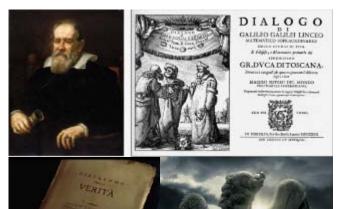
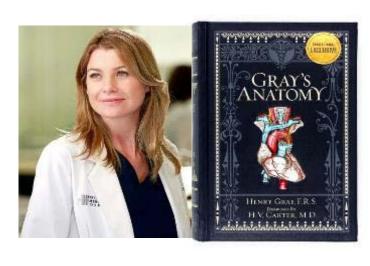




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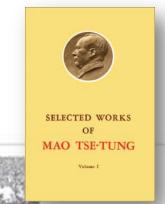
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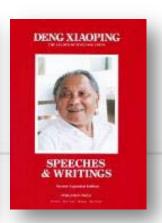


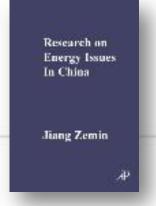




















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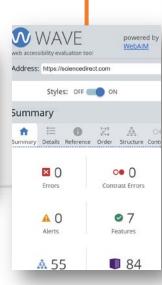
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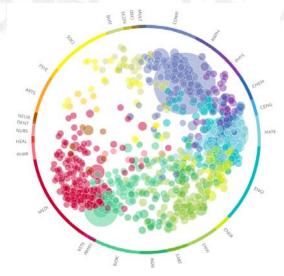
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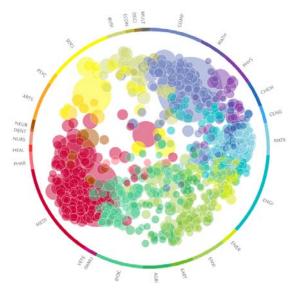


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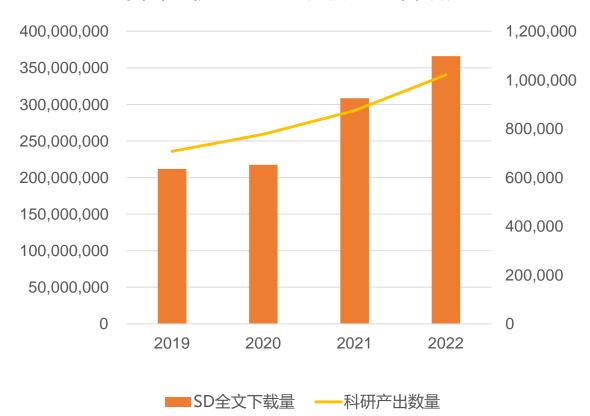
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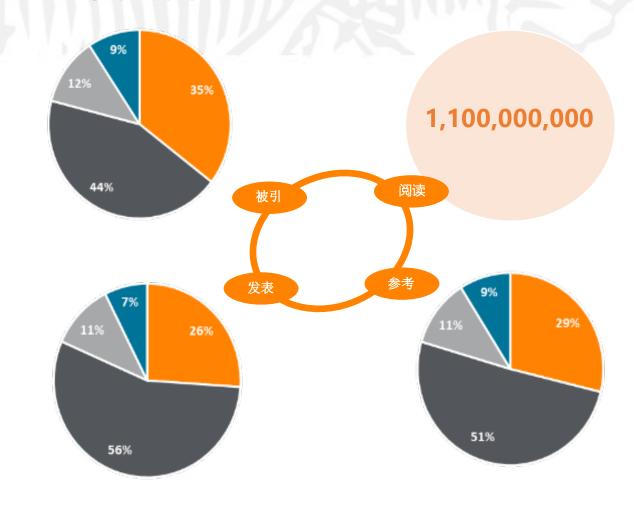




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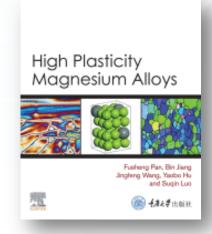


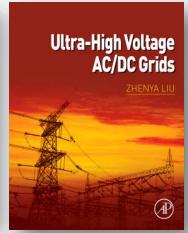


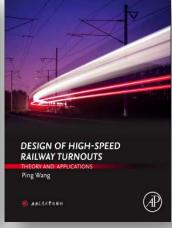












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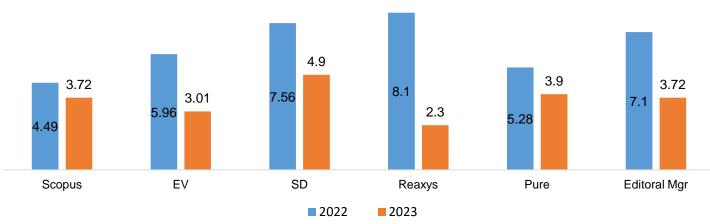
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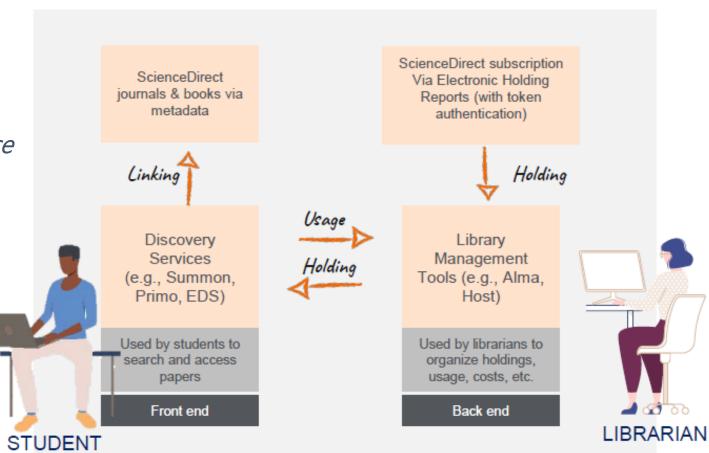
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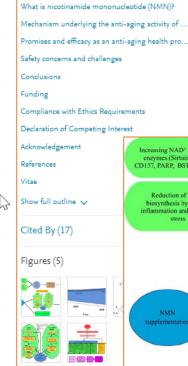
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Graphical abstract

Abstract

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Nicotinamide mononucleotide (NMN) as an anti-aging health product - Promises and safety concerns

Harshani Nadeeshani a, Jinyao Li b, Tianlei Ying c, Baohong Zhang d, Jun Lu a e f g h i j 🙎 🖼

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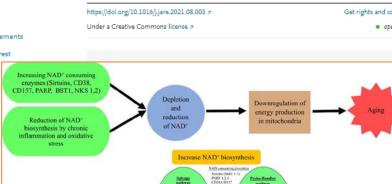
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epletion NAD* levels in the body



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Abstract

Background

Elderly population has been progressively rising in the world, thus the demand for antiaging heath products to assure longevity as well as to ameliorate age-related complications is also on the rise. Among various anti-aging health products, nicotinamide mononucleotide (NMN) has been gaining attentions of the consumers and the scientific community.

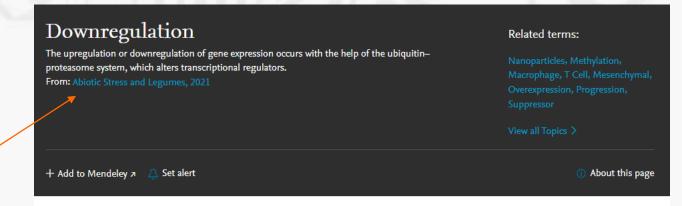
Aim of review

This article intends to provide an overview on the current knowledge on promises and safety concerns of NMN as an anti-aging health product.

Key scientific concepts of review

Nicotinamide adenine dinucleotide (NAD⁺) levels in the body deplete with aging and it is associated with downregulation of energy production in mitochondria, oxidative stress, DNA damage cognitive impairment and inflammatory conditions. However, NMN, as the precursor of NAD⁺, can slow down this process by elevating NAD⁺ levels in the body. A number of *in vivo* studies have indicated affirmative results of therapeutic effects for various age-induced complications with NMN supplementation. One preclinical and one clinical study have been conducted to investigate the safety concerns of NMN administration while a few more human clinical trials are being conducted. As there is a large influx of NMN based anti-aging products on the market, proper clinical investigations are urgently needed to find out the effectiveness and safety of NMN supplementation.

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miRNAs: Nanomachines That Micromanage the Pathophysiology of Diabetes Mellitus

Shilpy Sharma, ... Jeetender Chugh, in Advances in Clinical Chemistry, 2017

3.2.5 miR146a

Downregulation of miR146a has been observed in microvascular HRECs and endothelial cells from large vessels and retinal microvessels grown in high glucose concentration, thereby leading to inflammation [132,133]. It has been observed that overexpression of miR146a using miRNA mimics attenuates hyperglycemia-induced inflammatory pathways through reduced TLR4/NF- κ B and TNF α signaling [132]. While fibronectin has been identified as a target for miR146a, p300 has been shown to regulate miR146a levels [133]. These studies suggest that miR146a downregulation could be a key mechanism for increased extracellular matrix (ECM) protein production in diabetes. Contrary to these observations, increased miR146a levels have been reported

Epigenomic reprogramming of caloric restriction on aging

Samo Ribarič, in Nutritional Epigenomics, 2019

4.1 CR modulates post-translational modification of histones

CR promotes <u>histone deacetylation</u> and <u>methylation</u> leading to down-regulation of p53 (tumor suppressor protein p53 also known as tumor protein 53), FOXO (O subclass of the forkhead family of transcription factors), <u>Ku70</u> (protein encoded in humans by the gene XRCC6) and <u>p16</u>^{Ink4a} (gene encoding the <u>tumor suppressor protein</u> cyclin-dependent kinase inhibitor 2A or CDKN2A or multiple tumor suppressor 1 (MTS-1)) and up regulation of <u>hTERT</u> (gene encoding human <u>telomerase reverse transcriptase</u> a <u>catalytic subunit</u> of the enzyme telomerase) and <u>PGC1-ar</u> genes (peroxisome proliferator-activated receptor G co-activator 10) [95,97,98].

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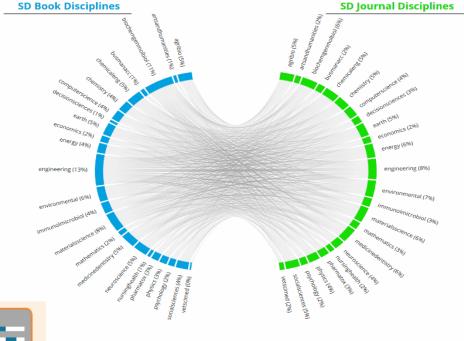
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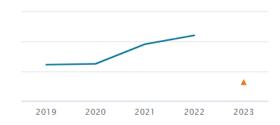
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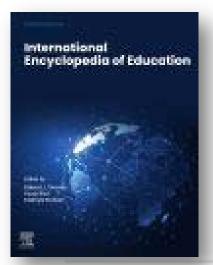
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