Brief Review on Health Effects of Laser Printer Emissions Measured as Particles

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Acknowledgement

This report was prepared by Dr Roger Drew of Toxikos Pty. Ltd. to assess the health hazards related to the emissions from laser printers in the workplace.

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The report was reviewed by Safe Work Australia’s Nanotechnology Work Health and Safety Advisory Group, the Nanotechnology Work Health and Safety Measurement Reference Group and other stakeholders. Comments were received from Professor Lidia Morawska (Queensland University of Technology), Dr Ian Gardner (Department of Defence), Halil Ahmet (WorkSafe Victoria), and Peter McGarry (Workplace Health and Safety Queensland).

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Toxikos Pty. Ltd. is a consulting company formed on December 1st 2000 to provide clients with independent excellence in toxicology and health based risk assessment. Its charter is to assist industry and government make science based decisions regarding potential effects and management of environmental and occupational chemicals. For over twelve years, prior to and since the establishment of Toxikos, staff have provided toxicology and health risk assessment advice to clients in a wide range of industries and government in Australia, New Zealand and South Africa.

About the author: Dr Roger Drew is Principal consultant of Toxikos Pty. Ltd. He has primary degrees in biochemistry and pharmacology and postgraduate degrees in toxicology. Postdoctoral training was undertaken at the National Institutes of Health, National Cancer Institute in the USA and he spent twelve years teaching medical students and conducting toxicological research at Flinders University of South Australia. He was corporate Toxicologist to ICI/Orica Pty Ltd for ten years before creating Toxikos Pty Ltd. Dr Drew is the only consultant toxicologist in Australia certified by the American Board of Toxicology.

Dr Drew has been a toxicology consultant to Australian Federal and State Authorities; a member of several standing expert committees of the National Health & Medical Research Council of Australia and the National Occupational Health and Safety Commission of Australia. He has been a member of many expert task groups of the World Health Organization for the International Programme on Chemical Safety.

Dr Drew was Adjunct Professor of Biochemical Toxicology at RMIT University and teaches various aspects of toxicology and risk assessment to undergraduate and postgraduate students at RMIT, Monash and Melbourne Universities. He is a member of several professional toxicology societies and is a recognised national and international expert in toxicology and risk assessment. He is currently on the editorial board of the international scientific journal “Regulatory Toxicology and Pharmacology” and Adjunct Associate Professor in the Department of Epidemiology and Preventive Medicine, Monash University
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Executive Summary

A research report by Queensland University of Technology and Workplace Health and Safety Queensland, commissioned by Safe Work Australia, details the level of workplace exposure to laser printer emissions and provides guidance on control measures to reduce exposure levels (Morawska et al., 2011). Safe Work Australia commissioned Toxikos Pty. Ltd. to undertake a brief review of the findings of this, and other research, to evaluate potential adverse health effects associated with exposure to laser printer emissions measured as particles and consider the results in relation to health risk in the workplace.

For this brief review no epidemiology studies directly associating laser printer emissions with adverse health outcomes were located.

The components of laser printer emissions are different to toner particles and the particulates of urban ambient air pollution. Recent research indicates emissions from laser printers are primarily aerosol condensates of volatile organic compounds (VOCs) or semi-volatile organic compounds (SVOCs). If the emissions of laser printers are primarily VOC, or SVOC, it would be logical to expect possible health effects to be more related to the chemical nature of the aerosol rather than the physical character of the ‘particulate’ since such emissions are unlikely to be or remain as ‘particulates’ after they come into contact with respiratory tissue.

Nevertheless assessment of risk from laser printer emissions has been conducted assuming the emissions could be either particulates or aerosols of VOC/SVOCs. A comparison of the maximum 8 hour TWA levels of exposure to laser printer emissions, as reported Morawska et al. (2011) approximately 1 metre from the printer, has been made with:

- WHO Air Quality Guidelines,
- Australian National Exposure Standards,
- the German Federal Environment Agency Indoor Guidelines, and
- toxicological thresholds of concern for concentrations of chemicals in air.

These screening risk assessments are necessarily conservative (i.e. biased towards safety) in order to take account of uncertainties in exposure, the nature of the emissions, and the identity/health effects of all laser printer emission components. Each of the assessments indicates the risk of direct toxicity and health effects from exposure to laser printer emissions is negligible; however, people responsive to unusual or unexpected odour may detect/react to the presence of emissions. This is consistent with the limited health information for emissions from office equipment in general.
1. Introduction

Safe Work Australia commissioned Queensland University of Technology and Workplace Health and Safety Queensland to undertake research on laser printer emissions in workplace environments. The research report describes the level of workplace exposure to laser printer emissions measured as particles and recommends guidance on control measures to reduce exposure levels (Morawska et al., 2011). An examination of the health hazards involved with exposure to laser printer emissions was not undertaken. Thus, Safe Work Australia commissioned Toxikos Pty Ltd to carry out a brief review of the findings of Morawska et al. (2011) and other research to evaluate potential adverse health effects associated with exposure to laser printer emissions and consider the results of Morawska et al. (2011) in relation to risks to health in the workplace.

Safe Work Australia provided Toxikos with a number of relevant references and this was supplemented by a literature search undertaken by Toxikos using the search words “printer emissions” or “printer particulates” coupled with “health effects” or variations of the word “toxicity”.

2. Background and Concerns

Airborne particles within an office environment are a combination of particles generated from various sources, included are laser printers which emit paper fibres, organic vapours and inorganic gases (He et al., 2007). Indoor air also contains particles generated outside, such as those from vehicle emissions, which have infiltrated into the building; in fact, Morawska et al. (2011) reports the 8 hour time-weighted average (TWA) printer particle exposures are generally below the local background levels (i.e. without printer contribution) for each office area for the equivalent period. This indicates the majority of the particle exposure experienced by office workers over the course of a working day came from sources other than printers.

Various studies have shown the particle diameter associated with emissions during operation of laser printers is predominantly within the ultrafine size range of less than 100 nm (< 0.1 µm) with an average size of around 30 – 50 nm (Namiki et al., 2006; He et al., 2007; Wensing, 2008; Morawska et al., 2009).

Particle emission levels are however printer specific and affected by printing conditions including the number of pages printed, cartridge age and toner coverage. It should also be appreciated that printer design is continuously evolving and consequently characterisation of emissions from older printers may not reflect those from newer machines.

Concern regarding potential health effects from exposure to particles in laser printer emissions stems from extrapolation of associations of ‘sick building syndrome’ (SBS)1 with poor indoor air quality, human health effects associated with particulate ambient air pollution and anxiety regarding uncertainty of effects after inhalation exposure to engineered nanoparticles. For the latter, toxicological experiments show they have potential to cause adverse health effects such as pulmonary inflammation and fibrosis but compelling evidence for effects in humans is lacking at this time (SWA, 2009). Possible reasons why health effects haven’t been observed in humans include the engineered nanoparticles or their agglomerates in workplace exposures lack toxicological potency, exposures are negligible or very low, there is little widespread use, appropriate studies haven’t been conducted, and some of the potential effects from high exposure may have long development latencies. A number of investigations have identified associations between SBS symptoms and the use or presence of carbonless paper (Skov, 1989; Stenberg, 1994; Fisk, 1993), video display

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1 Symptoms of SBS include irritation of eyes and respiratory tract, itchy skin, coughing, shortness of breath, headache, fatigue and/or malaise.
units (Skov, 1989; Stenberg, 1994), photocopiers (Skov, 1989; Stenberg, 1994; Fisk, 1993),
carpets (Fisk, 1993) and psychosocial/personal factors (Skov, 1989; Stenberg, 1994)
including allergies. While these studies have shown associations of SBS with the use of
office equipment, they fall short of indicating a causal relationship.

3. Emission Levels of Particles from Laser Printers

The research by Morawska et al. (2011) reported that laser printers which emit nanoparticles
are common within office workplaces, with 45 (42%) of the available printers initially
surveyed being classified as low to high emitters, and 62 (58%) as non-emitters. Of the 25
printers subjected to continuous particle measurement at one metre from the printer, 18
recorded a statistically significant increase in particle number concentration associated with
printing. In addition, four of five printers subjected to continuous particle measurement at two
metres from the printer also recorded a statistically significant increase in particle number
concentration associated with printing. Therefore these printers increased exposure of office
workers to particles above the local background particle exposure at both one and two
metres respectively.

Peak particle exposure was recorded one metre from printers during printing events at
greater than five times that of the eight-hour time weighted average (TWA) local office
background particle exposure for 11 printers, at four times for one printer, three times for two
printers, and between one and two times for eight other printers. The peak particle exposure
measurements ranged from $3.3 \times 10^3$ particles cm$^{-3}$ to $9.9 \times 10^4$ particles cm$^{-3}$ (this is the
particle saturation value of the condensation particle counter, and therefore particle
exposure was greater than this value).

However, average nanoparticle exposure in an eight hour time weighted average (TWA) is
predominantly from sources other than printers; the average printer particle emissions
ranged from $4.3 \times 10^1$ particles cm$^{-3}$ to $4.0 \times 10^3$ particles cm$^{-3}$.

Wensing et al. (2008) noted rapid decay in particle concentration in an emission test
chamber after a print test which prevented determination of aerosol composition or particle
size changes due to agglomeration.

4. What Are The Emitted Particles?

The nature of the particulates is important in determining or anticipating possible health
effects. The older literature suggests that the major particulate candidates are toner powder or
condensates of VOC/SVOC.

4.1 Toner Powder?

Based on their size being less than 0.1 µm, particles in laser printer emissions are unlikely to
be toner dust, which is comprised of much bigger particles, around 10 µm. Indeed Smola et al.
(2002) found toner dust was not emitted in measurable amounts. Wensing et al. (2008) also
doubts the ultrafine particulates are primarily toner and Ewers and Nowark (2006) argue toner
powder cannot be emitted from laser printers during operation.

While conducting this review it was noted that when justifying their experiments or speculating
about the potential health effects some authors assume toner powder may be in printer
emissions (e.g. Bai et al., 2010; Tang et al., 2010; Theegarten et al., 2010). In none of these
instances has it been demonstrated by the authors that toner dust is in the printer emissions.
Information on health hazards associated with toner particles is in Appendix B.
4.2 Volatile substances?

Wensing et al. (2006, 2008) undertook preliminary identification of particles emitted from chamber experiments using printers modified to operate without paper, they identified high boiling point siloxanes and other SVOCs derived from chemicals associated with thermoprotection of plastics. The authors also considered super-saturated water vapour released from the paper near the fuser unit would be able to form a condensate. In addition to the common VOCs (styrene, xylene, ethylbenzene, toluene), Barrero-Moreno et al. (2008) found 2, 3 dimethyl-2, 3-diisobutyl succinonitrile, a SVOC derived from thermal decomposition of a toner component, in laser printer emissions.

Destaillets et al. (2008) reviewed the literature for emissions from office equipment and describes a range of VOC/SVOC species in emissions from laser printers. Others have also indicated VOCs are the primary components of printer emissions (e.g. Ewers and Nowark, 2006).

Morawska et al. (2009) extended the above observations to show for the first time that the particles are volatile and are of secondary nature, being formed in the air from VOCs originating from both the paper and hot toner. In these experiments the authors reported there was no evidence of a non-volatile residue suggestive of seeded nucleation; i.e. there was no ‘solid’ core to the particulates. It was also shown that although ozone formation by printers may be low, ozone did play a role in secondary particle formation. The paper postulates that particles may be formed during laser printing via two processes:

1. Homogeneous nucleation of SVOC species to form particles. The VOCs identified that may act as nucleating species included xylenes and styrene, ethylbenzene, pentadecane, hexadecane, heptadecane and dimethyl phthalate, and

2. Secondary particle formation through a reaction between VOC species and ozone to produce a further SVOC species.

4.3 Conclusion

To date the majority of studies investigating particulate emissions from laser printers have focussed primarily on size characterisation. The evidence evaluated for this brief review indicates the majority of particulates emitted from laser printers are likely to be aerosols of VOCs and SVOCs. It is noted however that for the material gathered there is only a single study that has systematically, and adequately investigated this aspect. Greater confidence in the VOC/SVOC nature of laser printer emissions will be gained when independent laboratories substantiate and extend the findings of Morawska et al. (2009).

If the laser printer emissions are primarily VOC, or SVOC aerosols without a solid core as suggested by the work of Morawska et al. (2009) then they are expected to interact with membranes differently than particulate matter in urban air. For example macrophages will not be able to phagocytise these aerosols. The water and lipid solubility of the VOC and SVOC aerosols, aided by phospholipid pulmonary surfactants, will allow them to dissolve into mucus and cell membranes in much the same way as vapours and gases of similar physicochemical properties. Although the VOC and SVOC aerosols are measured as ‘particulates’ they will not remain physically as particulates once in contact with respiratory tissues. Thus possible health effects are expected to be more related to the intrinsic toxicity (i.e. as determined by the chemistry) of the VOC/SVOC rather than a physical characteristic of a ‘particulate’. Nevertheless because confirmation of the work of Morawska et al. (2009) is needed before a definitive conclusion can be pronounced regarding the non-physical particulate nature of laser printer emissions, in assessing the health risks in Section 6 it has
been assumed they may either behave as vapours/gases or as solid particulates such as found in polluted urban air.

It is reasonably clear the particulates in laser printer emissions are not unchanged toner powders. While toxicological information (Appendix B) for toner powder is relevant for workplace exposure to dusts in manufacturing, cartridge assembly and recycling, cartridge handling or laser printer maintenance, they are not relevant for judging health risks from laser printer emissions.

5. Potential Health Hazards Associated With Printer Emissions

Ewers and Nowark (2006) have reviewed the literature on health effects associated with printer emissions but almost all the information in the review relates to toners. Some of the individual case studies cited by the authors may however include ‘emissions’ other than toner. They concluded “So far, there have been no scientifically established indications that the operation of modern laser printers and copiers in offices and households leads to an increased health-relevant exposure caused by toners and VOC. Human biomonitoring examinations did not indicate an increased internal exposure to harmful substances for persons who work intensively with laser printers and copiers.” Since that review there have been a few additional human investigations and toxicological studies of printer emissions.

5.1 Human studies

Jaakkola et al. (2007a) conducted a cross-sectional study of 1016 adults in Finland, including 346 office workers, in relation to symptoms of sick building syndrome and exposure to fumes from printer and copier emissions (FPP), paper dust and carbonless copy paper (CCP). Both symptoms and exposures were estimated by self-administered questionnaire, the details of which are not provided. The authors report all three exposures were related to a significantly increased risk of general symptoms (headache and fatigue). Exposure to paper dust and to FPP was associated with upper respiratory and skin symptoms, breathlessness, tonsillitis and middle ear infections. It is noted the study was a cross section design and it appears the effects and exposure were requested for any time in the previous 12 months prior to the questionnaire, how the effects were linked to exposure is not explained but it found that women were more likely to be ‘exposed’. There is no information on how exposures were estimated, no statistical analysis, and there were markedly disparate numbers in the ‘exposed’ vs ‘non-exposed’ groups.

In a case–control study from a subset of same population, Jaakkola et al. (2007b) reported an association between adult onset asthma and paper dust and carbonless copy paper, but not with fumes from copiers and printers. Although the asthma patients were newly diagnosed, it is unclear whether the study is concerned with the induction of asthma or exacerbation of symptoms in those with existing asthma.

Theegarten et al. (2010) published a paper on a case report for a patient in Essen, Germany who had a laser printer on her work desk for three years, and had a three month history suffering from abdominal pain, weight loss and diarrhoea. The initial interpretation of colon biopsies were negative, endometriosis was suspected, which lead to laparoscopy and the discovery of black spots in the peritoneum. Light, electron microscopy and energy dispersive X-ray analysis showed these spots were submesothelial carbon aggregates consisting of nanoparticles (31 – 67 nm) around which was foreign body inflammation and macrophages with nanoparticles inside. The authors noted these nanoparticles were the same size as reported to be emitted from printers (Wensing et al., 2008) and they referred to occupational

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2 The subset was only those with no previously diagnosed asthma or long-term use of asthma medications and included 316 of 932 controls who were office workers.
literature indicating handling toners was associated with respiratory health effects, thoracic radiological abnormalities and in some cases granulomatous pneumonitis. Because the patient had a laser printer on her work desk for three years the authors assumed she had been exposed to toner dust. They concluded “We have shown that workers with toner dust exposure from laser printers can develop submesothelial deposition of CNP in the peritoneum”. This is an invalid conclusion; the black spots in the peritoneum of this patient were only shown to be carbon particles, they were not demonstrated to be toner dust. The patient would have been exposed to a range of particulates, not the least of which would be ambient particulate pollution. It is not mentioned whether the person was a past or current smoker. Re-examination of the colon biopsies revealed histological lesions consistent with Crohn’s disease which explained the patient’s health status. In this patient the presence of black spots (carbon nanoparticles) in the peritoneal biopsies was without health effects. The source of the black spots is unknown.

The titles of some papers (e.g. Adetunji et al., 2009) suggest health investigations from exposure to printer emissions have been undertaken but in fact the studies measure the amount and size distribution of particles and only speculate on health effects.

5.2 Toxicological investigations

A pilot chamber study on the genotoxic effects of printer emissions was undertaken by Tang et al. (2010) by directing emissions onto a cell culture. Five printers were evaluated. During printing operations four increased TVOC concentration in the chamber and three of these also had large increases in particulate (10 – 1000nm) emissions. While cell viability was unchanged, one hour exposure to emissions from two of the latter printers caused a significant increase in micronuclei formation in the exposed A549 cells, i.e. indicating the emissions were genotoxic. The printer with the largest TVOC emissions (but with no increase in particulates) did not increase micronuclei and the printer with the largest particulate emission (significantly higher than the other printers) also did not increase micronuclei. Thus there appears to be no simple correlation between emission composition as determined in the study and genotoxicity in the cells.

The authors did not identify the emission components associated with the genotoxicity, and suggested the TVOC increases were unlikely to account for the genotoxicity observed. They discuss other data from their laboratory indicating some but not all toners can induce micronuclei in A549 cells \(^3\), thereby implying the toner powder may be responsible.

From a human health risk aspect this experiment is difficult to interpret. The experimental setup is novel, and extrapolating results from \textit{in vitro} exposure of cancer cells to printer emissions that have been directed close to the cell culture surface is highly uncertain. The chamber containing the printers was small and air flow through it was adjusted to meet the EU standard of one exchange per hour. Thus, the volume dilution of the emissions was likely to have been very small relative to most office conditions. No ‘dose response’ exposures were undertaken and we note a concentration of formaldehyde (positive control) of 100 mg/m\(^3\) (1,000x greater than the WHO indoor air guideline) produced only slightly more micronuclei, but did compromise cell viability. Furthermore, many compounds that are positive in \textit{in vitro} micronuclei assays are negative in \textit{in vivo} assays.

\(^3\) The A549 cell line was established in 1972 from an excised adenocarcinoma that originated from alveolar basal epithelial cells.
Konga et al. (2009) used an asthma mouse model to expose mice to printer emissions, cigarette smoke and both simultaneously. The exposures used in this study were high. At the end of the exposures, lung tissues were collected and homogenized for analyses of a range of biochemical parameters. Tobacco smoke significantly affected many of the parameters measured but printer emissions had very limited effects. The combined exposure of printer emissions and tobacco smoke had greater effects than tobacco smoke on its own and the authors considered there were synergistic effects. Apart from showing that concomitant high exposures of both types of particles have greater effects than either alone the relevance of this study for human health is unclear. The exposures were uncharacterised, the animal model unusual and there are reporting deficiencies in the paper.

5.3 Summary and conclusion

No epidemiology studies directly associating laser printer emissions with adverse health outcomes were located. The concern regarding possible health effects is by association with health effects caused by ambient air pollution, indoor air quality (sick building syndrome, SBS), or concerns with inhaling nano-particulates per se. The literature indicates associations between health effects and emissions from various office based products and equipment including paper products, photocopiers, carpets, air conditioning and psychosocial factors. However cause and effect relationships with laser printers have not been established. A variety of methodological and reporting issues make the few toxicological studies concerning laser printer emissions difficult to interpret with respect to human health impacts.

6. Health Risks

6.1 Base assumptions

This report has concluded the emissions from laser printers are likely to be almost entirely VOC or SVOC aerosols, however since the depth of information is limited there is uncertainty regarding the absoluteness of this conclusion (Section 4). Consequently when considering the health impacts of laser printer emissions it is appropriate to be cautious regarding the constituents and particulate nature of the emissions. To accommodate this uncertainty, deliberations on health risks from exposure to laser printer emissions in this Section are considered as if they were either ‘solid’ particulates, or as aerosols of VOC/SVOCs.

The distinction between the two options for the nature of the printer emissions is pivotal for understanding potential health impacts. If laser printer emissions are primarily composed of VOC or SVOC aerosol particles as shown by Morawska et al. (2009) they are markedly different from the combustion carbon based particulates which have various metals associated with them that dominate urban air, and will behave quite differently in the milieu.

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4 The asthma mouse model was created by injecting BABL/C mice intraperitoneally on days 0, 7, 14, and 21 with 20 µg ovalbumin emulsified in 2.0 mg aluminium hydroxide adjuvant. The mice were exposed to an aerosolized 5% ovalbumin solution for 20 minutes per day after the second and last sensitization.

5 An inkjet printer was used for the study. The printer was placed in the chamber of volume 7200 cm³ and the mice were exposed to printer emissions 1 hour per day for 4 weeks. The mice were placed to the back side of the printer in the chamber because the printer emissions were found to be highest at the back when compared to the front side of the printer. The chamber was provided with holes for air exchange. Mice were exposed to tobacco smoke from cigarettes for 4 weeks, 1 hour per day. Tobacco smoke exposures were performed in the chamber at the same volume but how the smoke was generated is not provided. Simultaneous exposure was for 4 weeks, 1 hour per day.

6 Reactive oxygen species, lipid peroxidation levels, ATPase, DNA damage [8-hydroxy-2-deoxyguanosine], and mitochondrial function [enzyme activity, membrane potential, membrane fluidity protein expression].
of the lung than will particulate matter (PM) from ambient air pollution. It is also noted the UK Department of Health considered the findings on health effects of low particle concentrations from population-scale epidemiological studies of the outdoor ambient aerosol (i.e. urban particulate pollution) cannot be used to predict the effects of indoor concentrations of particles on health, nor be used as a basis for suggesting indoor guidelines. This conclusion was primarily reached because indoor aerosols differ significantly in source, chemical composition and size distribution from the outdoor aerosol (COMEAP, 2004).

It is emphasised it is unlikely, although not certain, that laser printer emissions are equivalent to ambient PM with respect to health effects. The ‘particulate’ analysis for laser emissions herein is provided for precautionary information.

As with risk assessments for chemicals, characterisation of potential health risks associated with emissions from laser printers is reliant upon exposure quantitation. Often this cannot be done accurately and consequently conservative (i.e. over predictive) exposure assumptions are often applied. Risk characterisation in screening, or Tier 1, risk assessments is undertaken by comparing exposure estimates with guideline criteria that are health protective. Such criteria are invariably linked with an assumed exposure time, e.g. 8 hours for occupational guidelines or 24 hours for public health guidelines.

For laser printers there is little information available to facilitate sensible time averaged quantitative predictions of personal exposure to emissions. Although there are a number of experimental studies (e.g. Lee et al. 2001, He et al. 2007, Morawska et al. 2009) that have measured particle emissions from laser printers they do not facilitate the required exposure estimations for risk assessment since the data is very dependent upon the experimental conditions of the studies. These investigations were undertaken to characterise the nature of laser printer emissions rather than characterise exposure. During the working day printer emission exposure is dependent upon such factors as closeness to the printer, room ventilation, printer model, printer age, cartridge model and age, toner coverage on the paper, type of paper, and the number of print runs during the day (He et al., 2007).

To some extent the above variables have been inherently addressed by the investigation of Morawska et al. (2011) who measured printer related air concentrations of particulates in 19 office environments in Australia. The measurements were done at the average breathing zone height of a seated office worker and at distance of 1m from the laser printer. This was the typical minimum distance between an occupant of a computer workstation and a desk-located laser printer. Morawska et al. (2011) consider the measurements were obtained for the potential worst case exposure scenario for an office worker. Furthermore Morawska et al. (2011) undertook time series measurements throughout the working day and expressed the printer particle exposures as 8 hour time weighted averages (TWAs). This facilitates risk characterisation with criteria that have averaging times associated with them.

In summary, Morawska et al. (2011) calculated 8 hr TWA printer particle exposures for 19 office environments at breathing zone height 1 m from the operating printer. Particle concentrations ranged from $4.3 \times 10^1$ particles cm$^{-3}$ to $4.0 \times 10^3$ particles cm$^{-3}$. This report uses the highest concentration reported by Morawska et al. (2011) as a reasonable worst case exposure. This data is for a high emitting printer that was frequently used during the day in an office environment in which background exposure was low.
6.2 Considering emissions as ‘particulates’

6.2.1 Particulate risk calculations

As part of the NANOSAFE-2 European Union Framework Programme 6 (FP6) project Hänninen et al. (2010) estimated the occupational and consumer risk for ultrafine particles emitted by laser printers. No toxicological or epidemiological data for particles emitted by laser printers were used in the risk estimations. Rather it was assumed the ultrafine particulates from laser printers were the same form as particulates in polluted ambient air (i.e. PM$_{10}$) and would have the same health effects.

The basic approach was to transform the concentration–effect relationships from human ambient particulate matter (PM$_{10}$) epidemiology, or observations from toxicological in vitro and in vivo animal studies, into comparable human dose-responses. From the epidemiological data on health effects of air pollution the end point used for calculating risk was mortality observed in the general population. It was assumed this was caused by retained cumulative alveolar lung deposition of the non-soluble fraction of ambient particles. The daily uptake of laser printer particles was estimated based on particle size distributions and lung deposition modelling using three dose metrics; mass, particle number and surface area. The predicted risks from exposure to emission particles from laser printers were 4–13 (based on particle mass), or 12–34 (based on particle number), deaths per million persons exposed. The authors point out these calculated risks are substantially lower than risks due to ambient particles but still higher than the normally accepted risk level of 1 in a million.

From an unpublished evaluation of toxicological data, Hänninen et al. (2010) reported that studies on ambient particles revealed consistent values for the lowest observed effect levels (LOELs). After converting this data into equivalent daily uptakes using allometric scaling, the assessment indicated no significant health risks due to printer particles.

There are many areas of uncertainty in the calculated risk estimates, arguably the most important is the absence of consideration of the different compositional characteristics of urban air pollution particulates compared with laser printer emissions; it was assumed printers were expected to emit mainly black carbon particles however from Section 4.3 this is likely an incorrect assumption.

It is the opinion of this report that laser printer emission risks calculated by Hänninen et al. (2010) should not be relied upon because:

- the underpinning assumption that particles in laser printers are the same as urban ambient PM is likely not correct,
- key to the risk calculations is the distillation of urban particulate dose response for lethality which has not been articulated by Hänninen et al. (2010),
- the mechanics and math of the processes used to generate the risk estimates are not provided, and

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7 The toxicological assessment is confusing. The methods section of Hänninen et al. (2010) paper indicates in vitro and in vivo studies on nanoparticle toxicity were reviewed whereas the abstract indicates ambient particles. The allometric scaling model developed by the authors for transforming the observed effect levels from in vivo and in vitro studies into human alveolar doses is described as being based on relative metabolic rates but the details are not provided. The toxicological assessments are not provided; the detailed review is cited as unpublished. It is however stated the most sensitive combination of toxicological effect were adopted for the risk assessment; this was intracellular calcium balance. There are also many unsupported/unexplained assumptions regarding exposure (e.g. floor area per printer, volume of room, air exchange, printing time, time activity of exposed persons, emission rates, intakes and alveolar disposition). While an overview of the methodological approach is provided in a figure, the mechanics of the process(es)/parameters integration are not provided.
an examination of the uncertainty in the range of exposure assumptions in this publication has not been taken into consideration by the authors.

6.2.2 Comparison with WHO air guidelines

WHO (2008) notes the major components of ambient PM are sulphate, nitrates, ammonia, sodium chloride, carbon, mineral dust and water; it consists of a complex mixture of solid and liquid particles of organic and inorganic substances suspended in the air. Notwithstanding there is a significant difference between the nature and size\(^8\) of particulate emissions from laser printers and ambient PM, an appreciation of the likely importance of laser printer derived indoor particulates for health, relative to ambient air particulates, can be gained by comparing the air concentrations of the former with the WHO Air Quality Guideline for particulate matter in ambient air, PM\(_{2.5}\); this is 25 µg/m\(^3\) (24 hour mean).

The maximum 8 hr TWA concentration of particulates 1 metre away from a printer measured by Morawska et al. (2011, Table 3) was 4,000 particles/m\(^3\). Assuming these particles are spherical with average size of 40nm and have a density\(^9\) similar to that of PM\(_{2.5}\) (~2g/cm\(^3\)), this particle number concentration translates into a mass concentration of 0.27 µg/m\(^3\) (see Appendix A for conversion of particle number concentration to particle mass concentration). This is approximately 100 times less than the WHO PM\(_{2.5}\) air guideline. After taking into account the different averaging times (8 hr vs 24 hr) the difference becomes approximately 300x.

Eroding the 300 fold exposure margin is the recognition:
- that the health effects of ambient PM may be more associated with the ultrafine fraction rather than the fine fraction,
- that such particles (i.e. <0.1 µm) within the mass of ambient PM\(_{2.5}\) will be a small portion of the 2.5 µm PM, perhaps only 1/10th or 1/100th, and
- the PM\(_{2.5}\) guideline does not represent a value that is ‘safe’. Rather it is an acceptable and achievable objective to minimize health effects in the context of local constraints, capabilities and public health priorities (WHO, 2008).

Taking all the above into consideration the comparison of maximum particle number concentration emission (expressed as mass concentration) from printers as measured by Morawska et al. (2011) could be interpreted as indicating a potential health impact from exposure to laser printer emissions, if a person was close to a printer, was lower or about equivalent to the WHO PM\(_{2.5}\) ambient air quality guideline. That is, the risk of direct health effects is low.

However these deliberations are subject to many of the uncertainties levelled at the risk estimates of Hänninen et al. (2010) and should therefore be regarded as being low confidence. It is also emphasized that this analysis has the basic assumption the emissions from laser printers are physically and chemically the same as ambient PM\(_{2.5}\), this is likely not

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\(^8\) The particles from laser emissions are <0.1 µm (approximate mean size of 0.05 µm), this will be a small fraction of the particles measured in PM\(_{2.5}\).

\(^9\) The assumption that the average density of ambient PM\(_{2.5}\) particulates may be ~2g/cm\(^3\) comes from a brief literature search.
- Pitz et al. (2008) indicates the density of PM\(_{2.5}\) in German urban air is 1.05 – 2.36 g/cm\(^3\);
- Molener (2000) reports densities of PM\(_{2.5}\) for various haze and dust species to be 1.0-2.3 g/cm\(^3\);
- Computations by Hand and Kreidenweis (2002) indicated the average density of PM\(_{2.5}\) to be 1.85 \pm 0.14 g/cm\(^3\); and
- DeBell (2006) recommends a default density of 1.9 g/cm\(^3\) for sea salt.
the case. Nonetheless there is uncertainty regarding the ‘particulate’ nature of laser printer emissions and this analysis was undertaken to provide a simple, albeit rough, screening assessment of health risks should laser printer emissions biologically behave like fine ambient air pollution (i.e. PM$_{2.5}$).

6.3 Considering emissions as ‘VOC’

6.3.1 Occupational exposure standards

As discussed in Section 4.3 it is difficult to visualise the VOC/SVOC laser printer aerosol particulates described by Morawska et al. (2009) physically remaining as ‘particles’ for very long in the pulmonary system. It is more likely health effects, if any, associated with exposure to the VOC or SVOC aerosols will be determined by the inherent chemical properties of the VOC/SVOC rather than the ‘particulate’ nature of the aerosol. It is therefore relevant, in the context of screening risk assessment, to compare the concentration of printer emissions (as VOCs) with the Australian National Exposure Standards (NES) in order to judge potential health impacts.

Using the maximum 8 hr TWA particle concentration as measured 1 metre away from a printer by Morawska et al. (2011) a mean diameter of 40 nm, and assuming all the aerosol particulate was styrene, Morris (2011, Appendix A) calculated a mass concentration of 0.12 µg/m$^3$ for this substance. Styrene has the lowest exposure standard (213 mg/m$^3$) of the VOCs identified by Morawska et al. (2009) that have an Australian occupational exposure standard and therefore for this risk assessment can be considered as a worst case chemical.

Thus the 8 hour exposure particulate TWA if assumed to be all styrene aerosol is about 1.7 million times less than the occupational exposure limit. This margin of exposure is extraordinarily high, and sufficient to account for the uncertainties associated with the assessment and variable response amongst individuals. It can be concluded with a reasonable amount of confidence that direct acute or chronic health risk from laser printer emissions is negligible when printer emissions are taken to be primarily composed of VOC/SVOC aerosols.

It is recognised the general public may be exposed to laser printer emissions and that the use of occupational exposure standards in a screening risk assessment may be inappropriate due to exposure of potentially more susceptible sectors of the population than are usually present in a healthy workforce. This issue is addressed in Section 6.3.1 where indoor air criteria derived for the general public are used to characterise risk. Nevertheless the margins of exposure relative to occupational standards are so big that even the most sensitive person within the general population is very unlikely to experience direct health effects from exposure to laser printer emissions.

It is however recognised the full chemical nature of laser printer emissions and their toxicity is unknown and there may be low concentrations of substances with different toxicological potency than styrene. This uncertainty is addressed in Section 6.4.

6.3.2 Indoor air guidelines

The German Federal Environment Agency (GFEA) Indoor Guidelines for selected VOC/SVOC and TVOC (total volatile organic compounds) are shown in Table 1 below. The Agency sets two guideline values specifically for indoor pollutants (RW I and RW II) which conventionally differ by a factor of 10. RW II is described as an effect-related value calculated on current toxicological and epidemiological knowledge of a substance’s effect threshold which takes uncertainty factors into account. RW I is ten times lower than RW II
values and is the concentration of a substance in indoor air for which, when considered individually, there is no evidence at present that lifelong exposure is expected to have any adverse health impacts. For precautionary reasons GFEA consider there is need for action in the concentration range between RW I and RW II (Salthammer, 2011).

When the maximum 8 hr TWA particulate number emissions measured by Morawska et al. (2011) in an office environment are recalculated as mass particulate concentration and assuming they are a single compound, for example styrene (Section 6.3.1; Appendix A), the concentration (0.12 µg/m$^3$) is significantly below the stringent RW1 German indoor air guideline indicating low potential for health effects. Particularly since the guideline assumes continuous exposure (i.e. 24 hr/d) whereas the printer particulate exposure is for 8 hours. Taking into consideration the exposure time difference, the printer particulate concentration when expressed as mass concentration of styrene is 750 times less$^{10}$ than the stringent German indoor air guideline for styrene.

Table 1: German Federal Environment Agency indoor guidelines for selected substances
(Information taken from Salthammer, 2011).

<table>
<thead>
<tr>
<th>Substance</th>
<th>RW II a (mg/m$^3$)</th>
<th>RW I a (mg/m$^3$)</th>
<th>Set</th>
</tr>
</thead>
<tbody>
<tr>
<td>Toluene</td>
<td>3</td>
<td>0.3</td>
<td>1996</td>
</tr>
<tr>
<td>Dichloromethane</td>
<td>2</td>
<td>0.2</td>
<td>1997</td>
</tr>
<tr>
<td>Styrene</td>
<td>0.3</td>
<td>0.03</td>
<td>1998</td>
</tr>
<tr>
<td>Bicyclic terpenes (a-pinene)</td>
<td>2</td>
<td>0.2</td>
<td>2003</td>
</tr>
<tr>
<td>Aliphatic hydrocarbons (C9–C14)</td>
<td>2</td>
<td>0.2</td>
<td>2005</td>
</tr>
<tr>
<td>Monocyclic terpenes (d-limonene)</td>
<td>10</td>
<td>1</td>
<td>2010</td>
</tr>
<tr>
<td>TVOC b (set 2007)</td>
<td>Level 1: ≤0.3</td>
<td>No hygienic objections, No health-related concerns</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Level 2: &gt;0.3–1</td>
<td>Still no relevant health-related concerns</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Level 3: &gt;1–3</td>
<td>Some objections and distinct health issues</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Level 4: &gt;3–10</td>
<td>Major objections and health concerns</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Level 5: &gt;10</td>
<td>Not acceptable – serious health concerns</td>
<td></td>
</tr>
</tbody>
</table>

$^{a}$ See text for description.

$^{b}$ Salthammer (2011) reports other countries have TVOC indoor guidelines of 0.2 – 0.6 mg/m$^3$.

6.4 Threshold of toxicological concern

The caveat in the above screening assessments is that not all the chemical components of the printer emissions have been characterised. Indeed, given the myriad of printers and operating circumstances, and difficulty gathering sufficient material to be analysed, it is probably unlikely that full chemical characterisation will be achieved for all printers. The uncertainty regarding the identity of emission components present at low concentrations can be addressed using the threshold of toxicological concern (TTC) concept. The notion of toxicological thresholds for non-carcinogenic compounds underpins the technical basis for developing exposure standards for protection of public health throughout the world. For carcinogens, guidelines are established using a risk level regarded as trivial for public health.

$^{10}$ $750 = 30 \, \mu g/m^3 + 0.12 \, \mu g/m^3 \times 24hr/8hr.$
The TTC is based on consideration of toxicologically rich databases created over the last fifty years from safety tests, conducted to standard protocols and good science, submitted to regulatory agencies in the USA and Europe. After analysing the data on hundreds of carcinogenic and non-carcinogenic substances, the US Food and Drug Administration (FDA 1995) and others (Frawley, 1967; Munro et al., 1996, 1999, 2002; Kroes et al., 2000, 2004, 2005; Kroes and Kozianowski, 2002) concluded that long term chemical intake of 1.5 µg/person/day, is unlikely to result in appreciable health risk even if the substance was later found to be a carcinogen. For a ‘standard’ 70 kg person this equates to a dose of 0.02 µg/kg/day. There are however a few substances that contain either structural alerts for high potency genotoxic carcinogens (aflatoxin-like compounds, N-nitroso- and azoxy-compounds) or have high potency biological interactions (dioxins and steroids) for which a TTC would not be appropriate (Kroes et al., 2004). It is unlikely that such substances will be in laser printer emissions (see also Appendix B).

Since its inception, the TTC process has been used successfully by the US FDA and WHO in assessing the safety of certain chemicals at low levels in foods. This includes direct and indirect (i.e. contaminants) food additives (FDA, 1995; EC, 1997; Munro et al., 1999, 2002; Cheeseman, 2005; JECFA, 1997; Renwick, 2004). More recently the European Medicines Agency has adopted the TTC concept to assess genotoxic impurities in pharmaceutical preparations (EMEA, 2006; Muller et al., 2006). Use of the TTC has also been proposed for ingredients used in personal care products (Blackburn et al., 2005; Kroes et al., 2007; Carthew et al., 2009).

The application of TTC as a tool for screening risk assessment of chemicals in air was first proposed by Drew and Frangos (2007) and extended to airborne mycotoxins (Hardin et al., 2009) and aerosol ingredients in consumer products (Carthew et al., 2009). Because exposures that are effective for induction of cancer are invariably much lower than those required for other health end points the TTC for air toxics was first directed to development of a generic air carcinogen guideline value (Drew and Frangos, 2007). It was reasoned that compliance with a generic carcinogen guideline would be protective for the other health effects and such chemical exposure would not need to be subject to a detailed health risk assessment.

The TTC for chemicals in air has been further developed by classifying individual compounds into toxicity classes based on chemical structure rules (Cramer et al., 1978) and then applying the TTC assigned to each class to develop a concentration of no toxicological concern (CoNTC) for each of the Cramer chemical classes. These CoNTCs are summarised in Table 2 and are used by the Western Australia Department of Health in screening risk assessments for chemicals in air; they cover a number of health end points and represent an air concentration (µg/m³) that can be breathed for the majority of a person’s life without significant risk of harm (WA DoH, 2010). However the CoNTCs do not apply to metals and metalloids because these substances are not in the databases used for TTC development. The end points of odour and sensory irritation are also not included within CoNTC derivation.
Table 2: Concentrations of no toxicological concern (CoNTC)
(Information taken from WA Department of Health WA DoH, 2010)

<table>
<thead>
<tr>
<th>Chemical Class/ Toxico logical endpoint</th>
<th>CoNTC $^d$ (µg/m$^3$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cramer Class I $^a$</td>
<td>5</td>
</tr>
<tr>
<td>Cramer Class II $^c$</td>
<td>1.5</td>
</tr>
<tr>
<td>Cramer Class III $^b$</td>
<td>0.2</td>
</tr>
<tr>
<td>Carcinogenicity</td>
<td>0.03</td>
</tr>
</tbody>
</table>

$^a$ Class I is comprised of substances of simple chemical structure with known metabolic pathways and innocuous end products which suggest a low order of toxicity
$^b$ Class III substances whose structure or presumed metabolism permit no strong presumption of safety, or even suggest significant toxicity (Cramer et al. 1978).
$^c$ Class II contains chemical structures that are intermediate.
$^d$ The CoNTC is an air concentration that can be breathed for the majority of a person’s life without significant risk of harm. In calculating CoNTCs it is assumed 50% of exposure may be via inhalation and that there is 100% pulmonary absorption.

As discussed in Section 6.3.1 and Appendix A the maximum 8 hr TWA particulate number concentration measured by Morawska et al. (2011) in an office environment when assumed to be a single compound, for example styrene, and recalculated as mass of particulate gives a concentration of about 0.12 µg/m$^3$. It is standard regulatory practice to adjust the measured exposures to match the time periods of the guideline. Thus this exposure for 8 hour/d and 5 days/week is equivalent to approximately 0.03 µg/m$^3$ assumed continuous exposure $^{11}$. Furthermore if it is conservatively assumed an unidentified or uncharacterised (i.e. no guideline or health information) compound within the emissions may comprise as much as 10% of the total emissions, the concentration of that substance may be about 0.003 µg/m$^3$. By comparison with the CoNTCs in Table 2 it can be seen health impacts are unlikely.

Although there may be uncertainty in the identity, toxicity and concentration of individual compounds in laser printer emissions, the conservatism embedded in the derivation of CoNTCs and in assumptions of exposure bias the screening assessment towards protection of human health. Therefore for estimated exposure concentrations that are less than the CoNTC there is high confidence of low likelihood of direct adverse health effects.

$^{11} 0.12 \text{ µg/m}^3 \div (24 \text{ hr/8 hr x 7d/5d}) = 0.028 \text{ µg/m}^3$, rounded to 0.03.
7. Summary

For this brief review no epidemiology studies directly associating laser printer emissions with adverse health outcomes were located.

Laser printer emissions are different to toner particles and the particulates of urban ambient air pollution. Recent research indicates emissions from laser printers are primarily aerosol condensates of VOCs or SVOCs. Thus it would be logical to expect possible health effects to be more related to the chemical nature of the aerosol rather than the physical character of the ‘particulate’ as such emissions are unlikely to be ‘particulates’ after they contact respiratory tissue.

In this report a comparison of the maximum 8 hour TWA levels of exposure to laser printer emissions, as reported by the research commissioned by Safe Work Australia (Morawska et al., 2011) has been made with:

• WHO Air Quality Guidelines,
• Australian National Exposure Standards,
• the German Federal Environment Agency Indoor Guidelines, and
• toxicological thresholds of concern for concentrations of chemicals in air.

These screening risk assessments are conservative in order to take account of uncertainty in the exposure, the nature of the emissions, and the identity/health effects of emission components. Each of the analyses indicates the risk of direct toxicity and health effects is negligible; however people who are responsive to unusual or unexpected odour may detect/react to the presence of printer emissions.
References


http://vista.cira.colostate.edu/improve/publications/graylit/014_AerosolByNeph/AerosolbyNeph.pdf.


APPENDIX A
Examining Data on Laser Printer Particle Emissions (Morris 2011)

The available data for exposure to laser printer emissions has the units of particle number per volume of air (particles/cm\(^3\)) however exposure guidelines have the units of mass per unit of air (µg/m\(^3\) or mg/m\(^3\)).

Assuming the particle is spherical the particle number concentration can be converted to mass concentration using Equation 1.

\[
\text{Particle Concentration (mg/m}^3\text{)} = \delta \frac{4}{3}\pi r^3 n \quad \text{.........Equation 1}
\]

Where:

\[
\frac{4}{3}\pi r^3 = \text{Volume of sphere}
\]

\(r = \text{Average radius of laser printer particle (20 nm = 20 \times 10^{-9} m). See below for rationale.}\)

\(\delta = \text{Density of particle (mg/m}^3\text{). This is dependent upon assumptions of particle constituency.}\)

- If assumed to be PM\(_{2.5}\) (\(\delta = 2 \text{ g/cm}^3\), or \(2 \times 10^9 \text{ mg/m}^3\)). See Section 6.2.1.
- If assumed to be styrene (\(\delta = 9.1 \times 10^8 \text{ mg/m}^3\)). See below and Section 6.3.1.

\(n = \text{Particulate number concentration as 8 hr TWA (4 \times 10^9 \text{ particles/m}^3). From Morawska et al. (2011); this concentration is considered to be reasonable worst case exposure. See Section 6.1.}\)

Morawska et al. (2011) concluded the particle diameter associated with the operation of laser printers in office locations is within the ultrafine size range of less than 100 nm diameter. This is consistent with previous measurements in which the printer emission particle diameter was 65 nm at the start of a print run but 28 nm at the end (Morawska et al. 2009). He et al. (2007) evaluated three printers and found the mean particle count median diameter was 76 ±11 nm, 46 ± 9 nm and 40 ± 4nm. These workers also found that while old cartridges generated a lower total number of particles they emitted a greater number of smaller particles, below 25 nm in diameter. Taking all the above into consideration it has been assumed for the purposes of screening risk assessment that the diameter of laser printer emission particulates is 40 nm. It should be noted that an increased particle size will result in an increased particle mass when Equation 1 is applied. The uncertainty around the size of the printer emission particulates can be quantified as follows; increasing the particulate diameter by two fold (e.g. from 40 nm to 80 nm) will increase the mass concentration by 8 fold. This uncertainty is easily accounted for by the margin of exposures calculated in the main body of this report.

Converting the particle concentration units enables simple risk characterisation using existing exposure guidelines if it is assumed the laser printer emissions consist of a single compound. For example, styrene is one of the VOCs commonly identified in printer emissions; at 213 mg/m\(^3\) it has the lowest 8 hr TWA guideline of any of the VOCs identified by Morawska et al. (2009) that have an Australian occupational exposure standard. It can therefore be considered as a worst case chemical for the purposes of a screening risk assessment when it is assumed all the printer particulates may be aerosols of styrene (see also Section 6.3.1).

In this simplified model, where it is assumed all the particles emitted are styrene, and gaseous emissions are negligible, the mass concentration of styrene after using equation 1 is 0.00012 mg/m\(^3\). This is only a very small fraction of the styrene NES (213 mg/m\(^3\)) indicating very low likelihood of direct adverse health effects.

Numerous organic substances, including carcinogens, have exposure standards (TWA) which are orders of magnitude higher than 0.00012 mg/m\(^3\) (see the Adopted National Exposure Standards for Atmospheric Contaminants in the Occupational Environment [NOHSC:1003(1995)]).
APPENDIX B
Health Hazards from Toner Particles

There have been many studies investigating the toxicological hazards of toner powders, many are premarketing, regulatory safety studies required for hazard classification. Toner powder is a complex mixture of chemicals (polymer binder, ferric oxide, pigment, carbon black, anti-tackifier, charge control agent, and mobile agent) that tends to be significantly larger than the fine particulates emitted by laser printers.

Toner powder toxicological information is relevant for occupational exposure to dusts in toner manufacturing, cartridge assembly and recycling, cartridge handling or laser printer maintenance, but not to consumers from laser printer emissions.

Bai et al. (2010) dosed mice intratracheally with toner powder (40 mg/kg, 4 times 2 days apart, particle size ~10µm) and undertook evaluations for up to 12 weeks post instillation. The total dose was high (3.36 mg/mouse, being about 2.5% of lung wet weight). It was found that there was a prolonged pulmonary inflammatory response, pulmonary histopathological changes and significant reduction in weight gain over the observation period. The high dose hinders data interpretation in relation to potential human health effects.

Ewers and Nowark (2006) have reviewed the literature on health effects associated with printer emissions. In relation to toner powder they argue it cannot be emitted from laser printers during operation and it is not possible to predict indoor air concentrations of harmful substances that may be in toners. Nevertheless these authors review a large body of toner toxicological information. They conclude the biological effects produced by the inhalation of toner dust are primarily related to particle properties (insolubility and persistence in the biological environment; particle size distribution, specific gravity). The chemical composition of the polymer matrix, the pigment and other ingredients seem to be of no relevance. They identified a chronic no observed effect level in rats of 1 mg/m³ and attributed particle accumulation, and lung damage to overload of lung clearance mechanisms (similar to that observed in the Bai et al. (2010) experiment). They conclude “So far, there have been no scientifically established indications that the operation of modern laser printers and copiers in offices and households leads to an increased health-relevant exposure caused by toners and VOC. Human biomonitoring examinations did not indicate an increased internal exposure to harmful substances for persons who work intensively with laser printers and copiers.”

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12 Assuming 55% lung deposition from breathing 10µm particles, the 40mg/kg intratracheal dose (4x) equates to a human inhaling ~140 mg/m³ 6hr/d for 4 days (SWA 2009, Appendix 2).