Mitochondrial deuterium depletion restrains prokaryote proliferation and virus hosting cellular events thus may alleviate the use of biologics

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Abstract

The proliferation of prokaryote organisms (1), virus hosting cellular events and rapid clonal expansion of transformed cells (2, 3) avail themselves via ATP-dependent selective hydrogen ($^1H^+$) efflux by proton transporter nanomotors. This process is to retain metabolic deuterium isotopes, i.e. heavy hydrogen ($^2H^+$), which nurture DNA instability (4). This later phenomenon maintains unlimited propagation of infectious agents via constitutively active nutrient-dependent and growth-related anabolic biochemical reaction channels. Biologics in the form of vaccines are increasingly utilized to prevent, treat and manage such cellulo-proliferative diseases that entail bacterial, viral and protozoal infections as suspected agents with disease causing etiologies. Oppositely, there are differentiated eukaryote cells with a strong restraint on growth that is attainable by deuterium depletion (deupletion) during matrix water production as the core metabolic phenotype occurring by natural ketogenic substrate oxidation in mitochondria (5). Nonetheless, long overlooked deuterium accumulation processes during continuous self-propagation of prokaryotes, virus affected and transformed cells with aneuploidy provide novel therapeutic arsenals to prevent and treat infections. Population scale natural ketosis based nutritional deupletion protocols (6) may bring about limited needs for the rapid increase in vaccinations. In other words, deupleting nutritional strategies in biology may efficiently control bacterial, fungal and virus infected cell proliferation by implementing natural ketosis as a metabolic intervention that can now be offered in place of the use of biologics. Endemics and epidemics of human diseases match inherent increases in the consumption of deuterium by means of potable water (7), farmed livestock, genetically modified produce and consuming highly processed carbohydrates with additives. The resulting population disparities in deuterium exposure, infections and cancer, along with other chronic disease conditions characterized by metabolic crowding, at the expense of complete substrate oxidation with consequent endogenous low deuterium metabolic water production further enfeeble immune functions.

Thus, deupleting research narratives that explain mammalian cell differentiation with constrained growth will likely attain great therapeutic benefits to halt prokaryote expansions and propagation, which can simply be achieved by replacing processed glycogenic substrates with low deuterium natural ketogenic ones in the nutrition of humans. A similar metabolic exchange of glycogenic lactate fermentation into the ketogenic propionate substrate in high performance athletes was shown to occur (8) during the continuous proliferation of resident prokaryotes in the gut (9) to collect deuterium, which is the primary function of the microbiome. Epithelial tissue residents, e.g. bacteria, yeast and viruses readily relay additional models of seasonal deupletion for disease prevention by prokaryotes.

Deuterium biochemistry by its strong kinetic oncoisotope effects can explain proliferation, reactivation, and expansion of microbes as well as that of transformed cells more elegantly and efficiently than all other prevailing theories. Incorporating medicinal deutenomics and deupletion sciences into the educational curricula of clinical and translational sciences may significantly alleviate the need of the repeated use of adjuvant contaminated biologics with unpredictable toxicity profiles and side effects.

Literature:

1.) Kotyk A., et al. Deuterons cannot replace protons in active transport processes in yeast. FEBS Lett. 1990 May 21;264(2):203-5. https://www.doi.org/10.1016/0014-5793(90)80248-h
2.) Somlyai G., et al. Naturally occurring deuterium is essential for the normal growth rate of cells. FEBS Lett. 1993 Feb 8;317(1-2):1-4. https://www.doi.org/10.1016/0014-5793(93)81479-j
3.) Perona R., et al. Increased pH and tumorigenicity of fibroblasts expressing a yeast proton pump. Nature. 1988 Aug 4;334(6181):438-40. https://www.doi.org/10.1038/334438a0
4.) Sobczyk L., et al. H/D isotope effects in hydrogen bonded systems. Molecules. 2013 Apr 16;18(4):4467-76. https://www.doi.org/10.3390/molecules18044467
5.) Boros LG., et al. Submolecular regulation of cell transformation by deuterium depleting water exchange reactions in the tricarboxylic acid substrate cycle. Med Hypotheses. 2016 Feb;87:69-74. https://www.doi.org/10.1016/j.mehy.2015.11.016
6.) Boros LG., et al. What to eat or what not to eat-that is still the question. Neuro Oncol. 2017 Apr 1;19(4):595-596. https://www.doi.org/10.1093/neuonc/now284
7.) Strekalova T., et al. Deuterium content of water increases depression susceptibility: the potential role of a serotonin-related mechanism. Behav Brain Res. 2015 Jan 15;277:237-44. https://www.doi.org/10.1016/j.bbr.2014.07.039
8.) Scheiman J., et al. Meta-omics analysis of elite athletes identifies a performance-enhancing microbe that functions via lactate metabolism. Nat Med. 2019 Jul;25(7):1104-1109. https://www.doi.org/10.1038/s41591-019-0485-4
9.) Boros LG. Elite athletes harbor performance-enhancing gut microbe that turns lactate into fat. https://www.laszlogboros.com/post/elite-athletes-harbor-performance-enhancing-gut-microbe-that-turns-lactate-into-fat

Key Words:
deuterium depletion, mitochondrial matrix, metabolic water, deuterium-depleted water, NADPH, structured water, interfacial water, quantum destabilization of water protons, natural ketosis, deutenomics, deupletion, deuccumulation, deuposition

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L'esaurimento del deuterio mitocondriale limita la proliferazione dei procarioti e gli eventi cellulari dei virus ospitanti e quindi può alleviare l'uso di sostanze biologiche

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Abstract

La proliferazione degli organismi procarioti (1), gli eventi cellulari dei virus ospitanti e la rapida espansione clonale delle cellule trasformate (2, 3) si avvalgono dell'efflusso di idrogeno ($^1H^+$) selettivo dipendente dall'ATP da parte dei nanomotori trasportatori di protoni. Questo processo serve a mantenere gli isotopi del deuterio metabolico, cioè l'idrogeno pesante ($^2H^+$), che alimenta l'instabilità del DNA (4). Quest'ultimo fenomeno mantiene una propagazione illimitata di agenti infettivi attraverso canali di reazione biochimici anabolizzanti costitutivamente attivi e dipendenti dalla crescita e da fattori nutritivi. I farmaci biologici sotto forma di vaccini sono sempre più utilizzati per prevenire, curare e gestire tali malattie cellulo-proliferative che comportano infezioni batteriche, virali e protozoarie come agenti sospetti con eziologie che causano malattie. Al contrario, ci sono cellule eucariote differenziate con una forte limitazione alla crescita che è raggiungibile dalla deplezione del deuterio (deupletion) durante la produzione di acqua di matrice come fenotipo metabolico centrale che si verifica dall'ossidazione del substrato chetogenico naturale nei mitocondri (5). Tuttavia, i processi di accumulo di deuterio da lungo tempo trascurati durante la continua auto-propagazione dei procarioti, le cellule affette da virus e trasformate con aneuploidia forniscono nuovi arsenali terapeutici per prevenire e curare le infezioni. I protocolli di deplezione nutrizionale basati sulla chetosi naturale su scala di popolazione (6) possono portare ad esigenze limitate per il rapido aumento delle vaccinazioni. In altre parole, strategie nutrizionali in biologia basate sulla deplezione del deuterio (deuplezione) può controllare efficacemente la proliferazione batterica, fungina e cellulare infetta da virus implementando la chetosi naturale come un intervento metabolico che ora può essere offerto al posto dell'uso dei farmaci biologici. Le endemie e le epidemie delle malattie umane corrispondono ad aumenti inerenti al consumo di deuterio mediante acqua potabile (7), bestiame d'allevamento, prodotti geneticamente modificati e consumo di carboidrati altamente trasformati con additivi. Le risultanti disparità di popolazione nell'esposizione al deuterio, nelle infezioni e nei tumori, insieme ad altre patologie croniche caratterizzate da sovraccarico metabolico, a scapito dell'ossidazione completa del substrato con conseguente produzione endogena di acqua metabolica a basso deuterio, indeboliscono ulteriormente le funzioni immunitarie.

Pertanto, le narrative di ricerca sulla deuplezione che spiegano la differenziazione delle cellule di mammifero con una crescita limitata probabilmente otterrà grandi benefici terapeutici per fermare le espansioni e la propagazione dei procarioti, che possono essere semplicemente ottenute sostituendo i substrati glicogeni elaborati con quelli chetogenici naturali a basso deuterio nella nutrizione umana. Un simile scambio metabolico della fermentazione del lattato glicogenico nel substrato chetogenico del propionato in atleti ad alte prestazioni è stato dimostrato (8) durante la continua proliferazione dei procarioti residenti nell'intestino (9) per raccogliere il deuterio, che è la funzione primaria del microbioma. Residenti del tessuto epiteliale, ad es. batteri, lieviti e virus trasmettono prontamente ulteriori modelli di deuplezione stagionale per la prevenzione delle malattie da procarioti.

La biochimica del deuterio con i suoi forti effetti oncoisotopici cinetici può spiegare la proliferazione, la riattivazione e l'espansione dei microbi nonché quella delle cellule trasformate in modo più

elegante ed efficiente rispetto a tutte le altre teorie prevalenti. Incorporare la deutenomica medicinale e le scienze della deplezione nei curricoli educativi delle scienze cliniche e traslazionali può alleviare in modo significativo la necessità dell'uso ripetuto di sostanze biologiche con adiuvanti contaminate con profili di tossicità ed effetti collaterali imprevedibili.

Letteratura:

1.) Kotyk A., et al. Deuterons cannot replace protons in active transport processes in yeast. FEBS Lett. 1990 May 21;264(2):203-5. https://www.doi.org/10.1016/0014-5793(90)80248-h
2.) Somlyai G., et al. Naturally occurring deuterium is essential for the normal growth rate of cells. FEBS Lett. 1993 Feb 8;317(1-2):1-4. https://www.doi.org/10.1016/0014-5793(93)81479-j
3.) Perona R., et al. Increased pH and tumorigenicity of fibroblasts expressing a yeast proton pump. Nature. 1988 Aug 4;334(6181):438-40. https://www.doi.org/10.1038/334438a0
4.) Sobczyk L., et al. H/D isotope effects in hydrogen bonded systems. Molecules. 2013 Apr 16;18(4):4467-76. https://www.doi.org/10.3390/molecules18044467
5.) Boros LG., et al. Submolecular regulation of cell transformation by deuterium depleting water exchange reactions in the tricarboxylic acid substrate cycle. Med Hypotheses. 2016 Feb;87:69-74. https://www.doi.org/10.1016/j.mehy.2015.11.016
6.) Boros LG., et al. What to eat or what not to eat-that is still the question. Neuro Oncol. 2017 Apr 1;19(4):595-596. https://www.doi.org/10.1093/neuonc/now284
7.) Strekalova T., et al. Deuterium content of water increases depression susceptibility: the potential role of a serotonin-related mechanism. Behav Brain Res. 2015 Jan 15;277:237-44. https://www.doi.org/10.1016/j.bbr.2014.07.039
8.) Scheiman J., et al. Meta-omics analysis of elite athletes identifies a performance-enhancing microbe that functions via lactate metabolism. Nat Med. 2019 Jul;25(7):1104-1109. https://www.doi.org/10.1038/s41591-019-0485-4
9.) Boros LG. Elite athletes harbor performance-enhancing gut microbe that turns lactate into fat. https://www.laszlogboros.com/post/elite-athletes-harbor-performance-enhancing-gut-microbe-that-turns-lactate-into-fat

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Il prof. Laszlo Boros ha recentemente vinto un processo come unico consulente di parte per un caso di cancerogenesi chimica contro la Terminix International Company (“una donna di 60 anni muore di leucemia mieloide acuta dopo aver bevuto da un pozzo contaminato dal termiticida” - Ammontare del verdetto: 1,47 milioni di dollari: https://www.scottmullinslaw.com/settlements-verdicts/) grazie ai suoi studi sulla deplezione del deuterio

1. Boros LG, Williams RD. Chronic isofenphos poisoning: case report of agnogenic myeloid metaplasia with a rapid progression into acute myeloid leukemia. Leuk Res. 1998 Sep;22(9):849-51. https://doi.org/10.1016/S0145-2126(98)00052-6

2. Boros LG, Williams RD. Isofenphos induced metabolic changes in K562 myeloid blast cells. Leuk Res. 2001 Oct;25(10):883-90. https://doi.org/10.1016/S0145-2126(01)00043-1
3. Williams RD, Boros LG, Kolanko CJ, Jackman SM, Eggers TR. Chromosomal aberrations in human lymphocytes exposed to the anticholinesterase pesticide isofenphos with mechanisms of leukemogenesis. Leuk Res. 2004 Sep;28(9):947-58. https://doi.org/10.1016/j.leukres.2003.12.014