

**Call for Evidence: Nanotechnologies and Food**

Evidence from

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I am a lung particle toxicologist and so I will start there. Nanoparticles are ubiquitous in the environment from natural and anthropogenic sources and have been throughout evolutionary time. Combustion-derived nanoparticles (CDNP; soots) are present in air, and to a greater extent in the last few hundred years than in previous times, and they are considered to drive a number of adverse effects in the lungs and cardiovascular system that are well-documented. These arise, in the opinion of many experts, primarily from inflammatory effects in the lungs. There is a hypothesis that there is also translocation of such NP to the blood and the brain and there is restricted evidence that there is limited translocation, using animal and experimentally-generated NP. There is no evidence currently that translocation of NP out of the lungs occurs in humans or leads to any adverse effects, although it is possible, even likely. I personally have come to believe that there is minor redistribution of very small particles from the lungs. The question is whether this translocation is important in any adverse effects, compared to the systemic effects caused of the inflammation at the primary site of deposition in the lungs, acting on other sites like atherosclerotic plaques or even the brain. This remains unanswered at the moment.

For manufactured NP, effects will depend on exposure and on the intrinsic hazard (e.g. surface reactivity, fibrous shape). The majority of bulk-produced NP (silica, alumina, TiO<sub>2</sub>, carbon black) presently are low toxicity but there is concern over carbon nanotubes and other high aspect ratio nanoparticles (HARN) because of their superficial similarity to asbestos. There may be higher hazard NP to which there will be exposure and so there must be vigilance but this can be foreseen with adequate testing. All-in-all it seems unlikely that there will be any large-scale pandemic of lung disease from bulk-manufacture NP if sensible hygiene standards are used but we must be watchful for increasing production of unusual NP like HARN and some metals (Copper, silver, possibly).

The gut can be considered to have undergone similar evolutionary forces to allow it to deal with nanoparticles over evolutionary time. The gut will certainly be evolved to deal with natural particles which has always been present on food and probably only in the last few hundred years will these have been thoroughly removed by washing prior to preparation/eating. So nanoparticles in soils will be able to be dealt with by the gut. A key question is whether the gut has evolved to deal with the traffic of CDNP from the lungs that it encounters from the normal process of mucociliary clearance. This delivers 99.9% of all particles that deposit in the airspaces to the gut. It's true that the stomach and its acid environment stands a key gatekeeper and that all particles will be acid-treated prior to entering the gut, but many particles will not be dissolved by the acid in the stomach and will continue, albeit with surface modification due to acid treatment, to the intestine. One of these modifications could be to the aggregation status (I don't know if it would cause more or less aggregation) and that could be important in subsequent effects on the intestine. The PM<sub>10</sub> epidemiology literature, which documents in large part the adverse effects of CDNP, since the CDNP is probably the most pathogenic fraction of the PM cloud, does not pick up an adverse effect on the gut. This may be a result of some quirk of reporting/ death

certificates etc but, taken at face value, it does suggest that the delivery of CDNP to the gut from the lung does not have an adverse effect on the gut.

The question is whether any manufactured NP might have such an effect. The ante is greatly increased when the NP are added to food purposely. All toxicity is dose-related and so the likelihood of an adverse effect increases with dose and so adding NP to food definitely increases the likelihood of adverse effects. It is to be hoped that the companies that make the food have testing procedures in animals that demonstrate no ill-effects – such data should be made available to the Committee. The likely effects might include pro-inflammatory effects and immunological abnormalities.

Another problem lies with the normal flora of the gut , which could well be unbalanced if there was selective toxicity toward commensals – silver NP seem a particular threat in this area.

There is a suggestion that asbestos exposure is linked to cancer of the stomach and colon although this is disputed, and would arise from the delivery of fibres to the gut via mucociliary clearance. It is just possible that a HARN might be especially active in this regard.

Use of NP in food will greatly increase the likelihood for release of NP into the environment during manufacture and disposal and in human waste, where there might be ecotoxicological effects that are not in my area of expertise.

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