CYCLING, AIR POLLUTION AND HEALTH: Oxidative Stress as a Mediator of Systemic Inflammation

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OBJECTIVES
1. To determine the potential consequences of air pollution exposure in healthy cyclists.
2. To investigate the role of systemic inflammation and oxidative stress as a mediator of these effects.

HYPOTHESIS
Short-term cycling along a route with higher traffic-related air pollution will result in observed systemic inflammation and oxidative stress.

INTRODUCTION
- Cycling is promoted as a healthy transportation choice, but cycling on roads with automobiles, trucks, and buses may be exposed to elevated levels of air pollution.
- Traffic-related air pollution is linked to systemic inflammation, which can lead to cardiovascular morbidity and mortality.
- It has been hypothesized that this inflammation reflects oxidative stress, characterized by a predominance of reactive oxygen species (ROS).
- Few air pollution studies have examined inflammation and oxidative stress simultaneously, especially in healthy individuals.
- This information will help the development of healthy cycling infrastructure and will add to the health perspectives.

METHODS
Study design:
- 18 healthy cyclists (age 18-38) were randomized to cycling on two routes (separated by 14 days) with differing levels of traffic-related air pollution.
- Each ride was separated by at least 14 days.
- Systemic inflammatory (CRP, IL-6 and endothelial activation) and oxidative stress (8-OHdG) markers were measured before and after each ride.

RESULTS
- More ultrafine particle levels 50% higher on high vs low traffic route.
- Endothelial function decreased in cyclists riding along the high vs low traffic route.
- Small increases in IL-6 vs post exposure on the high traffic route.
- No change in CRP levels observed between routes.
- Elongation increases in CRP levels for high vs low traffic route.
- Little correlation was seen between CRP and markers of systemic inflammation.

CONCLUSIONS
These measures suggest that cycling in regions of relatively increased traffic density may have an acute additive effect on an endothelial dysfunction. Further, the role of stress is mediated through classical pathways of inflammation and oxidative stress remains unclear.

REFERENCES

ACKNOWLEDGMENTS
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Website: http://inflam.uhn.bruce.ca/publications