Reviewer’s report

Title: Chronic exposure to particulate matter in photocopier units causes oxidative stress and systemic inflammation among photocopier operators

Version: 1 Date: 21 March 2013

Reviewer: Stephan van Eeden

Reviewer’s report:

This paper Elango and co-workers addresses the problem of the local (lung) and systemic effects of exposure to photocopier associated air pollution. They measured spirometry and several circulating pro-inflammatory mediators in a cross sectional designed study. They showed that chronic exposure to photocopier dust does not alter spirometry parameters but it did elicit a systemic inflammatory response characterize by a pro-oxidative state, an increase in leukocyte associated pro-inflammatory mediators (LTB4, IL-8, ECP, ICAM-1) but no increase in CRP (the only acute phase protein they measured). They conclude that photocopier dust exposure elicit a systemic inflammatory response that could potentially impact downstream cardiovascular disease.

Major compulsory Comments:

1) The subject selection process is unclear from the MS. A) Were all 4 units they use similar (using similar instrumentation, work conditions/ventilation similar); B) how were the subjects that participate in the study selected, randomly? and were they evenly distributed between sites. Similar questions needs to be answered for the controls. This is not to be bias in subject selection

2) In these type of studies one need to know what was the outdoors air pollution measures just out- side the work places (to show a unique exposure environment). This need to be added to table one

3) When was the air quality tested in the units (end of a work day? and was it done similarly in all units)

4) What was the ultra fine component measurements (this is the most dangerous part of printer and photocopier toner and also the component with the strongest association with systemic inflammation and cardiovascular disease)

5) The current smoking status is most likely the more important parameter to report (will influence the measurements more that pack years). How many were current/active smokers

6) Statistic used is very basic, why not model and correct for confounders such as cigarette smoke, age sex etc. Corrections for multiple comparisons need to be done

7) The discussion section is to long and speculative, focus on the positive and important negative findings

Minor essential Comments:
1) Authors need to mention all the underlying diseases subjects were screened for that could potentially impact results (specifically inflammatory conditions).

2) All the inflammatory markers can be collated in a table.

3) Figure 1, 4 and 5-9 are redundant. This data can be either in a table or report in the text.

4) Unclear why “mixed cells” are reported. Why not report Eosinophil, monocyte and basophil counts individually. The band cell counts is a good marker of systemic inflammation (shown in cigarette smoking and air pollution), and may significantly strengthen the paper if that was elevated in the exposed group.

5) CRP can be variable, what about measuring fibrinogen, good acute phase protein that relate to cardiovascular disease.

**Level of interest:** An article of importance in its field

**Quality of written English:** Acceptable

**Statistical review:** Yes, and I have assessed the statistics in my report.

**Declaration of competing interests:**

No financial or any other interest