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SuperGreen protects against pollution-induced metabolic dysfunction

In this preclinical study, we evaluated the protective effects of long-term SuperGreen supplementation on mitochondrial function, oxidative stress, and inflammation following exposure to air pollution. SuperGreen supplementation restored cellular energy production, reduced markers of oxidative damage, and suppressed pro-inflammatory pathways in the body. These findings suggest that SuperGreen is a promising nutritional intervention for alleviating the adverse effects of air pollution exposure on the body's metabolic and inflammatory responses.

Background

Air pollution is an environmental challenge that not only stresses the lungs but also places hidden strain on the rest of the body's organ systems, disrupting metabolism and mitochondrial function. It can exacerbate asthma, osteoporosis, and insulin resistance, with individuals living in urban areas or with existing respiratory conditions being especially vulnerable [1–4].

Among potential nutritional interventions, compounds with antioxidant and anti-inflammatory activity—such as chlorophyll derivatives—have emerged as promising candidates for supporting metabolic resilience. SuperGreen is a dietary supplement containing sodium copper chlorophyllin (SCC), a stable and bioavailable form of chlorophyll, which has been studied for its anti-inflammatory, antibacterial, and antioxidant benefits. This study aimed to evaluate whether SuperGreen could help mitigate the adverse effects of air pollution exposure on the lungs, adipose tissue, muscle, and overall health.

Methods

Healthy male and female mice were randomly assigned to one of four conditions (n=8 per group): control (CON), SuperGreen (SG), diesel particulate matter exposure (DPM), and DPM exposure with SG (DPM + SG). After four weeks, mitochondrial respirometry, ATP quantification, redox, inflammation, and tissue histology assays were performed. This study was conducted in accordance with established ethical standards and approved by the Institutional Animal Care and Use Committee at Brigham Young University (protocol #20-0203).

Data are presented as the means ± SD. The differences between treatment groups were assessed by one- and two-way analysis of variance (ANOVA). When ANOVA indicated significant differences, student's t-tests were used with Bonferroni correction for multiple comparisons. Significance was determined as being at $p \leq 0.05$.

	32 mice	4 different groups	4 weeks

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Results

Energy Production and Oxidative Stress in the Lungs
In the lungs, exposure to diesel particulate matter (DPM) significantly reduces cellular energy production (ATP), and increases reactive oxygen species (ROS) production. However, drinking SuperGreen (DPM+SG) reversed both effects of pollution exposure (Figure 1).

Figure 1

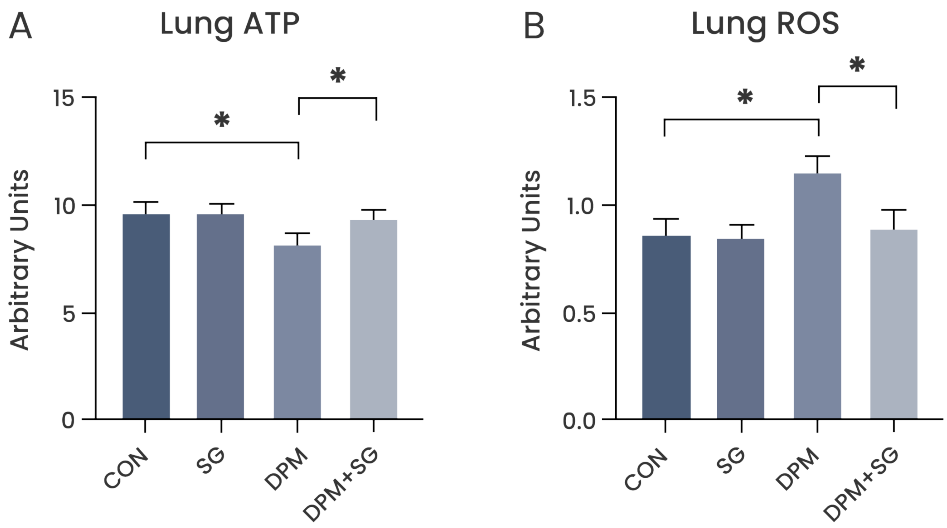

Figure 1. Pollution exposure resulted in a decrease in ATP alongside an increase in ROS in the lungs. SuperGreen exerted a protective effect in the midst of pollution exposure. (ATP: adenosine triphosphate, the primary unit of cellular energy; ROS: reactive oxygen species, compounds that contribute to oxidative stress in the body; CON: control, SG: SuperGreen, DPM: pollution exposure, and DPM+SG: pollution exposure and SuperGreen. *p ≤ 0.05)

Energy Production and Oxidative Stress in Muscle Tissue

Similarly, SuperGreen protected skeletal muscle cells from ROS production and partially restored cellular energy levels (Figure 2). This trend suggests that SuperGreen may exert protective effects on mitochondrial function, likely through mechanisms involving improved bioenergetic efficiency and reduced oxidative stress.

Systemic Inflammation

Pollution exposure markedly increased circulating pro-inflammatory signals (IL-6, MCP-1, TNF-α, and IL-1β), indicating a strong inflammatory response. This response was dampened by drinking SuperGreen, suggesting an anti-inflammatory effect.

Figure 2

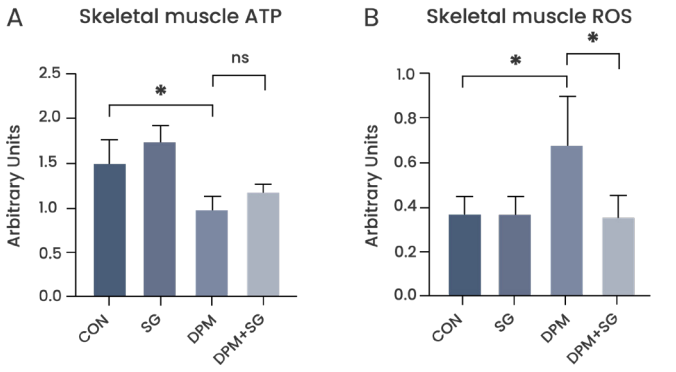

Figure 2. ATP levels were significantly reduced while ROS were elevated in skeletal muscle due to pollution exposure. SuperGreen exerted a protective effect from the ROS produced as a result of pollution exposure. (ATP: adenosine triphosphate, the primary unit of cellular energy; ROS: reactive oxygen species, compounds that contribute to oxidative stress in the body; CON: control, SG: SuperGreen, DPM: pollution exposure and DPM+SG: pollution exposure and SuperGreen. *p ≤ 0.05, ns: non-significant.)

Figure 3

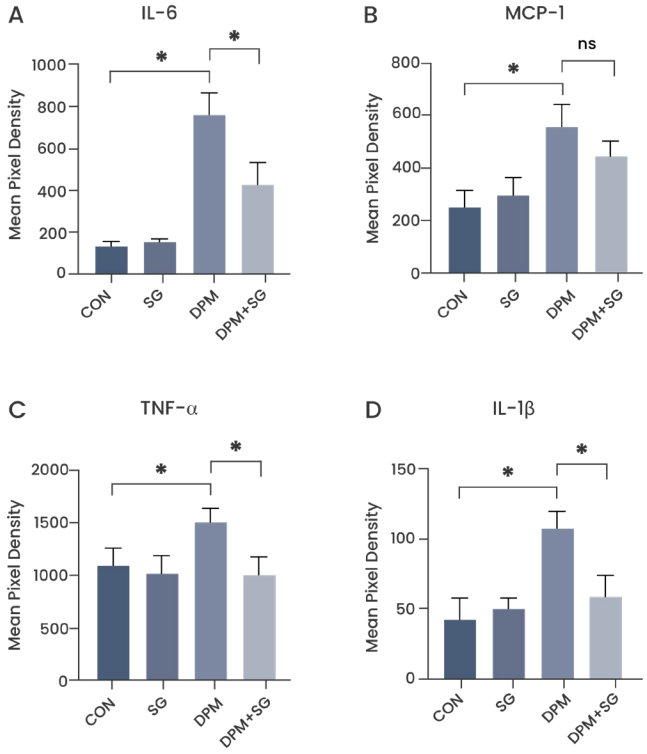

Figure 3. Drinking SuperGreen significantly lowered several key markers of systemic inflammation despite DPM exposure. (CON: control, SG: SuperGreen, DPM: pollution exposure, and DPM+SG: pollution exposure and SuperGreen. *p ≤ 0.05, ns: non-significant.)

Adipose Tissue Structure

Pollution exposure led to enlarged fat cells, which are more prone to insulin resistance, inflammation, and metabolic stress. However, drinking SuperGreen ameliorated this effect and resulted in noticeably smaller fat deposits (data not shown). These results suggest that SuperGreen may help protect against pollution-driven changes in fat tissue, supporting healthier metabolic function in the face of environmental stress.

Conclusion

SuperGreen is a functional nutritional intervention that helps protect the body against environmental stress and inflammation. In this study, long-term SuperGreen intake mitigated the harmful effects of pollution exposure by restoring energy production, reducing oxidative stress, blunting the inflammatory response, and normalizing fat cell size. These findings highlight SuperGreen's ability to defend against pollution-induced damage, offering a practical approach to support respiratory, muscular, and metabolic health.

References

1. McCook, J. P., Stephens, T. J., Jiang, L. I., Law, R. M., & Gotz, V. (2016). Ability of sodium copper chlorophyllin complex to repair photoaged skin by stimulation of biomarkers in human extracellular matrix. Clinical, Cosmetic and Investigational Dermatology, 9, 167–174. https://doi.org/10.2147/CCID.S111139
2. Liu, W., Xu, Z., & Yang, T. (2018). Health Effects of Air Pollution in China. International Journal of Environmental Research and Public Health, 15(7), 1471. https://doi.org/10.3390/ijerph15071471
3. Sun, H., Wan, Y., Pan, X., You, W., Shen, J., Lu, J., Zheng, G., Li, X., Xing, X., & Zhang, Y. (2024). Long-term air pollution and adverse meteorological factors might elevate the osteoporosis risk among adult Chinese. Frontiers in Public Health, 12. https://www.frontiersin.org/journals/public-health/articles/10.3389/fpubh.2024.1361911
4. Zhang, Y., Yang, X., Jiang, W., Gao, X., Yang, B., Feng, X. L., & Yang, L. (2024). Short-term effects of air pollutants on hospital admissions for asthma among older adults: A multi-city time series study in Southwest, China. Frontiers in Public Health, 12. https://www.frontiersin.org/journals/public-health/articles/10.3389/fpubh.2024.1346914

Read the publication here: https://pmc.ncbi.nlm.nih.gov/articles/PMC11858186/

Radford, J. H., Evans, E. P., Edwards, I. T., Arroyo, J. A., Bikman, B. T., & Reynolds, P. R. (2025). Diesel Particulate Matter (DPM)-Induced Metabolic Disruption in Mice Is Mitigated by Sodium Copper Chlorophyllin (SCC). Nutrients, 17(4), 717. https://doi.org/10.3390/nu17040717