

# Draft 2: Mitochondria

<b>Maternal Immune Activation</b>	<p><a href="#"><u>Developmental Stage-Dependent Changes in Mitochondrial Function in the Brain of Offspring Following Prenatal Maternal Immune Activation (2023)</u></a></p> <p><a href="#"><u>Maternal immune activation primes deficiencies in adult hippocampal neurogenesis (2021)</u></a></p>
<b>Immune System</b>	<p><a href="#"><u>Mitochondrial Dysfunction and the Aging Immune System.</u></a></p> <p>McGuire PJ. <i>Biology (Basel)</i>. 2019 May 11;8(2):26. doi: 10.3390/biology8020026. PMID: 31083529 Free PMC article. Review.</p> <p>Many of these mitochondrial functions decline with age, and are the basis for many diseases of aging. ...In this review, three main issues facing the aging immune system are discussed: (1)</p>

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April 20, 2024

Page 1 of 8

inflamm-aging; (2) susceptibility to infection and (3) declinin ...

[T cells with dysfunctional mitochondria induce multimorbidity and premature senescence \[deterioration with age\].](#)

Desdín-Micó G, Soto-Heredero G, Aranda JF, Oller J, Carrasco E, Gabandé-Rodríguez E, Blanco EM, Alfranca A, Cussó L, Desco M, Ibañez B, Gortazar AR, Fernández-Marcos P, Navarro MN, Hernaez B, Alcamí A, Baixauli F, Mittelbrunn M. *Science*. 2020 Jun 19;368(6497):1371-1376. doi: 10.1126/science.aax0860. Epub 2020 May 21. PMID: 32439659

The effect of immunometabolism on age-associated diseases remains uncertain. In this work, we show that T cells with dysfunctional mitochondria owing to mitochondrial transcription factor A (TFAM) deficiency act as accelerators of senescence [deterioration with age]. ...Blocking tumo ...

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Page 2 of 8

<p><b>Microbiome</b></p>	<p><a href="#">Gut bacteria signaling to mitochondria in intestinal inflammation and cancer.</a></p> <p>Jackson DN, Theiss AL. Gut Microbes. 2020 May 3;11(3):285-304. doi: 10.1080/19490976.2019.1592421. Epub 2019 Mar 26. PMID: 30913966 Free PMC article. Review.</p> <p>Both dysbiosis of the gut microbiota and mitochondrial dysfunction are associated with chronic intestinal inflammation and CRC. This review discusses mitochondrial metabolism of gut mucosal cells, mitochondrial dysfunction, and known gut ...</p>
<p><b>Endocrine System</b></p>	<p><a href="#">Mediators of allostasis and systemic toxicity in bipolar disorder.</a></p> <p>Grande I, Magalhães PV, Kunz M, Vieta E, Kapczinski F. Physiol Behav. 2012 Apr</p>

	<p><b>12;106(1):46-50. doi: 10.1016/j.physbeh.2011.10.029. Epub 2011 Nov 3. PMID: 22079584 Review.</b></p> <p><b>Glucocorticoids are fundamental mediators; when chronically in excess, glucocorticoids initiate a series of bodily dysfunctions that may include cortisol-related mitochondrial dysfunction, oxidative stress, inflammation and decrease in the expression of neuro ...</b></p>
<p><b>Epigenetics</b></p>	<p><b><a href="#">Metabolic Disease Programming: From Mitochondria to Epigenetics, Glucocorticoid Signalling and Beyond.</a></b></p> <p><b>Grilo LF, Tocantins C, Diniz MS, Gomes RM, Oliveira PJ, Matafome P, Pereira SP. Eur J Clin Invest. 2021 Oct;51(10):e13625. doi: 10.1111/eci.13625. Epub 2021 Jun 13. PMID: 34060076 Review.</b></p> <p><b>This review summarizes recent biomedical discoveries in the Developmental Origins of Health and Disease (DOHaD) hypothesis and</b></p>

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Page 4 of 8

	<p>highlight possible developmental programming mechanisms, including prenatal structural defects, metabolic (mitochondrial dysfunction, oxidative stress, p ...</p>
<p><b>Trauma</b></p>	<p><a href="#">Epigenetic embedding of childhood adversity: mitochondrial metabolism and neurobiology of stress-related CNS diseases.</a></p> <p>Bigio B, Sagi Y, Barnhill O, Dobbin J, El Shahawy O, de Angelis P, Nasca C. Front Mol Neurosci. 2023 Jul 25;16:1183184. doi: 10.3389/fnmol.2023.1183184. eCollection 2023. PMID: 37564785 Free PMC article. Review.</p> <p>This invited article ad memoriam of Bruce McEwen discusses emerging epigenetic mechanisms underlying the long and winding road from adverse childhood experiences to adult physiology and brain functions. The conceptual framework that we pursue suggest multidimensional biolo ...</p>

	<p><a href="#"><u>The role of suboptimal mitochondrial function in vulnerability to post-traumatic stress disorder.</u></a></p> <p>Preston G, Kirdar F, Kozicz T. J Inherit Metab Dis. 2018 Jul;41(4):585-596. doi: 10.1007/s10545-018-0168-1. Epub 2018 Mar 28. PMID: 29594645 Review.</p> <p>Post-traumatic stress disorder remains the most significant psychiatric condition associated with exposure to a traumatic event, though rates of traumatic event exposure far outstrip incidence of PTSD. Mitochondrial dysfunction and suboptimal mitochondrial fu ...</p>
<p><b>Toxins</b></p>	<p><a href="#"><u>Bisphenol A-Induced Endocrine Dysfunction and its Associated Metabolic Disorders.</u></a></p> <p>Maniradhan M, Calivarathan L. Endocr Metab Immune Disord Drug Targets. 2023;23(4):515-529. doi: 10.2174/1871530322666220928144043. PMID: 36173044 Review.</p>

**Humans are exposed to BPA, which directly or indirectly causes endocrine dysfunctions that lead to metabolic disorders like obesity, fatty liver diseases, insulin resistance, polycystic ovarian syndrome, and other endocrine- related imbalances. ...Due to its ...**

**[Mitochondria as a Source and a Target for Uremic Toxins.](#)**

**Popkov VA, Silachev DN, Zalevsky AO, Zorov DB, Plotnikov EY. Int J Mol Sci. 2019 Jun 25;20(12):3094. doi: 10.3390/ijms20123094. PMID: 31242575 Free PMC article. Review.**

**However, mitochondria can be the source of uremic toxins as well, as the mitochondrion can be the site of complete synthesis of the toxin, whereas in some scenarios only some enzymes of the pathway of toxin synthesis are localized here. ...Additionally ...**
