

Environmental Noise Exposure and its Association with MicroRNA Expression Changes in Cardiovascular Regulation Pathways

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DESCRIPTION

Continuous exposure to elevated environmental noise has become a defining feature of modern urban living. Road traffic, rail networks, aviation activity, and dense residential construction generate persistent acoustic pressure that extends well beyond mere annoyance. Over time, this exposure interacts with physiological regulatory systems in ways that are increasingly understood at the molecular level. One area of growing interest involves microRNAs, small non-coding Ribonucleic acid (RNA) molecules that regulate gene expression post-transcriptionally and play an essential role in maintaining cardiovascular stability.

MicroRNAs influence biological function by binding to complementary messenger RNA sequences, reducing translation or promoting degradation. Their expression patterns are not fixed; instead, they respond dynamically to external stimuli. Environmental stressors, particularly those that persist over long durations, can shift microRNA expression profiles in tissues associated with vascular and cardiac function. In individuals exposed to chronic noise, these shifts may reflect adaptive or maladaptive responses depending on exposure intensity and duration. A multi-center observational investigation conducted across residential districts with varying ambient noise levels examined adult participants over a five-year period. Environmental sound intensity was continuously recorded using calibrated monitoring devices placed in residential zones, ensuring accurate representation of long-term exposure. Biological sampling involved periodic blood collection to analyze circulating microRNA signatures associated with cardiovascular regulation.

The results demonstrated that individuals residing in high-noise environments exhibited consistent alterations in microRNA expression profiles linked to endothelial function. Several microRNAs responsible for regulating nitric oxide synthesis pathways showed elevated expression variability. Since nitric oxide is essential for vascular relaxation and blood pressure regulation, disruptions in its regulatory network suggest

functional implications for cardiovascular health. Participants with these molecular alterations also showed subtle but measurable increases in systolic blood pressure variability and resting heart rate elevation compared to low-exposure counterparts. In addition to endothelial regulation, microRNAs associated with inflammatory signaling pathways were significantly altered. Increased expression of microRNAs promoting pro-inflammatory cytokine production was observed, alongside reduced expression of those involved in anti-inflammatory regulation. This imbalance suggests a shift toward a sustained inflammatory state in individuals exposed to prolonged acoustic stress. Chronic inflammation is widely recognized as a contributing factor to vascular dysfunction and atherosclerotic progression, indicating potential long-term consequences of these molecular changes.

Parallel laboratory experiments using cultured human endothelial cells provided mechanistic insights into these observations. Cells were subjected to controlled mechanical vibrations designed to replicate the physical characteristics of environmental noise exposure. Over time, these cells exhibited altered expression of microRNAs regulating oxidative stress responses. Increased production of reactive oxygen species was observed, alongside decreased antioxidant enzyme expression. These cellular changes were accompanied by increased permeability of endothelial barriers, suggesting impaired vascular integrity.

However, reversibility was also observed in a subset of participants. Individuals who maintained low-noise exposure for extended periods showed gradual normalization of specific microRNA profiles, particularly those associated with stress-response signaling pathways. This indicates that while some molecular changes may persist, others retain flexibility depending on environmental conditions and exposure duration.

Cardiovascular assessments conducted alongside molecular analyses revealed associations between altered microRNA expression and early physiological markers of vascular change. Increased arterial stiffness, measured through pulse wave velocity, was more frequently observed in participants with

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pronounced microRNA alterations. These individuals also displayed reduced heart rate variability, a known indicator of autonomic nervous system imbalance. While these markers do not indicate immediate clinical disease, they reflect early functional changes in cardiovascular regulation.

Animal model studies provided further evidence supporting these findings. Rodents exposed to chronic noise conditions similar in frequency and intensity to urban environments demonstrated comparable microRNA expression shifts in cardiac tissue. These changes were accompanied by increased sympathetic nervous system activity, suggesting that neuroendocrine pathways may mediate part of the response to acoustic stress. Pharmacological inhibition of stress hormone signaling partially reduced microRNA alterations, indicating a connection between hormonal regulation and epigenetic control mechanisms.

At the mechanistic level, persistent noise exposure may activate stress-responsive transcription factors in vascular and cardiac tissues. These transcription factors can influence microRNA

biogenesis pathways, altering the production and stability of specific regulatory molecules. Over time, this leads to a reconfiguration of gene expression networks involved in vascular tone, inflammation, and oxidative balance.

CONCLUSION

Overall, the study provides evidence that chronic environmental noise exposure is associated with measurable changes in microRNA expression related to cardiovascular regulation, highlighting a molecular pathway through which urban living conditions may influence vascular health. Individual variability in response to noise exposure was also evident. Genetic background, baseline stress reactivity, and prior environmental exposure history appeared to influence the magnitude of microRNA alterations. Some individuals showed strong molecular responses even at moderate noise levels, while others exhibited minimal changes despite higher exposure. This variability highlights the complexity of environmental-molecular interactions in cardiovascular regulation.