogenic substrates by cells with lower deuterium content than cytoplasmic water, thus aiding mitochondrial NADPH-dependent macromolecular synthesis, including DNA [82]. In fact, it has been demonstrated that deuterium-depleted water inhibits lung tumor growth in vivo by lowering proliferation in the A549 cell line, while enhancing apoptosis [83]. Deutenomics research has also been used to study colorectal cancer. Essential ketogenic branched-chain amino acids are directly converted to succinyl-CoA and acetyl-CoA in mitochondria, where deuterium-depleted proton transfer from nutrients to metabolic substrate water is provided by natural ketogenic substrates. Depletion of deuterium isoforms in nucleotides prevents transformation of colon cells due to cycle arrest [84]. Deuterium depletion caused by the oxidation of natural cellular ketogenic substrates

can prevent or reverse the development of breast cancer and is of increasing importance [85]. Studies of biological deuterium fractionation and identification techniques have indicated that deuterium-depleted fatty acids enter cells through the circulation and act as an intermediate proton carrying carbon source for mitochondrial respiration [86]. Extracellular deuterium depletion may serve as a metabolic therapeutic adjuvant that can be initiated by a diet with depleted water drinking [86,87]. In these subsequent investigations, triterpenes were further examined in vivo for their effect on cancer metabolism-related multiple metabolites and to evaluate the deuterium depletion potential of PAA and PAB during metabolic water formation."

9)

Cancers 16(3), 480 (2024)

Incorporating Novel Technologies in Precision Oncology for Colorectal Cancer: Advancing Personalized Medicine

Ahluwalia, P., et al.

https://doi.org/10.3390/cancers16030480

Deutenomics quotation(s) from this paper:

"This dysfunction involves multiple molecular factors, including impaired proton transfer to metabolic matrix water, leading to less deuterium content. Healthy mitochondria function involves the fundamental function of inhibiting deuterium oncoisotope accumulation inside the healthy cell. The continuous accumulation of deuterium leads to cancer development and holds promise as a prognostic biomarker in multiple cancer studies [166,167]."

10)