Aging

1-Mitochondria in health, disease, and aging

By Harrington, JS (Harrington, John S. S.) [1] ; Ryter, SW (Ryter, Stefan W. W.) [2] ; Plataki, M (Plataki, Maria) [1] ; Price, DR (Price, David R. R.) [1] ; Choi, AMK (Choi, Augustine M. K.) [1]**Source PHYSIOLOGICAL REVIEWS Volume 103 Issue 4 Page 2349-2422 DOI 10.1152/physrev.00058.2021 Published OCT 1 2023 Indexed 2023-08-08 Document Type Review**

Abstract

Mitochondria are well known as organelles responsible for the maintenance of cellular bioenergetics through the production of ATP. Although oxidative phosphorylation may be their most important function, mitochondria are also integral for the synthesis of metabolic precursors, calcium regulation, the production of reactive oxygen species, immune signaling, and apoptosis. Considering the breadth of their responsibilities, mitochondria are fundamental for cellular metabolism and homeostasis. Appreciating this significance, translational medicine has begun to investigate how mitochondrial dysfunction can represent a harbinger of disease. In this review, we provide a detailed overview of mitochondrial metabolism, cellular bioenergetics, mitochondrial dynamics, autophagy, mitochondrial damage - associated molecular patterns, mitochondria-mediated cell death pathways, and how mitochondrial dysfunction at any of these levels is associated with disease pathogenesis. Mitochondria-dependent pathways may thereby represent an attractive therapeutic target for ameliorating human disease.

Keywords

Author Keywords

inflammationmitochondriamitochondrial dynamicsmitochondrial dysfunctionmitophagy

Keywords Plus

NF-KAPPA-BPULMONARY ARTERIAL-HYPERTENSIONDYNAMIN-RELATED PROTEIN-1ENDOPLASMIC-RETICULUM STRESSPERMEABILITY TRANSITION POREFORMYL-PEPTIDE RECEPTORSTUMOR-NECROSIS-FACTORE3 UBIQUITIN LIGASEPOTENTIAL THERAPEUTIC TARGETDEFECTIVE AXONAL-TRANSPORT

Aging

2-Nonlinear dynamics of multi-omics profiles during human aging

By Shen, X (Shen, Xiaotao) [1] , [2] , [3] ; Wang, C (Wang, Chuchu) [4] , [5] ; Zhou, X (Zhou, Xin) [1] , [6] ; Zhou, W (Zhou, Wenyu) [1] ; Hornburg, D (Hornburg, Daniel) [1] ; Wu, S (Wu, Si) [1] ; Snyder, MP (Snyder, Michael P.) [1] , [6] (provided by Clarivate) **Source NATURE AGING Volume 4 Issue 11 Page 1619 DOI 10.1038/s43587-024-00692-2 Published NOV 2024 Early Access AUG 2024 Indexed 2024-08-18 Document Type Article**

Abstract

Aging is a complex process associated with nearly all diseases. Understanding the molecular changes underlying aging and identifying therapeutic targets for aging-related diseases are crucial for increasing healthspan. Although many studies have explored linear changes during aging, the prevalence of aging-related diseases and mortality risk accelerates after specific time points, indicating the importance of studying nonlinear molecular changes. In this study, we performed comprehensive multi-omics profiling on a longitudinal human cohort of 108 participants, aged between 25years and 75years. The participants resided in California, United States, and were tracked for a median period of 1.7years, with a maximum follow-up duration of 6.8years. The analysis revealed consistent nonlinear patterns in molecular markers of aging, with substantial dysregulation occurring at two major periods occurring at approximately 44years and 60years of chronological age. Distinct molecules and functional pathways associated with these periods were also identified, such as immune regulation and carbohydrate metabolism that shifted during the 60-year transition and cardiovascular disease, lipid and alcohol metabolism changes at the 40-year transition. Overall, this research demonstrates that functions and risks of aging-related diseases change nonlinearly across the human lifespan and provides insights into the molecular and biological pathways involved in these changes. Understanding the molecular changes underlying aging is important for developing biomarkers and healthy aging interventions. In this study, the authors used comprehensive multi-omics data to reveal nonlinear molecular profiles across chronological ages, highlighting two substantial variations observed around ages 40 and 60, which are linked to increased disease risks.

Keywords

Keywords Plus

CARDIOVASCULAR-DISEASEMETABOLISMBIOMARKERSACIDS

Aging

3-Challenges and recommendations for the translation of biomarkers of aging

By Herzog, CMS (Herzog, Chiara M. S.) [1] ; Goeminne, LJE (Goeminne, Ludger J. E.) [2] ; Poganik, JR (Poganik, Jesse R.) [2] ; Barzilai, N (Barzilai, Nir) [3] ; Belsky, DW (Belsky, Daniel W.) [4] ; Betts-LaCroix, J (Betts-LaCroix, Joe) [5] ; Chen, BH (Chen, Brian H.) [6] , [7] ; Chen, MCL (Chen, Michelle) [8] ; Cohen, AA (Cohen, Alan A.) [9] ; Cummings, SR (Cummings, Steven R.) [6] , [7] ; Group Author Biomarkers Aging Consortium (Biomarkers Aging Consortium) [1] (provided by Clarivate) **SourceNATURE AGING Volume 4 Issue 10 Page 1372-1383 DOI 10.1038/s43587-024-00683-3 Published OCT 2024 Early Access SEP 2024 Indexed 2024-09-23 Document Type Review**

Abstract

Biomarkers of aging (BOA) are quantitative parameters that predict biological age and ideally its changes in response to interventions. In recent years, many promising molecular and omic BOA have emerged with an enormous potential for translational geroscience and improving healthspan. However, clinical translation remains limited, in part due to the gap between preclinical research and the application of BOA in clinical research and other translational settings. We surveyed experts in these areas to better understand current challenges for the translation of aging biomarkers. We identified six key barriers to clinical translation and developed guidance for the field to overcome them. Core recommendations include linking BOA to clinically actionable insights, improving affordability and availability to broad populations and validation of biomarkers that are robust and responsive at the level of individuals. Our work provides key insights and practical recommendations to overcome barriers impeding clinical translation of BOA. Biomarkers of aging have potential to accelerate the clinical translation of interventions that promote healthy aging but are currently limited to research. The authors identify six barriers to be overcome to enable biomarker translation, providing a roadmap to clinical implementation.

Keywords

Keywords Plus

HEALTH-CAREMORTALITY

Aging

4-Organ aging signatures in the plasma proteome track health and disease

By Oh, HSH (Oh, Hamilton Se-Hwee) [1] , [2] , [3] ; Rutledge, J (Rutledge, Jarod) [2] , [3] , [4] ; Nachun, D (Nachun, Daniel) [5] ; Pálovics, R (Palovics, Robert) [2] , [3] , [6] ; Abiose, O (Abiose, Olamide) [3] , [6] ; Moran-Losada, P (Moran-Losada, Patricia) [2] , [3] , [6] ; Channappa, D (Channappa, Divya) [2] , [3] , [6] ; Urey, DY (Urey, Deniz Yagmur) [2] , [7] ; Kim, K (Kim, Kate) [2] , [3] , [6] ; Sung, YJ (Sung, Yun Ju) [8] , [9] ; (provided by Clarivate) Source NATURE Volume 624 Issue 7990 Page 164-+ DOI 10.1038/s41586-023-06802-1 Published DEC 7 2023 Indexed 2024-03-23 Document Type Article

Abstract

Animal studies show aging varies between individuals as well as between organs within an individual1-4, but whether this is true in humans and its effect on age-related diseases is unknown. We utilized levels of human blood plasma proteins originating from specific organs to measure organ-specific aging differences in living individuals. Using machine learning models, we analysed aging in 11 major organs and estimated organ age reproducibly in five independent cohorts encompassing 5,676 adults across the human lifespan. We discovered nearly 20% of the population show strongly accelerated age in one organ and 1.7% are multi-organ agers. Accelerated organ aging confers 20-50% higher mortality risk, and organ-specific diseases relate to faster aging of those organs. We find individuals with accelerated heart aging have a 250% increased heart failure risk and accelerated brain and vascular aging predict Alzheimer's disease (AD) progression independently from and as strongly as plasma pTau-181 (ref. 5), the current best blood-based biomarker for AD. Our models link vascular calcification, extracellular matrix alterations and synaptic protein shedding to early cognitive decline. We introduce a simple and interpretable method to study organ aging using plasma proteomics data, predicting diseases and aging effects.
Blood plasma protein data was combined with machine learning models for a simple method to determine differences in organ-specific aging; the study provides a basis for the prediction of diseases and aging effects using plasma proteomics.

Keywords

Keywords Plus

CLIMATE-CHANGE MITIGATIONLAND-USE CHANGEALZHEIMERS-DISEASEGLOBAL VEGETATIONABOVEGROUND BIOMASSSPATIALLY EXPLICITCARBON STOCKSSOIL CARBONFORESTWORLDS

Aging

5-The role of p21 in cellular senescence and aging-related diseases

By Yan, JY (Yan, Jiayu) [1] , [2] , [3] , [4] ; Chen, SY (Chen, Siyi) [1] , [2] , [3] , [4] ; Yi, ZM (Yi, Zimei) [1] , [2] , [3] , [4] ; Zhao, RW (Zhao, Ruowen) [1] , [2] , [3] , [4] ; Zhu, JY (Zhu, Jiayu) [1] , [2] , [3] , [4] ; Ding, SW (Ding, Shuwen) [1] , [2] , [3] , [4] ; Wu, JH (Wu, Junhua) [1] , [2] , [3] , [4] **(provided by Clarivate) Source MOLECULES AND CELLS Volume 47 Issue 11 DOI 10.1016/j.mocell.2024.100113 Article Number 100113 Published NOV 2024 Early Access OCT 2024 Indexed 2024-11-15 Document Type Review**

Abstract

During the aging process or disease progression, normal cells and tissues in the body undergo various stresses, leading to cell damage and the need for repair, adaptation, apoptosis, or defense responses. Cellular senescence is a key player in this process, influencing the rate of aging and disease progression. It can be triggered by different stress factors, resulting in irreversible cell cycle arrest and functional decline. Senescent cells often show high expression of cell cycle factors such as p21 and p16, which are involved in cell cycle arrest. p16 has long been recognized as a significant marker of aging. Recent evidence suggests that p21high cells and p16high cells represent distinct cell populations in terms of cell type, tissue location, accumulation kinetics, and physiological functions. This article focuses on recent advancements in understanding p21-dependent cellular senescence. It starts by providing an overview of the role of p21 in 3 primary cellular senescence phenotypes where it plays a crucial role. It then delves into the pathogenesis of diseases closely linked to p21dependent cellular senescence, particularly metabolic disorders and cardiovascular diseases. The article also discusses progress in p21-related animal models and outlines strategies for utilizing p21 to intervene in cellular senescence by delaying aging, eliminating senescent cells, and rejuvenating senescent cells. This review systematically examines the pathogenesis of p21-dependent cellular senescence, emphasizing its importance in studying aging heterogeneity and developing new senolytic therapies. It aims to stimulate future research on leveraging p21 to enhance the characteristics of senescent cells, allowing more precise methods for eliminating harmful senescent cells at the right time, thereby delaying aging and potentially achieving rejuvenation. (c) 2024 The Author(s). Published by Elsevier Inc. on behalf of Korean Society for Molecular and Cellular Biology. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

Keywords

Author Keywords

Aging-related diseasesCellular senescencep21SenolyticSenomorphic

Keywords Plus

CYCLIN-DEPENDENT KINASEP16(INK4A)-INDUCED SENESCENCEPULMONARY-FIBROSISSTEM-CELLSEXPRESSIONAPOPTOSISPROMOTESPATHWAYSTRESSPROLIFERATION

Aging

6-Kaempferol: Paving the path for advanced treatments in aging-related diseases

By Hussain, MS (Hussain, Md Sadique) [1] ; Altamimi, ASA (Altamimi, Abdulmalik Saleh Alfawaz) [2] ; Afzal, M (Afzal, Muhammad) [3] ; Almalki, WH (Almalki, Waleed Hassan) [4] ; Kazmi, I (Kazmi, Imran) [5] ; Alzarea, SI (Alzarea, Sami I.) [6] ; Gupta, G (Gupta, Gaurav) [7] , [8] ; Shahwan, M (Shahwan, Moyad) [8] , [9] ; Kukreti, N (Kukreti, Neelima) [10] ; Wong, LS (Wong, Ling Shing) [11] ;
(provided by Clarivate) Source EXPERIMENTAL GERONTOLOGY Volume 188 DOI 10.1016/j.exger.2024.112389 Article Number 112389 Published APR 2024 Early Access MAR 2024 Indexed 2024-05-08 Document Type Review

Abstract

Aging -related diseases (ARDs) are a major global health concern, and the development of effective therapies is urgently needed. Kaempferol, a flavonoid found in several plants, has emerged as a promising candidate for ameliorating ARDs. This comprehensive review examines Kaempferol's chemical properties, safety profile, and pharmacokinetics, and highlights its potential therapeutic utility against ARDs. Kaempferol's therapeutic potential is underpinned by its distinctive chemical structure, which confers antioxidative and anti-inflammatory properties. Kaempferol counteracts reactive oxygen species (ROS) and modulates crucial cellular pathways, thereby combating oxidative stress and inflammation, hallmarks of ARDs. Kaempferol's low toxicity and wide safety margins, as demonstrated by preclinical and clinical studies, further substantiate its therapeutic potential. Compelling evidence supports Kaempferol's substantial potential in addressing ARDs through several mechanisms, notably anti-inflammatory, antioxidant, and anti-apoptotic actions. Kaempferol exhibits a versatile neuroprotective effect by modulating various proinflammatory signaling pathways, including NF-kB, p38MAPK, AKT, and the beta-catenin cascade. Additionally, it hinders the formation and aggregation of beta-amyloid protein and regulates brain -derived neurotrophic factors. In terms of its anticancer potential, kaempferol acts through diverse pathways, inducing apoptosis, arresting the cell cycle at the G2/M phase, suppressing epithelialmesenchymal transition (EMT) -related markers, and affecting the phosphoinositide 3-kinase/protein kinase B signaling pathways. Subsequent studies should focus on refining dosage regimens, exploring innovative delivery systems, and conducting comprehensive clinical trials to translate these findings into effective therapeutic applications.

Keywords
Author Keywords
KaempferolAging-related disordersToxicityFlavonoidAntioxidantTherapeutic implications

Keywords Plus
PHASEOLUS-VULGARIS L.OXIDATIVE STRESSIN-VITROFLAVONOL GLYCOSIDESCELLULAR SENESCENCERAT MODELOSTEOBLAST DIFFERENTIATIONARTICULAR CHONDROCYTESMULTIDRUG-RESISTANCENETWORK PHARMACOLOGY

7-Mitochondria in oxidative stress, inflammation and aging: from mechanisms to therapeutic advances

By Xu, XY (Xu, Xieyang) [1] , [2] , [3] ; Pang, Y (Pang, Yan) [1] , [2] , [3] ; Fan, XQ (Fan, Xianqun) [1] , [2] , [3] (provided by Clarivate) **Source SIGNAL TRANSDUCTION AND TARGETED THERAPY Volume 10 Issue 1 DOI 10.1038/s41392-025-02253-4 Article Number 190 Published JUN 11 2025 Indexed 2025-06-18 Document Type Review**

Abstract

Mitochondria are the energy production centers in cells and have unique genetic information. Due to the irreplaceable function of mitochondria, mitochondrial dysfunction often leads to pathological changes. Mitochondrial dysfunction induces an imbalance between oxidation and antioxidation, mitochondrial DNA (mtDNA) damage, mitochondrial dynamics dysregulation, and changes in mitophagy. It results in oxidative stress due to excessive reactive oxygen species (ROS) generation, which contributes to cell damage and death. Mitochondrial dysfunction can also trigger inflammation through the activation of damage-associated molecular patterns (DAMPs), inflammasomes and inflammatory cells. Besides, mitochondrial alterations in the functional regulation, energy metabolism and genetic stability accompany the aging process, and there has been a lot of evidence suggesting that oxidative stress and inflammation, both of which are associated with mitochondrial dysfunction, are predisposing factors of aging. Therefore, this review hypothesizes that mitochondria serve as central hubs regulating oxidative stress, inflammation, and aging, and their dysfunction contributes to various diseases, including cancers, cardiovascular diseases, neurodegenerative disorders, metabolic diseases, sepsis, ocular pathologies, liver diseases, and autoimmune conditions. Moreover, we outline therapies aimed at various mitochondrial dysfunctions, highlighting their performance in animal models and human trials. Additionally, we focus on the limitations of mitochondrial therapy in clinical applications, and discuss potential future research directions for mitochondrial therapy.

Keywords

Keywords Plus

CYCLIC GMP-AMPACTIVATED PROTEIN-KINASEAGE-RELATED-CHANGESREACTIVE OXYGENNLRP3 INFLAMMASOMESKELETAL-MUSCLEGASDERMIN-DCELL-DEATHCOMPLEX-IDNA-DAMAGE

Aging

8-Functional Role of Taurine in Aging and Cardiovascular Health: An Updated Overview

By Santulli, G (Santulli, Gaetano) [1] , [2] ; Kansakar, U (Kansakar, Urna) [1] ; Varzideh, F (Varzideh, Fahimeh) [2] ; Mone, P (Mone, Pasquale) [2] ; Jankauskas, SS (Jankauskas, Stanislovas S.) [1] ; Lombardi, A (Lombardi, Angela) [1] (provided by Clarivate) **Source NUTRIENTS Volume 15 Issue 19 DOI 10.3390/nu15194236 Article Number 4236 Published OCT 2023 Indexed 2023-10-22 Document Type Review**

Abstract

Taurine, a naturally occurring sulfur-containing amino acid, has attracted significant attention in recent years due to its potential health benefits. Found in various foods and often used in energy drinks and supplements, taurine has been studied extensively to understand its impact on human physiology. Determining its exact functional roles represents a complex and multifaceted topic. We provide an overview of the scientific literature and present an analysis of the effects of taurine on various aspects of human health, focusing on aging and cardiovascular pathophysiology, but also including athletic performance, metabolic regulation, and neurological function. Additionally, our report summarizes the current recommendations for taurine intake and addresses potential safety concerns. Evidence from both human and animal studies indicates that taurine may have beneficial cardiovascular effects, including blood pressure regulation, improved cardiac fitness, and enhanced vascular health. Its mechanisms of action and antioxidant properties make it also an intriguing candidate for potential anti-aging strategies.

Keywords
Author Keywords
aging2-aminoethanesulfonic acidcardiovascular riskenergy drinksinflammationmetabolismoxidative stresssupplementstauric acidtaurine

Keywords Plus
SULFINIC ACID DECARBOXYLASEENDOPLASMIC-RETICULUM STRESSIMPROVES INSULIN SENSITIVITYCONGESTIVE-HEART-FAILURECARDIAC OXIDATIVE STRESSMESENCHYMAL STEM-CELLSDIETARY TAURINEBLOOD-PRESSUREENDOTHELIAL DYSFUNCTIONSECRETORY PHENOTYPE

Aging

9-Aging drives cerebrovascular network remodeling and functional changes in the mouse brain

By Bennett, HC (Bennett, Hannah C.) [1] ; Zhang, QG (Zhang, Qingguang) [2] , [3] ; Wu, YT (Wu, Yuanting) [1] , [5] ; Manjila, SB (Manjila, Steffy B.) [1] ; Chon, U (Chon, Uree) [1] , [6] ; Shin, D (Shin, Donghui) [1] ; Vanselow, DJ (Vanselow, Daniel J.) [1] ; Pi, HJ (Pi, Hyun-Jae) [1] ; Drew, PJ (Drew, Patrick J.) [2] , [4] ; Kim, Y (Kim, Yongsoo) [1] , [2] (provided by Clarivate) **Source NATURE COMMUNICATIONS Volume 15 Issue 1 DOI 10.1038/s41467-024-50559-8 Article Number 6398 Published JUL 30 2024 Indexed 2024-08-09 Document Type Article**

Abstract

Aging is frequently associated with compromised cerebrovasculature and pericytes. However, we do not know how normal aging differentially impacts vascular structure and function in different brain areas. Here we utilize mesoscale microscopy methods and in vivo imaging to determine detailed changes in aged murine cerebrovascular networks. Whole-brain vascular tracing shows an overall similar to 10% decrease in vascular length and branching density with similar to 7% increase in vascular radii in aged brains. Light sheet imaging with 3D immunolabeling reveals increased arteriole tortuosity of aged brains. Notably, vasculature and pericyte densities show selective and significant reductions in the deep cortical layers, hippocampal network, and basal forebrain areas. We find increased blood extravasation, implying compromised blood-brain barrier function in aged brains. Moreover, in vivo imaging in awake mice demonstrates reduced baseline and on-demand blood oxygenation despite relatively intact neurovascular coupling. Collectively, we uncover regional vulnerabilities of cerebrovascular network and physiological changes that can mediate cognitive decline in normal aging.

Keywords

Keywords Plus

CORTICAL BLOOD-FLOWCOGNITIVE IMPAIRMENTCHOLINERGIC CIRCUITSHEMODYNAMIC SIGNALSPERICYTE LOSSLAYER 6BCORTEXMICEBARRIERAGE

Aging

10-Sex and gender differences in cognitive resilience to aging and Alzheimer's disease

By Arenaza-Urquijo, EM (Arenaza-Urquijo, Eider M.) [1] , [2] ; Boyle, R (Boyle, Rory) [3] ; Casaletto, K (Casaletto, Kaitlin) [4] ; Anstey, KJ (Anstey, Kaarin J.) [5] , [6] , [7] ; Vila-Castelar, C (Vila-Castelar, Clara) [3] ; Colverson, A (Colverson, Aaron) [8] ; Palpatzis, E (Palpatzis, Eleni) [1] , [2] ; Eissman, JM (Eissman, Jaclyn M.) [9] , [10] ; Ng, TKS (Ng, Ted Kheng Siang) [11] , [12] ; Raghavan, S (Raghavan, Sheelakumari) [13] ; Group Authors Reserve Resilience & Protective Factors Profess Interest Area S (Reserve Resilience & Protective Factors Profess Interest Area S) ; ADDRESS Special Interest Grp (ADDRESS Special Interest Grp) (provided by Clarivate) **Source ALZHEIMERS & DEMENTIA Volume 20 Issue 8 Page 5695-5719 DOI 10.1002/alz.13844 Published AUG 2024 Early Access JUL 2024 Indexed 2024-07-18 Document Type Review**

Abstract

Sex and gender-biological and social constructs-significantly impact the prevalence of protective and risk factors, influencing the burden of Alzheimer's disease (AD; amyloid beta and tau) and other pathologies (e.g., cerebrovascular disease) which ultimately shape cognitive trajectories. Understanding the interplay of these factors is central to understanding resilience and resistance mechanisms explaining maintained cognitive function and reduced pathology accumulation in aging and AD. In this narrative review, the ADDRESS! Special Interest Group (Alzheimer's Association) adopted a multidisciplinary approach to provide the foundations and recommendations for future research into sex- and gender-specific drivers of resilience, including a sex/gender-oriented review of risk factors, genetics, AD and non-AD pathologies, brain structure and function, and animal research. We urge the field to adopt a sex/gender-aware approach to resilience to advance our understanding of the intricate interplay of biological and social determinants and consider sex/gender-specific resilience throughout disease stages.Highlights Sex differences in resilience to cognitive decline vary by age and cognitive status. Initial evidence supports sex-specific distinctions in brain pathology. Findings suggest sex differences in the impact of pathology on cognition. There is a sex-specific change in resilience in the transition to clinical stages. Gender and sex factors warrant study: modifiable, immune, inflammatory, and vascular.

Keywords
Author Keywords
brain maintenancecardiovascularcognitive declinecognitive reserveeducationgeneticsinequalitieslifestyleTDP43

Keywords Plus
APOLIPOPROTEIN-E GENOTYPESMALL VESSEL DISEASEWHITE-MATTERRISK-FACTORSBRAIN STRUCTUREAPOE EPSILON-4OLDER-ADULTSPERIVASCULAR SPACESCORTICAL THICKNESSPHYSICAL-ACTIVITY

11-H3K18 lactylation of senescent microglia potentiates brain aging and Alzheimer's disease through the NF?B signaling pathway

By Wei, L (Wei, Lin) [1] , [2] ; Yang, XW (Yang, Xiaowen) [2] ; Wang, J (Wang, Jie) [2] ; Wang, ZX (Wang, Zhixiao) [2] ; Wang, QG (Wang, Qiguang) [2] ; Ding, Y (Ding, Yan) [2] ; Yu, AQ (Yu, Aiqing) [1] , [2]
Source JOURNAL OF NEUROINFLAMMATION Volume 20 Issue 1 DOI 10.1186/s12974-023-02879-7 Article Number 208 Published SEP 11 2023 Indexed 2023-09-25 Document Type Article

Abstract

Cellular senescence serves as a fundamental and underlying activity that drives the aging process, and it is intricately associated with numerous age-related diseases, including Alzheimer's disease (AD), a neurodegenerative aging-related disorder characterized by progressive cognitive impairment. Although increasing evidence suggests that senescent microglia play a role in the pathogenesis of AD, their exact role remains unclear. In this study, we quantified the levels of lactic acid in senescent microglia, and hippocampus tissues of naturally aged mice and AD mice models (FAD(4T) and APP/PS1). We found lactic acid levels were significantly elevated in these cells and tissues compared to their corresponding counterparts, which increased the level of pan histone lysine lactylation (Kla). We aslo identified all histone Kla sites in senescent microglia, and found that both the H3K18 lactylation (H3K18la) and Pan-Kla were significantly up-regulated in senescent microglia and hippocampus tissues of naturally aged mice and AD modeling mice. We demonstrated that enhanced H3K18la directly stimulates the NF?B signaling pathway by increasing binding to the promoter of Rela (p65) and NF?B1(p50), thereby upregulating senescence-associated secretory phenotype (SASP) components IL-6 and IL-8. Our study provides novel insights into the physiological func-tion of Kla and the epigenetic regulatory mechanism that regulates brain aging and AD. Specifically, we have identi-fied the H3K18la/NF?B axis as a critical player in this process by modulating IL-6 and IL-8. Targeting this axis may be a potential therapeutic strategy for delaying aging and AD by blunting SASP.

Keywords
Author Keywords
Senescent microgliaNF & kappa;BBrain agingAlzheimer's disease

Aging

12-Effect of Solution and Aging Treatment on the Microstructure and Properties of LAZ931 Mg-Li Alloy by Friction Stir Processing

By Fang, Z (Fang, Zhe) [1] , [2] ; Xu, SW (Xu, Shuaiwei) [1] ; Wang, ZX (Wang, Zhixin) [1] ; Sun, YF (Sun, Yufeng) [2] **(provided by Clarivate) Source METALS Volume 15 Issue 3 DOI 10.3390/met15030314 Article Number 314 Published MAR 13 2025 Indexed 2025-04-01 Document Type Article**

Abstract

Heat treatment processes play a pivotal role in optimizing the microstructure and mechanical properties of Mg-Li alloys, thereby enhancing their performance and expanding their potential applications in structural and lightweight engineering fields. In this study, the influence of solution and aging treatments on the microstructure, phase transformation, and microhardness of friction-stir-processed (FSPed) LAZ931 Mg-Li alloy was investigated to obtain the optimal solution treatment temperature and time. An optimal solution treatment at 460 degrees C for 0.5 h under an Ar gas atmosphere facilitated complete alpha-phase dissolution with subsequent aging at 125 degrees C, triggering precipitation-mediated hardening. An X-ray diffraction (XRD) analysis identified a new MgLi2Al phase in the stirring zone (SZ) in addition to the alpha, beta, and AlLi phases. Aging kinetics at 125 degrees C showed that SZ hardness increased to 110.5 HV after solution treatment, which was 5.3% higher than the base metal (BM). After 3 h of aging, microhardness peaked at 86.5 HV before decreasing due to the decomposition of the metastable MgLi2Al phase into the stable AlLi phase. The microhardness stabilized at around 78 HV, which was 16.2% higher than that of the original SZ. These experimental results provide a fundamental understanding of property structure for meeting the growing demand for lightweight materials and improving material properties.

Keywords

Author Keywords

LAZ931 Mg-Li alloyfriction stir processingsolution aging treatmentmicrostructuremicrohardness

Keywords Plus

MECHANICAL-PROPERTIESHEAT-TREATMENT

Aging

13-Intelligent rehabilitation in an aging population: empowering human-machine interaction for hand function rehabilitation through 3D deep learning and point cloud

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By Xing, ZZ (Xing, Zhizhong) [1] , [2] , [3] ; Meng, ZJ (Meng, Zhijun) [2] ; Zheng, GF (Zheng, Gengfeng) [3] ; Ma, GL (Ma, Guolan) [4] , [5] ; Yang, L (Yang, Lin) [6] , [7] ; Guo, XJ (Guo, Xiaojun) [8] ; Tan, L (Tan, Li) [1] ; Jiang, YQ (Jiang, Yuanqiu) [1] ; Wu, HD (Wu, Huidong) [1] (provided by Clarivate) **Source FRONTIERS IN COMPUTATIONAL NEUROSCIENCE Volume 19 DOI 10.3389/fncom.2025.1543643 Article Number 1543643 Published MAY 2 2025 Indexed 2025-05-20 Document Type Article**

Abstract

Human-machine interaction and computational neuroscience have brought unprecedented application prospects to the field of medical rehabilitation, especially for the elderly population, where the decline and recovery of hand function have become a significant concern. Responding to the special needs under the context of normalized epidemic prevention and control and the aging trend of the population, this research proposes a method based on a 3D deep learning model to process laser sensor point cloud data, aiming to achieve non-contact gesture surface feature analysis for application in the field of intelligent rehabilitation of human-machine interaction hand functions. By integrating key technologies such as the collection of hand surface point clouds, local feature extraction, and abstraction and enhancement of dimensional information, this research has constructed an accurate gesture surface feature analysis system. In terms of experimental results, this research validated the superior performance of the proposed model in recognizing hand surface point clouds, with an average accuracy of 88.72%. The research findings are of significant importance for promoting the development of non-contact intelligent rehabilitation technology for hand functions and enhancing the safe and comfortable interaction methods for the elderly and rehabilitation patients.

Keywords

Author Keywords

3D perceptionneural networkhuman-machine interactiondeep learningnon-contact rehabilitation