

The potential role of mercury exposure and n-3 polyunsaturated fatty acids on systemic lupus erythematosus pathogenesis

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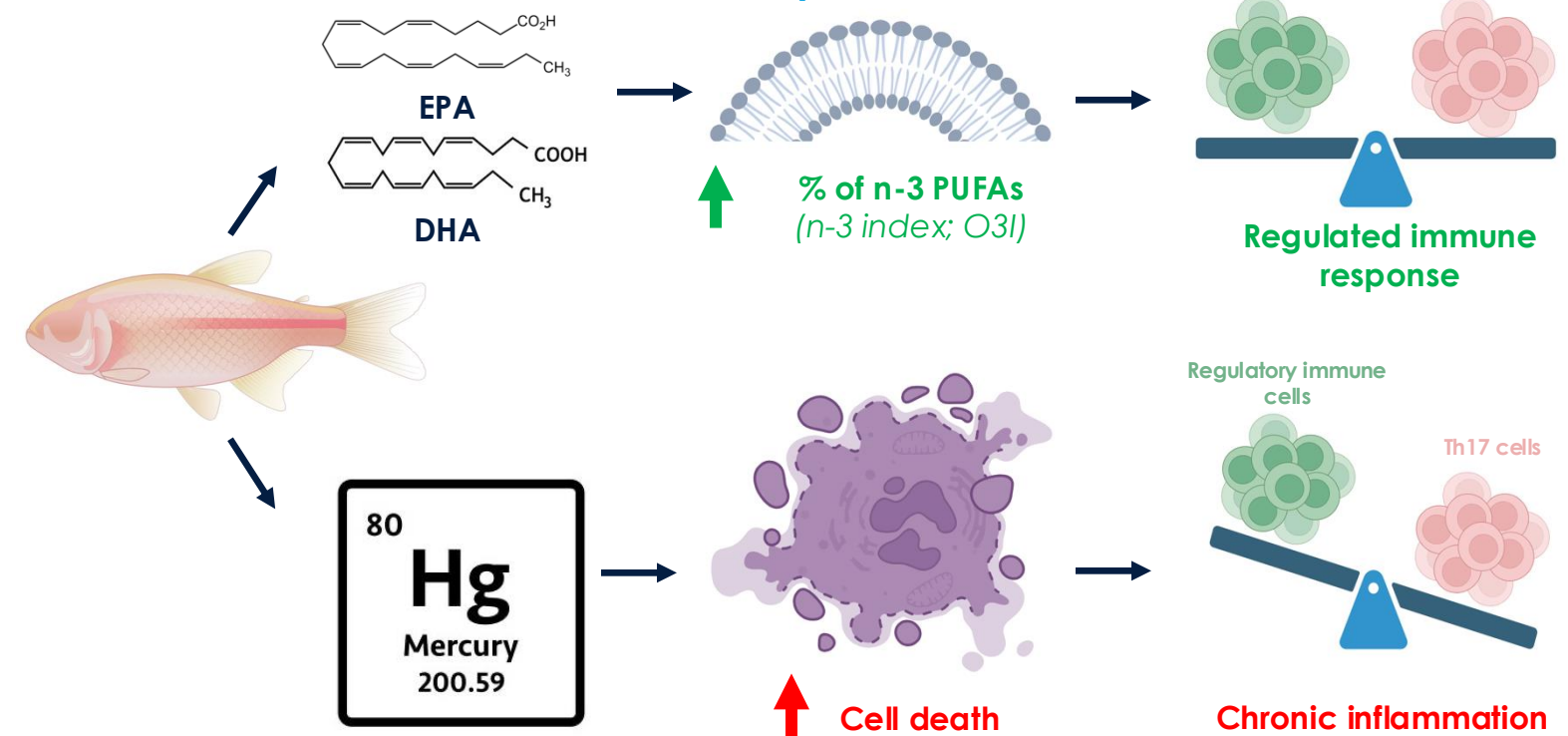
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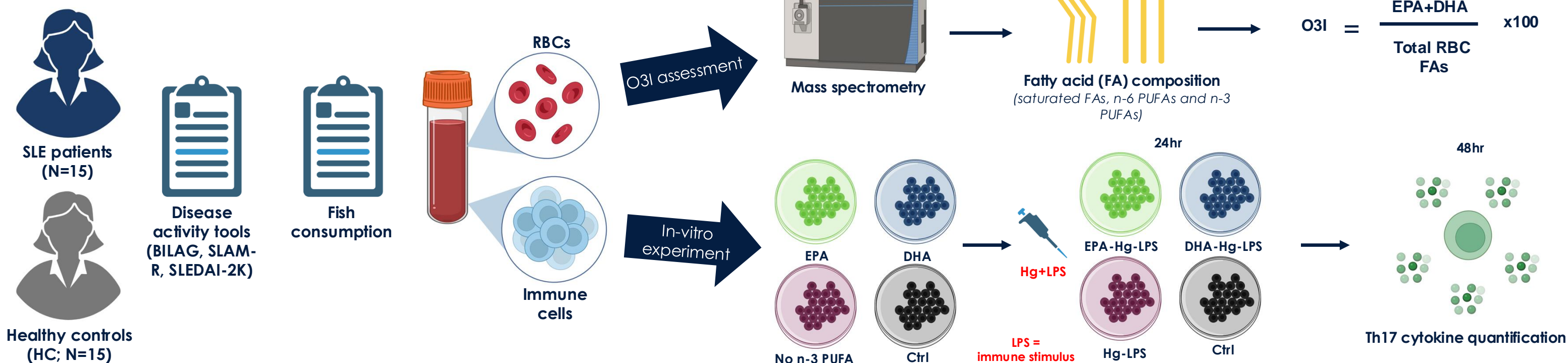
- SLE is a chronic autoimmune disorder where the immune system mistakenly attacks its own tissues¹
- Diet may influence SLE disease through its impact on cell death and immune function²
- Fish are a rich source of anti-inflammatory n-3 polyunsaturated fatty acids (PUFAs), eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), that positively regulate the immune system³
- Fish consumption is the primary exposure to mercury (Hg), an immuno-toxin⁴
- Hg exposure in healthy adults has been reported to increase Th17 cytokines, which have been implicated in autoimmune conditions, such as SLE^{5, 6}

No research has examined the effects of mercury and n-3 polyunsaturated fatty acids on Th17 cytokines in SLE pathology

Immune mechanisms from fish consumption^{7, 8}

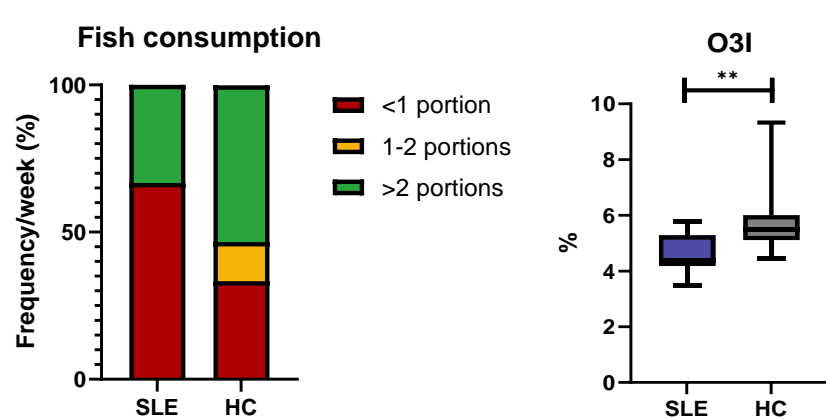


Methodology

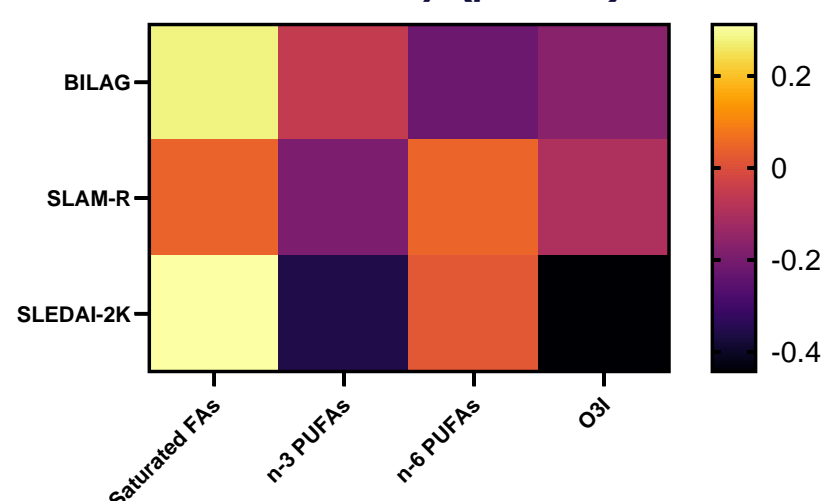


Results

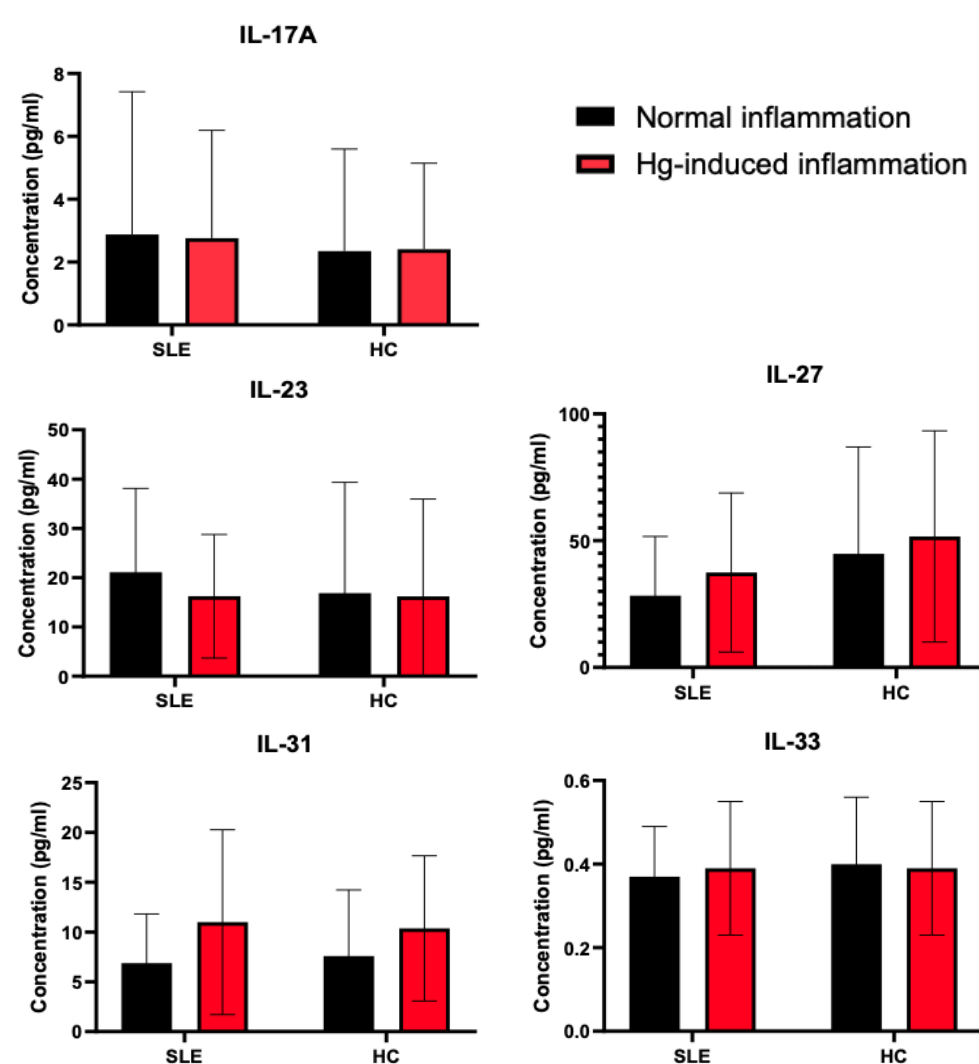
O3I was significantly lower in SLE patients compared to HC (**p<0.01)



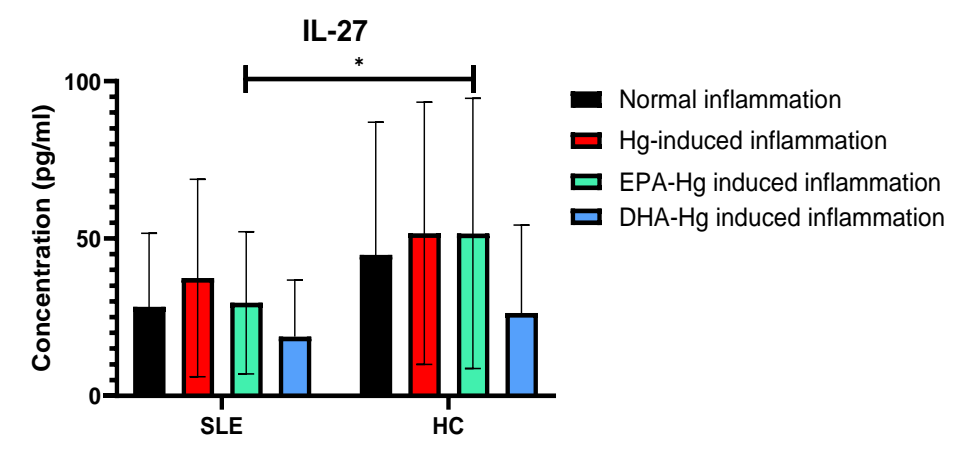
O3I was negatively correlated with disease activity (p>0.05)



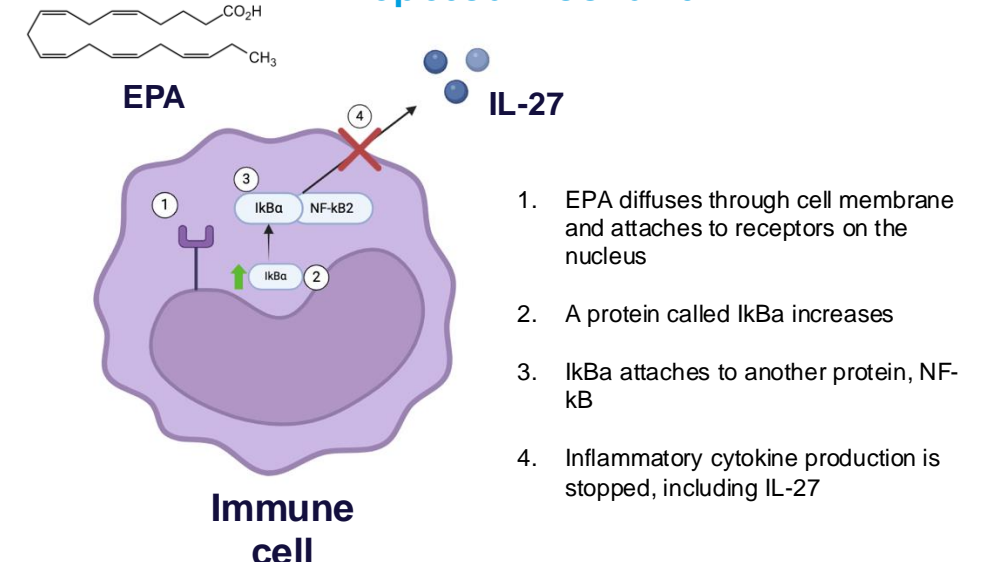
No significant differences in Hg-induced inflammation between SLE patients and HC (p>0.05)



EPA significantly protected against Hg-induced inflammation in SLE patients compared to HC (*p<0.05)



Proposed mechanism



Societal and scientific impact

- No apparent risk of mercury exposure on Th17 inflammation and highlights the safety of fish consumption for SLE patients
- Reductions in IL-27 support the protective role of n-3 PUFAs in autoimmune responses
- SLE patients might benefit from increasing their O3I through fish consumption and associations with disease activity should subsequently be re-examined

References

- Rees, F. et al. (2017), *Rheumatol*, 56, 1945–1961. 2) Islam, M.A. et al. (2020), *Front Immunol*, 2020, 11, 1477. 3) Calder, P.C. (2006), *AJCN*, 83, 1505S1519S. 4) G. Björkstrand, G. et al. (2020), *Clin Immunol*, 2020, 213, 108352. 5) Gardner, R.M. et al. (2010), *Toxicol Lett*, 198, 182–190. 6) Talaat, R.M. et al. (2015), *Cytokine*, 72, 146–153. 7) He, K. et al. (2009), *AJC*, 103, 1238–1243. 8) Pollard, K.M. et al. (2019), *BBA - Gen Subj*, 1863, 129299.