

# Study: 'E-waste pollution' a threat to human health

30 May 2011

In addition to its damaging effect on the environment and its illegal smuggling into developing countries, researchers have now linked e-waste to adverse effects on human health, such as inflammation and oxidative stress - precursors to cardiovascular disease, DNA damage and possibly cancer.

In a study published today, Tuesday 31 May, in IOP Publishing's journal *Environmental Research Letters*, researchers took air samples from one of the largest e-waste dismantling areas in China and examined their effects on human lung epithelial cells.

E-waste, or electronic waste, describes end-of-life electrical goods such as computers, televisions, printers, and mobile phones. Each year between 20? million tons of e-waste is generated worldwide, 100,000 tons of which is exported from UK shores, according to a recent BBC Panorama programme. A large proportion of worldwide e-waste is exported to China.

Due to the crude recycling process, many pollutants, such as persistent organic pollutants and heavy metals, are released from e-waste, which can easily accumulate in the human body through the inhalation of contaminated air.

After exposing the cultured lung cells to the organic-soluble and water-soluble constituents of the samples, the researchers tested for the level of Interleukin-8 (IL-8), a key mediator of inflammatory response, and Reactive Oxygen Species (ROS), chemically reactive molecules that can cause extensive damage in excess.

The samples were also tested for the expression of the p53 gene - a tumour suppressor gene that produces a protein to help counteract cell damage. If there is evidence of this gene being expressed it can be seen as a marker that cell damage is taking place.

The results showed that the samples of pollutants caused significant increases in both IL-8 and ROS levels - indicators of an inflammatory response and oxidative [stress](#) respectively. Significant increases were also observed in the levels of the p53 protein with the risk of organic-soluble pollutants being much higher than water-soluble pollutants.

Co-author of the study Dr Fangxing Yang, of Zhejiang University, said, "Both inflammatory response and oxidative stress may lead to DNA damage, which could induce oncogenesis, or even cancer. Of course, inflammatory response and oxidative stress are also associated with other diseases, such as cardiovascular diseases."

In this study, the researchers took samples of the air from Taizhou of Zhejiang province - a dismantling complex that involves more than 60,000 people and dismantles more than two million tons of e-waste to recycle metals each year.

To obtain the samples, the researchers used two sampling sites that were located downwind of a dismantling industrial park in Taizhou, set up by the local government in 2004.

It is well known that [inflammatory response](#) and oxidative stress can lead to DNA damage and therefore activate the p53 gene to counteract this damage. The study did not find any significant correlation between IL-8 and ROS and p53 expression; however the researchers suggest that this may be due to the various other endpoints, not examined in this study, which can damage DNA.

A further study will attempt to characterise the components present in the polluted air and identify the key contributors to these adverse effects.

Dr Yang continued, "From these results it is clear that the 'open' dismantlement of [e-waste](#) must be forbidden with more primitive techniques improved. As the results show potential [adverse effects](#) on

[human health](#), workers at these sites must also be given proper protection.

"Furthermore, one must consider the initial manufacturing process of electrical goods and look to utilise more environmentally and human friendly materials in their production."

**More information:** "Comparisons of IL-8, ROS and p53 responses in human lung epithelial cells exposed to two extracts of PM2.5 collected from an e-waste recycling area, China" *Environ. Res. Lett* **6**, 024013. [iopscience.iop.org/1748-9326/6/2/024013](https://iopscience.iop.org/1748-9326/6/2/024013)

Provided by Institute of Physics

APA citation: Study: 'E-waste pollution' a threat to human health (2011, May 30) retrieved 9 September 2020 from <https://phys.org/news/2011-05-e-waste-pollution-threat-human-health.html>

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