

*Hot particles no longer*

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Among all plausible safety concerns, the question of 'hot particles' was the most rapidly dealt with by the scientific community (around 1976). The concern has been kept alive (mostly by LeRoy Moore) for more than 40 years despite its rapid dismissal as a special risk by the scientific community.

It is *not* that 'hot particles' do not exist (they will be defined below and are present around many radioactive sites); it is that (i) they are quite unlikely to be inhaled, and if they were, (ii) they are not appreciably more dangerous than other means of ingesting Pu (around Rocky Flats), or other  $\alpha$ -emitting radionuclides. These other pathways to ingestion are already included, for example, in the RESRAD software used by the Office of Legacy Management of the Department of Energy to model (via various scenarios, discussed elsewhere) radiation exposure around and within the Rocky Flats National Wildlife Refuge.

RESRAD is discussed at more length in the document [From radiation dose to cancer risk](#).

Because good, clear reports on the topic exist, it is easier to simply quote from them than to develop a detailed response.

*What are 'hot particles'?*

On the EPA web site [1] is a clear report (in parts technical) entitled *Health effects of alpha-emitting particles in the respiratory tract: Report of Ad Hoc Committee on "Hot particles" of the Advisory Committee on the biological effects of*

You will need to search for this by name: 'alpha-emitting particles in the respiratory tract' should do it

*ionizing radiation*, prepared [2] by the National Academy of Sciences and the National Research Council for the EPA in 1976.

Since the early 1950's, various groups with responsibility for determining the effects of radiation sources on human health have recognized the possibility that radioactive material deposited in tissues of the body as high specific activity [**very radioactive-Ed**] particles might be a greater health hazard than the same source distributed more homogeneously. This has been referred to as the "hot particle" problem. In 1974 Cochran and Tamplin hypothesized that the intense and highly localized dose from inhaled insoluble plutonium particles larger than a specified size causes greater tissue damage, and is therefore more carcinogenic, than more uniformly-delivered irradiation. On this basis Cochran and Tamplin advocated a 115,000-fold reduction in the current radiation standards governing exposure to insoluble alpha-emitting ("hot") particles.

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Large particles never make it into the lungs; tiny particles are breathed in and out again (and are too small to statistically contain much Pu anyway). So the issue could arise for particles only in a 'window' of particle size.

### *A long history of negative findings about dangers of hot particles*

#### The 1976 EPA report discussed above

... The evidence does not support the NRDC petition for a special, lower radiation protection standard for inhaled alpha-emitting particles. The current state of knowledge about the 'hot particle' problem can be summarized as follows:

1. In animals, all experimental data so far obtained indicate that when insoluble plutonium particles are inhaled, the major radiation dose in the lungs occurs in the pulmonary (i.e., alveolar) region. The principal delayed effect in the lung of breathing these particles is induction of alveolar cancers. An analysis of mortality from these cancers in beagle dogs indicates that if there is a hot-particle effect, Cochran and Tamplin have overestimated the cancer risk per particle by at least two orders of magnitude. However, analyses indicate that the observed lung cancer mortality in these dogs can be adequately accounted for by the conventional method of averaging the absorbed alpha radiation dose over the entire lung. Therefore, it is concluded that if there is a "hot particle" risk, it is small by comparison with the lung cancer risk attributable to the generalized alpha radiation.
2. In human beings, epidemiological evidence gained from experience with inhalation of alpha-emitting radon daughters and with

One 'order of magnitude' =  $10^1$  = factor of 10; two orders of magnitude =  $10^2$  factor of 100, etc.

external X or gamma irradiation of the thorax strongly suggests that the radiocarcinogenic sensitivity of the tracheobronchial region is greater than that of the alveolar regions where inhaled plutonium is retained. Therefore, we would not expect the human cancer risk from alpha irradiation of the deep lung tissues to be underestimated by applying risk factors obtained from human experience with cancer induced by irradiation of the lining of the bronchial tree.

3. Current evidence indicates that the cancer hazard from insoluble particulate plutonium deposited in the lungs is not markedly greater than would be caused by the same quantity of radioactivity distributed more uniformly. The experimental evidence suggests that the carcinogenic response is more a function of the amount of radioactivity in the lung than of its distribution.

There are useful and clear (if technical) appendices in the report.

A 1987 German article [3] came to somewhat surprising conclusions:

The biological relevance of local megadoses from particulate  $\alpha$ -emitters was discussed and studied in the seventies. . . . Experimental findings from animal studies and theoretical considerations lead to the hypothesis that a strongly inhomogeneous irradiation induces less health detriment than a homogeneous irradiation. The main argument being that in the case of  $\alpha$ -particles, a clearly defined tissue area is irradiated at dose rates leading to acute cell death or total loss of proliferative capability. Outside this area, the dose is practically zero. Hence, almost all ionizations and excitations occur in dead tissue . . . The wasting of a large fraction of the radiation dose on dead cells is thought to cause the relative innocuousness of  $\alpha$ -hot particles. . .

In several facilities involved in the US military bomb program, groups of workers were exposed to measurable amounts of airborne, particulate plutonium activity. A population of 25 workers involved in the Manhattan Project showed initial lung depositions of plutonium in the range of 15 kBq. This would correspond to about 100,000 hot particles with a diameter of  $1\mu\text{m}$ . [In] 1972, more than 25 years after the contaminations, the average body burden still amounted to about 3.7 kBq  $^{239}\text{Pu}$  with about 5 to 10% residing in the lung. No health detriments traceable to these extremely hot particle exposures have been found so far.

Another group was exposed during a fire in Rocky Flats on October 15th, 1965. The initial lung burden corresponded to about 10,000 to 100,000 plutonium particles of the size generated in the fire. . . . Although a longer follow-up of this population has to be awaited to draw final conclusion, the lack of lung diseases in the first 15 years allows to refute some of the hypotheses on the extreme radiotoxicity of hot particles.

They conclude, "Contrary to the impression at first glance, radiation from hot particles was never shown to be more

I find this interesting but not very plausible: as time goes on, what happens to the killed cells? Don't they get replaced by new, living cells (possibly with mutations)? So, I don't think this argument can result in a steady-state arrangement.

radiotoxic than the same activity uniformly distributed in the organ. Both from theoretical considerations, from animal studies and from human epidemiology, it seems that wasting of dose on dead cells introduces a safety factor. Therefore, the general conclusion stressed also in review papers by Bair et al. and Feinendegen et al. is that, with increasing inhomogeneity, the detrimental effects of ionizing radiation to the lung tend to decrease.”

Harrison’s 2003 article [4] in the Journal of Radiological Protection entitled *Carcinogenic risk from hot particle exposures—has ICRP got it right* remarks

The argument has become very familiar—that radionuclides introduced into the environment from nuclear installations, fall-out from weapons testing, or whatever source, are responsible for substantial increases in cancer rates, and, because current risk estimates do not support this conclusion, they must be very wrong. It is argued that there must be some way in which low levels of artificial radionuclides, levels that result in tissue doses lower than from naturally-occurring radionuclides, pose a risk that is yet to be appreciated. One obvious problem with current risk estimates, it is suggested, is the simplistic averaging of doses from hot particles—see, for example, the home page of the Low Level Radiation Campaign website ([www.llrc.org](http://www.llrc.org)) . . .

It can be concluded that, on current evidence, hot particle effects do not provide a mechanism for doses from environmental levels of artificial radionuclides to be more effective in causing cancer than larger doses from naturally-occurring radionuclides. The ICRP approach of averaging dose to target cells and tissues appears to give reasonably reliable estimates of risk.

Charles, Mill, and Darley [5] in 2003 report in a 23-page careful review with 86 references remark: “These conjectural arguments [of extra carcinogenicity for hot particles—Ed] were reviewed by a number of workers. Their conclusions supported the continued use of mean organ dose as a predictor of cancer risk. Albert, whose work on skin cancer induction in rat skin was the basis for this conjecture, has also criticised the rationale and conclusions of Tamplin and Cochran.”

“In summary, in very broad terms (within a factor of  $\sim \pm 3$ ) the results of a large number of animal studies and a growing number of in vitro studies are in agreement regarding the lack of evidence to support a large hot-particle enhancement factor. Human evidence is lim-

ited but does not support any significant hot-particle enhancement. All of this is in stark contrast to the claims made more than 30 years ago, which fuelled so much concern, that this could be as much as five orders of magnitude.”

Section 3.6 (Carcinogenic risks of particulates) of the 2004 English CERRIE report [6] concluded

16 The Committee examined the suggestion that spatially non-uniform radiation exposures, from radioactive particulates, may be much more carcinogenic than uniform exposures throughout tissue volumes. It therefore commissioned a literature review of the possible carcinogenic effects of particulate radioactive materials, which has since been published. . . The review concluded that, on current evidence, the conventional assumption of average dose to a tissue or relevant component should provide a reasonable estimate of carcinogenic risk within a factor of three up or down of the central estimate . . .

18 Data on lung cancer mortality following occupational inhalation of plutonium aerosols, and on the incidence of liver cancer and leukaemia due to Thorotrast administration for clinical diagnoses, did not support a significant risk enhancement factor for particles. Very few animal studies, including mainly lung and skin exposures, provided any indication of a particle enhancement. Some recent in vitro malignant transformation experiments provided evidence for an enhanced cell transformation for ‘hot’ particle exposures but the effect was modest. However, most doses were very high: few studies concerned doses below 100 mGy—the area of interest to the Committee.

19 It appeared from the literature review that there was no convincing evidence from worker, animal or in vitro studies that ‘hot’ particles that delivered high doses to a small surrounding volume of tissue were more hazardous than more uniform irradiation. However, the situation for ‘warm’ particles that delivered lower doses was less clear due to the paucity of direct observations.

20 Most [10 of 12–Ed.] of the Committee agreed that little information existed which supported enhanced risks from exposures to ‘hot’ particles, although most studies used relatively high doses. Two members considered that the possibility that ‘warm’ particles presented a high risk could not be ruled out. The remainder of the Committee remained unconvinced or uncertain of this hypothesis mainly because of the paucity of evidence presented. . .

The last definitive discussion of the health impacts of ‘hot particles’ I could find occurs in a 2007 journal article (*i.e.*, 21 years after the Chernobyl incident (which itself rekindled concern and re-examination of hot particle issues) entitled *Hot particle dosimetry and radiobiology—past and present* [7]

The claim of increased carcinogenicity was referred to as the 'hot particle hypothesis'. After a decade of intensive experimental and theoretical investigation an ICRP review of the biological effects of inhaled radionuclides in 1980 refuted this claim in the context of inhaled hot particles. The 'hot particle hypothesis' in the context of skin exposure has been refuted by a series of skin cancer induction experiments in mice and in cell studies of induced malignant transformation. The ICRP has maintained its advice that the use of mean organ or tissue dose is appropriate for the evaluation of carcinogenic risk for radiological protection purposes.

... Our understanding of the radiobiological effects of hot particles, and our ability to measure relevant doses from them, has increased dramatically over the past 25 years.

The likelihood of a casual encounter with a hot particle in the environment, even in the environs of Dounreay is very low. ... It is possible to predict significant health effects but only by making assumptions which have a low probability, such as exceptionally long residence/transit times. This low probability must be compounded with the already very low probability of encountering a particle. The challenge for regulators remains, as in other situations of environmental contamination, to determine the extent to which remediation is required in the light of potential health effects with a low probability of occurrence.

Dounreay is a now-decommissioned Scottish nuclear installation with 5 reactors. Chunks of old fuel rod washed ashore on public beaches in 2007, provoking intense investigation of 'hot particles' in the United Kingdom.

### *Takeaway messages*

- Hot particles (small, highly radioactive  $\alpha$ -particle emitting specks of dirt or other material) definitely exist around radiation-contaminated sites. They were thought in the early 1970s to be especially dangerous via inhalation.
- Journal-published research work from the 1970s through 2007 has repeatedly demonstrated experimentally that inhaled Pu is *not* significantly different than other sources of Pu ingestion. Some argue that high *local* tissue doses if 'hot particles' are actually inhaled kill surrounding cells which nonetheless 'screen' living cells farther away, *reducing* the harmful effect of ionizing radiation in the lung.
- Inhaled Pu is *already included* in the RESRAD software used to describe Pu exposure and cancer risk within and around Rocky Flats, which have been assessed as

posing excess cancer risks of between 1 in 100,000 and 1 in 1 in 1,000,000.

## References

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