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Short Communication

POSTCOVID-19 WAR era, age-related diseases caused by trace elements and heavy metals' exposure could affect accelerated excessive morbidity and mortality rates

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Abstract

In these POSTCOVID-19 war periods, worldwide, mortality rates for 2025 show a projected death rate of 8 deaths per 1000 population based on Macrotrends' historical data, a slight increase from 2024. In the European Union, accelerated excessive mortality rates were observed in the first half of 2025, according to the WHO statistics. There are several causes that directly highlight the main inducers and initiators that may play a pivotal role in accelerating excessive morbidity and mortality rates, as previously described (Badlou BA et al. 2018-2025, 1-4). Though almost all relevant and exact mechanisms were not investigated appropriately, elucidating novel action metabolisms of death receptor activities needs to be elucidated.

Key words: diseases; cells; stroke; cancer

Introduction

What is known? Globally, noncommunicable diseases (NCDs), especially ischemic heart disease, remain the leading cause of death, responsible for most premature deaths worldwide, particularly in low- and middle-income countries. These chronic conditions—including stroke, cancer, chronic respiratory diseases, and diabetes—are driven by factors such as underdeveloped countries and their populations, which are more quickly affected by aging- related diseases, collateral damage from routine lifestyle behaviors, and manipulated by tobacco and alcohol (ab)use. On the other hand, an increasing number of individuals suffering from obesity, unhealthy diets, and physical inactivity—issues that could be addressed through stronger public health policies—may be the main causes during this POSTCOVID-19 period. Furthermore, certain forms of medical discrimination, limited access to Medicare and Medicaid, selective prognostics and diagnostics, and deviations in specific know-how are other aspects that are known yet often neglected and denied by those in control.

What is unknown? There are still many angles of H&D aspects that remain unknown (2025). For instance, how long could COVID-19 patients be cured in less than a day? How could the NCDs be prevented ASAP, and appropriately? BBADVIES and RESEARCH team (BBART) initiated a couple of pilot studies to unravel certain cause-and-effect relationships of processes causing accelerated excessive morbidity and mortality rates in the last 4- 5 years. [1-4] Besides, BBRT's acquired research studies and data processed and presented offline and online to peer reviewers and tried to develop new and novel pro- and diagnostics approaches for our clients and patients, worldwide. While a specific 2025 global mortality report isn't available, aging populations are the primary driver for the rising number of Alzheimer's deaths worldwide. The progression of neurodegeneration from cell death, influenced by a combination of genetic, lifestyle, and environmental factors like hypertension, diabetes, and lack of physical activity, causes Alzheimer's. Alzheimer's is a form of dementia caused by the build-up of abnormal amyloid and tau proteins in the brain. The leading causes of dementia are aging, but also lifestyle and environmental factors, including unhealthy diet, smoking, harmful alcohol (ab)use, chronic inactivity, traumatic depression, loneliness and significant social isolation, and finally, environmental air pollution. Besides, our recent BBART pilot studies of 15 random clients are also indicating/ confirming that the heavy metal exposure (HME) is the main cause of the pathologies. HME is increasingly recognized as a contributor to aging-related diseases, particularly through its impact on kidney and liver function, protein synthesis, and amino acid metabolism. Breakdown of how these toxicants affect biological systems in subjects is already described (refs 4-8). The

impact of HME on aging-related disease is generally assumed on the direct initiation of peroxidation and aging of different organs, tissues, cells, and associated molecules irreversibly. Chronic exposure to heavy metals like lead, cadmium, mercury, and arsenic, as described, accelerates cellular aging by promoting oxidative stress and (pro-)inflammatory responses (refs4-7???). Besides, HME can disrupt mitochondrial function, prevent cytochrome functions, cause genetic damage, and halt DNA repair mechanisms, leading to age-related (ir)reversible pathologies, chronically. Although the impact of HME on liver and renal function in the older adult differs from that of adolescents and adults. Sia Pan et al. 2024 research study suggests that elevated levels of mixed metals exposure are linked to proteinuria, BUN, and chronic kidney disease (CKD), with Uric Acid serving as a mediating factor. (ref 4) Future studies and breakdowns in detail from the organism toward organs, molecular, and cellular levels might step-by-step help to understand basically and clinically why? How? Which mechanisms play a pivotal role in pathophysiological and age-related diseases progressions, directly? Furthermore, Tubular Damages are assumed to be related to HME, which might cause acute tubular necrosis and impair glomerular filtration rate (GFR), which naturally declines with age, and stress-associated peroxidation and proinflammatory responses, however. On the other hand, disorders in Uric Acid(UA) Mediation and metabolism might play either an independent or direct role in mediating kidney damage from mixed HME and affected kidney and liver. Moreover, simultaneous excessive and accelerated proteinuria, increasing both BUN and CKD progressions, and GFR failure that could cause different pathologic stone-forming, loss of carbohydrates, and vital lipoproteins, which are unequivocally important factors in age- related hemostasis and regeneration.Chronic exposure to different electrical fields and Biomagnetic fields (5Gs) might have proapoptotic and proinflammatory side effects, initiating certain (un)known damages on the kidney and liver tissues/metabolism, which are not elucidated yet. On the other hand, heavy metals disrupt liver enzyme activity and impair detoxification pathways. (refs) Cadmium and mercury can induce hepatotoxicity by altering redox balance and triggering apoptosis. (refs) Besides, because liver (dys)functioning affects amino acid metabolism and hepatic protein synthesis, however, dysfunctions could compound aging-related decline in elderly and sensitive subjects. Concerning Protein Synthesis and Amino Acid Release also affected after hepatic exposure to increased levels of heavy metals in systemic circulation. Furthermore, inhibition of Ribosomal function could be affected as well. Metals like mercury and arsenic interfere with ribosomal RNA, reducing protein synthesis significantly, when elderly subjects are chronically exposed to different traumatic and synthetic noxious i.e. electrical and magnetic fields. However, when amino acid production and release are chronically got in imbalanced processes, then liver and kidney damage in subjects, is inevitable. Chronic HME and traumatic distress conditions can impair amino acid recycling and release, affecting muscle maintenance and immune function, as well. Selenium and Manganese; these elements may counteract some toxic effects and support protein synthesis, indeed if they get bioavailable in systemic circulation, appropriately. Hypothetically, physiological processes are (in)directly depending on vitality and functional tissues and cells in a random body. On the other hand, bioavailability of internal and external factors can play a pivotal role in a balanced, healthy subject. Age-related deterioration of tissue integrity and cellular (ir)responsivity can provide certain infrastructures that are needed for a healthy body. Controversially, 30-plus subjects mostly are suffering from a significant decrease in stem cell regeneration and renewal, aging-associated, remarkably. [4-8] Recent studies raised evidence-based indications that indeed metabolic dysfunctions, which are correlated to HME in the environment, chronically, induce speculatively considerable public health challenges, worldwide. [5-7] The mediating effect of biological aging on the association between metal exposure and the risk of metabolic dysfunction correlated to kidney and liver diseases, while urinary Ba concentration was negatively correlated with phenotypic age and biological age. [5] Moreover, heavy metals have long been regarded as important environmental pollutants due to their toxicity and persistence in ecosystems, might affecting neurodegenerative diseases, and some unknown metabolic processes, resulting in accelerated age-related morbidities, as well. [1-8] Taken together, chronic HME can deter liver and kidney metabolism (essential organs for metabolic compensations) of the elderly and could cause significant decay of subjects if they don't get timely and appropriate Medicare and Medicaid. Further investigation is needed to unravel how liver and kidney protein metabolism and amino acid metabolism could be restored appropriately. Chronic HME and might some still unknown pathogens affect death receptors to become activated, which the main clinical mechanism is not still elucidated completely.

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