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Traffic-Derived Metal Bioaccumulation in Bone Tissue as a Driver of Osteoporosis

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Extended Abstract

Long term air pollution exposures, such as fine particulate matter (PM), are a well-established risk factor in the development of adverse cardiopulmonary outcomes [1]. However, recent studies have expanded this scope to include a wider range of health conditions such as osteoporosis and bone fragility [2]. This is particularly concerning given the global impact of osteoporosis, affecting 500 million people and resulting in 13.5 million fractures a year [3]. While there is extensive research into traditional sources of air pollution, such as exhaust emissions [4], the role of other sources, including brake, tire and road wear collectively termed Non-exhaust Emissions (NEE), remains largely overlooked. NEE contributions have recently become a critical environmental concern by the planned phasing out of the combustion engine and adoption of electric vehicles (EVs) as countries meet their NetZero commitments [5]. This change fails to consider the potential rise in non-exhaust emissions from the heavier EVs [6]. We hypothesize that long term accumulation of metals from non-exhaust traffic emissions plays a role in the onset of osteoporosis. Several lines of evidence support this hypothesis: epidemiological studies linking

air pollution exposure to increased fracture risk, particularly in populations near busy roads; clinical studies identifying metal accumulation in osteoporotic bone; and molecular studies highlighting how metals interfere with the bone remodeling process. To establish case and effect several mechanisms of toxicity potentially explaining this relationship are suggested: metals accumulating in bone by substituting calcium in hydroxyapatite; metals generating reactive oxygen species (ROS), downregulating the Wnt/β-catenin signaling pathway and metals disrupting endocrine and renal function promoting bone loss through upregulated estrogen metabolism and elevated parathyroid hormone (PTH). To test this hypothesis, a research method that integrates in vitro, in vivo, ex vivo and computational modeling techniques is proposed. In vitro studies analyzing the effects of individual and combinations of metals on osteoblasts and osteoclasts to examine the changes in markers of bone formation, resportion and matrix production. In vivo studies that expose mouse models to various sources traffic-related PM evaluating how bioaccumulation of metal affects bone structure. Computational methods that use mathematical models to predict the long-term metal induced changes in bone. Ex vivo human studies using inductively coupled plasma mass spectrometry (ICP-MS) and micro-CT imaging to respectively, provide precise quantification of metals which are known to bioaccumulate in bone and investigate changes in trabecular thickness and connectivity comparing the bone of those living in high and low pollution environments. By understanding the mechanisms underpinning air pollution-induced bone damage this research could go on to inform significant policy changes such as: revised vehicle emission standards that consider non-exhaust sources, wider adoption of cars designed to meet Euro 7 emissions standards, urban planning strategies that reduce exposure to traffic-related metals and enhanced monitoring of bone health in populations living near high traffic areas.

References

[1] Chen, J. and G. Hoek, "Long-term exposure to PM and all-cause and cause-specific mortality: A systematic review and meta-analysis," Environ. Int., vol. 143, p. 105974, 2020.

[2] S. R. Mousavibaygei, A. Bisadi, and F. ZareSakhvidi, "Outdoor air pollution exposure, bone mineral density, osteoporosis, and osteoporotic fractures: A systematic review and meta-analysis," Sci. Total Environ., vol. 865, p. 161117, 2023.

[3] M. K. Javaid, R. Pinedo-Villanueva, A. Shah, Z. Mohsin, M. Hiligsmann, A. Motek-Soulié, N. R. Fuggle, P. Halbout, and C. Cooper, "The Capture the Fracture® Partnership: an overview of a global initiative to increase the secondary fracture prevention care for patient benefit," Osteoporos. Int., vol. 34, no. 11, pp. 1827–1835, 2023.

[4] N. A. Janssen, G. Hoek, M. Simic-Lawson, P. Fischer, L. van Bree, H. ten Brink, M. Keuken, R. W. Atkinson, H. R. Anderson, B. Brunekreef, and F. R. Cassee, "Black carbon as an additional indicator of the adverse health effects of airborne particles compared with PM10 and PM2.5," Environ. Health Perspect., vol. 119, no. 12, pp. 1691–1699, 2011.

[5] H. J. Jin, Y. K. Bae, M. Kim, S. J. Kwon, H. B. Jeon, S. J. Choi, S. W. Kim, Y. S. Yang, W. Oh, and J. W. Chang, "Comparative analysis of human mesenchymal stem cells from bone marrow, adipose tissue, and umbilical cord blood as sources of cell therapy," Int. J. Mol. Sci., vol. 14, no. 9, pp. 17986–18001, 2013.

[6] V. R. J. H. Timmers and P. A. J. Achten, "Non-exhaust PM emissions from electric vehicles," Atmos. Environ., vol. 134, pp. 10–17, 2016.