to participatory breeding options, but an aid to incorporate specific traits in a site-consonant mode of expression. Farmers are firm that high yields alone are not adequate; they prefer varieties satisfying their taste though not high-yielding. Under such circumstances, modern technologies with emphasis on high yields alone would be inappropriate. It has been further emphasized that participatory varietal improvement initiatives must be supplemented with necessary R&D to produce a better farm technology and maintain it green. Examples include no-till farming, mulch-till farming, integrated nutrient management, rotational grazing (moving livestock to different pastures to reduce the build-up of manure, instead of collecting manure) and organic production[8].

However, the need for relevant basic research and innovative options has to be admitted. An example, in the context of improving varieties by enhancing expression of quantitative traits, is that of characterising G in quantitative terms (a possible approach could be through molecular genomics?) in the model $P = G + E + (G \times E)$, where P, G and E are the phenotype, the genotype and the environment and $(G \times E)$, the genotype–environment interaction. Such basic research needs to be complemented by mission-oriented strategic research at the target areas to accord basic results an application potential. In India, basic research is mostly confined to university-based science departments and some research institutes. Applied and strategic research is done in applied science departments of universities, some private organizations and NGOs. The field extension of the research results is carried out by government extension agencies and to a limited level by NGOs and individual agencies. The resulting benefits to the unreached farmers are additive at best. With an ideological and structural reconstruction, such additive benefits stand a high chance to become multiplicative. Can we then say that revitalizing tradition synergized with science would provide a green framework for improving the lot of poor and unreached farmers?

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Plutonium dispersal and health hazards from nuclear weapon accidents†

*Zia Mian, M. V. Ramana and R. Rajaraman**

We analyse the dispersal of plutonium into the atmosphere and consequent biological hazards from nuclear weapon accidents. Such accidents involving nuclear weapons could be caused, for example, by missile and jet fuel fires and explosions. We use the wedge model of aerosol dispersal to estimate the amount of plutonium that would be inhaled by a surrounding population and the resulting radiological damage in the form of increased cancer fatalities in the event of such an accident. These considerations are then applied to possible accidents in South Asia and inferences drawn.

IN this article we analyse nuclear weapon accidents that result in the dispersal of plutonium into the atmosphere and its impact on public health. Our motivation for doing so derives from the current situation in the South Asian subcontinent. Both India and Pakistan tested nuclear weapons in May 1998. Although there is no public information on how many weapons each country has or the state of their deployment, the general impression is that neither India nor Pakistan has yet fully deployed its nuclear weapons. But it is possible that this comparatively less dangerous situation may change in

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the not-too-distant future and weapons may be deployed.

Deployment carries a number of dangers. Apart from command and control issues, there are possible accidents involving assembled nuclear weapons, with the risks increasing when the weapons are deployed on delivery vehicles (missiles, airplanes, etc.) carrying highly explosive liquid fuels, and further increasing when the weapon systems are kept on a high state of alert. Such accidents could be caused by any number of factors, including air crashes, fires and missile fuel explosions; accidents can also happen in storage and during transport of nuclear weapons.

Nuclear weapons and their delivery vehicles are complex engineering systems with highly combustible, explosive and hazardous components. It is only to be expected that accidents will occur from time to time. Indeed, since the beginning of the nuclear age there have been numerous accidents. It is believed that there were at least 230 accidents involving the nuclear weapons of the US, the Soviet Union and the UK between 1950 and 1980 (ref. 1). Another assessment lists 383 US naval nuclear weapon accidents alone[2]. An official summary of nuclear weapon accidents released by the US Department of Defense in 1981 admits to 32 serious accidents just between 1950 and 1980 (ref. 3). This includes a number of instances where the high explosive (HE) in nuclear weapons has burnt or detonated and led to contamination[4]. Information about accidents in the erstwhile Soviet Union is harder to obtain, but one source lists over 25 serious nuclear weapon accidents there[5].

Both India and Pakistan have been developing and testing a number of ballistic missiles. There are no reports yet of accidents involving nuclear weapons or ballistic missiles in either country. But given the long history of accidents in the other nuclear weapon states, one is not being unduly alarmist in suggesting that if India and Pakistan were to deploy their nuclear weapons, they too shall face the risk of such accidents, particularly since India's *Prithvi* and Pakistan's *Ghauri* missiles are propelled by highly volatile hypergolic liquid propellants[6]. It is not possible to reliably quantify the probability of such 'Beyond Design Basis Accidents'. Even if it is small, it is certainly not zero and if such an accident happens, its consequences are serious, as we shall show.

Although motivated by these concerns, the body of our work does not involve strategic or military issues. Rather, we study the contamination and biological hazards of plutonium dispersed from possible nuclear weapon accidents. We begin by classifying the different categories of accidents that could be triggered by external events like missile fuel explosions, fires and collisions. Then we focus on one category in which the HE in the weapon detonates leading to the dispersal of substantial amounts of plutonium into the atmosphere as an aerosol. We model the spread and deposition of this contamination cloud and estimate the amount of plutonium that would be inhaled by the surrounding population. Next we assess the health hazards of such plutonium inhalation and estimate the ensuing risk of cancer fatalities. Then we apply these general considerations to hypothetical examples in South Asia followed by a discussion of the results and their implications.

Plutonium dispersal analysis

Categories of accidents

A fire or fuel explosion near a nuclear weapon could lead to a range of consequences depending on the source and intensity of the fire, as well as the design of the weapon and its high explosive. It may be useful to classify such incidents into four categories of increasing health risk to members of the general public:

1. The HE in the nuclear weapon does not burn or detonate; the fissile material is unaffected.
2. The HE in the weapon catches fire and burns but does not detonate. This could lead to melting and fragmentation of the fissile material core.
3. The HE detonates and converts the fissile material into aerosol. However, there is no nuclear yield.
4. The HE detonates and causes a nuclear explosion.

An example of the first category of accidents is the 1980 Titan II Intercontinental Ballistic Missile (ICBM) explosion where the fuel tank of the missile exploded, catapulting its 9 megaton warhead about 200 m. Fortunately, it was recovered intact[7].

More serious because of the distinct possibility of plutonium contamination are accidents of the second category, where the fire or explosion leads to the burning of the HE inside the weapon. Of the 32 cases of US nuclear weapon accidents for the period 1950–1980 in the official list released by the US Department of Defense, there were 8 incidents of HE burn, half of which led to plutonium contamination (see ref. 4). An example of this was the accident at the McGuire Air Force Base, where on 7 June 1960 a BOMARC missile was destroyed when a high-pressure helium tank exploded and ruptured the fuel tank (see ref. 4). It is significant that the missile was reportedly in a 'ready storage' condition, permitting launch in two minutes when the accident occurred. Although safety devices prevented the HE from detonating, it did burn and along with it the entire warhead containing plutonium, enriched uranium and bottled tritium. The use of water in fire-fighting led to further spread of this radioactive material, leading to

contamination of the ground beyond the area beneath the molten debris. Nevertheless, the plutonium contamination was by and large limited to within 30 m from the explosion.

The release of plutonium into the environment carries a range of public health risks[8]. The two primary routes of exposure are ingestion and inhalation. Ingestion of plutonium is a less significant risk since almost all of the plutonium is excreted within a few days[9]. The more serious risk comes from inhalation of very small plutonium particles (a few microns in diameter), which can stay imbedded deep in the lungs typically for periods of the order of a year. In accidents like the one at the McGuire Base, where the HE did *not* detonate, the estimated amount of plutonium converted into respirable aerosol is only about 0.05–0.07% (ref. 10). Essentially all of the plutonium in the form of larger particles will remain within a hundred metres or so. Due to this small amount of aerosol and short range of dispersal, accidents of the second category have limited public health impact. There is, however, a hazard to those in the immediate vicinity and those involved in decontaminating the affected area.

Category 3 accidents – HE detonation and Pu dispersal: The third category of accidents consists of those where the HE inside the weapon actually detonates. The most famous examples in this category are the accidents at Palomares, Spain in 1966 and Thule, Greenland in 1968. In both cases, the aircraft carrying nuclear weapons crashed and the HE surrounding the nuclear core detonated. This led to dispersal of plutonium over a large region[11]. In such accidents effectively all of the plutonium will be oxidized into plutonium oxide (PuO_2) and aerosolized[10]. Of this aerosol, about 20% will be particles of respirable size[12]. This aerosol will rise with the hot gases created by the explosion, mix with the air and spread. Any prevailing wind could transport it to considerable distances. The detailed concentration profile of the aerosol as a function of time will be complicated and depends sensitively on the specific parameters associated with the accident event. These factors include the power of the explosion, the prevailing winds, the terrain, particulate matter in the atmosphere to which the aerosol may become attached and so on.

To get an order of magnitude estimate of the dispersal of plutonium, we employ a simple analytical model that has been used previously for estimating the effects of a nuclear weapon accident[13]. This is the 'wedge model', developed originally as part of the American Physical Society's study on light-water reactor safety[14].

The wedge model makes some simplifying assumptions about the aerosol concentration profile. Upon explosion, the PuO_2 aerosol will rapidly mix upward within the mixing layer of the atmosphere, which typically has a height H between 300 and 2500 m. The model assumes that this height remains constant as the plume moves downwind in a fixed direction and at a constant speed. Its crosswind spread (i.e. perpendicular to the wind direction) is taken to be an arc subtending a wedge angle θ at the explosion point. This angle generally varies from 0.05 to 0.3 radians.

The aerosol cloud will move downwind at the wind velocity u, and will be centred after time t at a distance $r = ut$ with some radial (downwind) spread $d(r)$. This geometry is illustrated in Figure 1, where the annular segment gives the horizontal section of the plume at some instant of time t. Within this plume-volume of height H, angular width θ, and thickness $d(r)$ the aerosol concentration $\rho(r)$ is assumed to be uniform at any given time, although it will decrease with r because of spreading in width and deposition of the aerosol on the ground.

Let $Q(r)$ be the amount of plutonium present in the plume at distance r (i.e. after time $t = r/u$). Assuming that the amount deposited on the ground is proportional to the amount present in the atmosphere, we have.

$$Q(r) = Q_0 \exp(-r/L), \tag{1}$$

where Q_0 is the total mass of plutonium that became aerosolized into a respirable form and the length L represents the average distance travelled by an aerosol particle before it gets deposited. Clearly

$$L = Hu/v, \tag{2}$$

where v is by definition the deposition velocity. The amount deposited per unit area is:

$$\sigma(r) = \frac{-1}{r\theta}\frac{dQ}{dr} = \frac{1}{rL\theta} Q_0 \exp(-r/L). \tag{3}$$

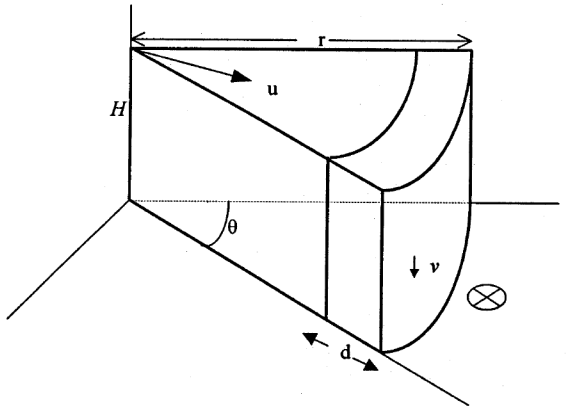

Figure 1. Schematic representation of the plume configuration at some instant of time in the wedge model. The aerosol-contaminated air is in the annular segment.

The plume has a volume $Hr\mathrm{d}(r)\theta$, and hence the concentration is:

$$\rho(r,\theta,z)=\frac{1}{rH\theta \mathrm{d}(r)}Q_0\exp(-r/L). \tag{4}$$

Now, consider a person (shown by a cross in Figure 1) who is about to be immersed in the cloud. Since the plume has thickness $\mathrm{d}(r)$, it will pass the person in time $\tau = \mathrm{d}(r)/u$. If her breathing rate is b m^3/s, she will breathe in a volume $b\tau$ containing an amount of plutonium given (in mg) by:

$$m(r)=\frac{Q(r)}{Hr\mathrm{d}(r)\theta}\frac{b\mathrm{d}(r)}{u}=\frac{Q_0 b}{Hru\theta}\exp(-r/L). \tag{5}$$

Notice that this result is independent of the thickness $\mathrm{d}(r)$.

There is now a considerable amount of theoretical and empirical support for the assumption that the biological risk from radiation exposure is a linear function of radiation dose at low doses[15]. Then, if a given dose is shared among N people, the risk of cancer death per person is reduced to $1/N$, but since each of the N people now suffers this risk, the total probable number of cancer deaths remains the same. Thus, in the linear approximation, the probable total number of cancer deaths depends only on the total amount of plutonium inhaled by the exposed populace.

If the population density per unit area within the wedge is $P(r, \theta)$, then the total amount of plutonium inhaled by the population will be:

$$\begin{aligned} M &= \int_0^\infty \mathrm{d}r \int_0^\theta \mathrm{d}\theta' rm(r)P(r,\theta') \\ &= \frac{Q_0 b}{Hu\theta}\int_0^\infty \mathrm{d}r\int_0^\theta \mathrm{d}\theta' P(r,\theta')\mathrm{e}^{-r/L}. \end{aligned} \tag{6}$$

For cases where the population density can be taken to be uniform with $P(r, \theta) = P_0$, this reduces to:

$$M_u=\frac{bQ_0P_0L}{Hu}=\frac{bP_0Q_0}{v}, \tag{7}$$

where v is the deposition velocity of the aerosol. An example of such a case would be if the explosion takes place within or upwind of a large densely-populated city, large enough for most of the aerosol to be deposited. This urban accident scenario would be the worst case short of a nuclear explosion.

A more realistic example may be one where a middle-sized city of width a happens to lie downwind within the wedge angle at a distance R from some cantonment or airfield where the explosion occurs (see Figure 2). For simplicity we will assume that the city is an annular piece at distance R from the explosion and subtending an angle $\theta_c = a/R$. Within the city we can approximate the population density as uniformly equal to P_0. For such a situation one goes back to general formula, eq. (6). Then we have, for the plutonium breathed in by that city's populace during plume passage, the result:

$$\begin{aligned} M &= \frac{Q_0 b}{Hu\theta}\int_0^\infty \mathrm{d}r\int_0^\theta \mathrm{d}\theta' P(r,\theta')\mathrm{e}^{(-r/L)} \\ &= \frac{Q_0 b}{Hu\theta}P_0\theta_c\int_R^{R+a}\mathrm{d}r\mathrm{e}^{(-r/L)} \\ &= M_u\frac{\theta_c}{\theta}[\mathrm{e}^{-R/L}-\mathrm{e}^{-(a+R)/L}], \end{aligned} \tag{8}$$

where M_u is the urban accident case result described earlier. In addition, there would be the plutonium breathed in by the people in the surrounding countryside.

Re-suspension: The formulae derived above apply to the plutonium inhaled from the original contaminated plume from the explosion as it moves downwind. As we shall see, even if the explosion takes place in a densely-populated area, the amount inhaled during plume passage is only about 0.1% of the total respirable plutonium aerosol content. The rest is deposited on the ground. But, as time passes, the plutonium that has been deposited on the ground could be re-suspended into the air through a variety of mechanisms like winds, clearing of debris, people walking, traffic, sweeping, eventual digging or ploughing of the land and so on. Unless the populace moves out of the entire contaminated area for a very long period of time or the area is decontaminated, they will continue to breathe in plutonium. Grazing animals, inhaling near soil level, might be particularly prone to such uptake of resuspended plutonium[16].

This phenomenon is usually parameterized by a re-suspension coefficient K, defined as the ratio of the plu-

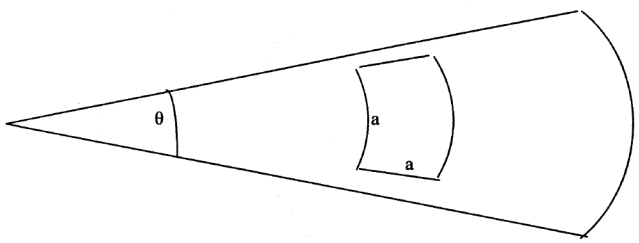

Figure 2. Schematic representation of a city of width a lying in the path of the plume at a distance R from an explosion.

tonium concentration in the air to the surface density of plutonium on the ground[17]. K is a function of time and the height above the ground that the concentration in air is measured. The literature lists many different functional forms to model the resuspension coefficient[18]. The leading term at some fixed height in most of these has an exponential form: $K(t) = K_0\exp(-st)$.

Integrating this over time gives M_r, the amount of resuspended plutonium that is inhaled:

$$M_r = Mv\left[\left(\frac{1}{s}\right)K_0(1-\exp(-st))\right],$$

where M is the plume passage inhalation amount derived earlier in eq. (6), v is the deposition velocity and s is an empirically determined parameter. Long-term evacuation or effective decontamination of the entire area for a large, densely-populated city would be very difficult. In that case, the population would be exposed to this resuspended plutonium, for a period t of several decades. The parameter s is of the order 1 (year)$^{-1}$. Then to a very good approximation the term $\exp(-st)$ can be neglected and the above expression reduces to

$$M_r = \frac{MvK_0}{s}. \tag{9}$$

The total amount of plutonium inhaled during plume passage and subsequent re-suspension is:

$$M_{\text{tot}} = M\left(1+\frac{vK_0}{s}\right) \tag{10}$$

We will later apply these formulae to estimate the amount of plutonium inhaled by the population and the consequent cancer fatality toll in hypothetical category 3 explosions in the South Asian subcontinent. The numerical values of some of the parameters that enter into the formulae are known more precisely than others. The breathing rate b is known fairly precisely as a function of age and of activity level. The wind velocity can vary a lot, but its value at a given place and time may be available. The total amount of plutonium will depend on the weapon and is, in principle, known very precisely, but less certain is the fraction that is converted to a respirable PuO_2 aerosol. A figure of 20% has been suggested as a reasonable working number; however, experiments have yielded figures ranging from 10 to nearly 100% (see Sutcliffe *et al.*[12] and Fetter and von Hippel[13]). Some of the other parameters which can also vary considerably, fall in the following ranges (see ref. 13).

Mixing layer height H, 300 to 2500 m; wedge angle θ, 0.1 to 0.3 radians; and deposition velocity v, 0.001 to 0.1 m/s (see table VI, B-1 in ref. 17).

Following the American Physical Society study on reactor safety and Fetter and von Hippel[13], we choose representative values of these parameters. These will be specified when we apply these results to the South Asian example.

Notice, however, that the parameters that enter in eq. (7), which corresponds to the uniform population density case, are relatively well-defined. The main variation comes from the deposition velocity v, which may be as high as 0.1 m/s if it is raining.

Health impact of exposure to plutonium

The principal risk from exposure to plutonium is by inhalation. The typical plutonium used in nuclear weapons is mostly plutonium-239 with a 24,400 year half-life, but has small admixtures of higher isotopes such as plutonium-240 (half-life of 6580 years) and plutonium-241 (half-life of 13.2 years) coming from successive neutron captures. What is termed weapons-grade plutonium, for example, typically contains about 93.8% of Pu-239, 5.8% of Pu-240 and 0.35% of Pu-241 (ref. 19). Each milligram of plutonium-239 corresponds to an activity in Bequerels (Bq, number of disintegrations per second) of:

$$\text{Activity} = \frac{\text{Mass}}{\text{Atomic weight}} \times \frac{\text{Avagadro number} \times \ln(2)}{\text{Half-life}}$$

$$= 2.3\times10^6 \frac{\text{Bq}}{\text{mg}}.$$

Because of the much shorter half-lives of the higher isotopes, they would also contribute significantly to the activity. Including the contribution from the alpha decay of Pu-240 increases the specific activity to 2.65 Mbq/mg (ref. 20).

To calculate the radiation dose per Bq of inhaled plutonium, we adopt the model developed by the International Commission on Radiological Protection (ICRP), which describes the deposition of inhaled plutonium and its subsequent behaviour[21]. Based on this model, we calculate an effective dose coefficient (EDC, which gives the equivalent radiation dose coming from breathing in 1 Bq of the substance) for different isotopes of plutonium in different forms. The different forms of plutonium refer to their behaviour in terms of solubility. The ICRP categorizes slow absorption forms as Type S, medium absorption forms as Type M, and fast absorption forms as Type F. Oxides of plutonium formed at high temperatures, about 1000°C, as in the case of plutonium aerosolized in explosions, are assigned to Type S. The equivalent whole-body dose coefficients in humans of different ages are given in Table 1 (ref. 22).

Table 1. Equivalent whole-body doses for different isotopes and solubilities of inhaled plutonium and americium

Compound	Effective dose coefficient (Sv/Bq) at age		
	3 months	15 years	Adult (male)
Pu-239 (Type S)	4.3×10^{-5}	1.7×10^{-5}	1.6×10^{-5}
Pu-239 (Type M)	8.0×10^{-5}	4.7×10^{-5}	5.0×10^{-5}
Pu-239 (Type F)	2.1×10^{-4}	1.1×10^{-4}	1.2×10^{-4}
Pu-240 (Type S)	4.3×10^{-5}	1.7×10^{-5}	1.6×10^{-5}
Pu-241 (Type S)	2.2×10^{-7}	1.7×10^{-7}	1.7×10^{-7}
Am-241 (Type S)	4.6×10^{-5}	1.7×10^{-5}	1.6×10^{-5}

For completeness, we also include the dose coefficient of americium-241, which results from the radioactive decay of plutonium-241. A quality factor or relative biological effectiveness index (RBE) of 20 for alpha radiation and 1 for beta and gamma radiation is used[23].

At the relatively low levels of plutonium inhalation that most people would be exposed to following dispersal from an accident, the primary effects of exposure to plutonium are an increased probability of cancers of the lung, liver and bone (see *Health Risks of Radon and other Internally Deposited Alpha-emitters*, ref. 15). Lesser effects resulting from the inhalation of plutonium oxide include a reduction in the number of white blood cells, among the most radiosensitive cells in the body; chromosomal aberrations in blood lymphocytes; and in the case of rats, increased incidence of mammary tumours (see *Health Risks of Radon and others Internally Deposited Alpha-emitters*, ref. 15).

Human epidemiological studies of the effects of plutonium inhalation are limited. Hence, two approaches can be used to estimate risks. The first involves the use of estimated life-time risks obtained from laboratory animal experiments. Difficulties with this approach relate to the many differences between animals and humans, including differences in histological types of cancers, differences in confounding exposures (e.g. smoking), differences in spontaneous risks and differences in life span. The second approach involves expressing risks obtained from humans exposed to alpha radiation from radon decay products, for which data from epidemiological studies with miners are available, or to low-LET (ref. 24) X-ray and gamma radiation in terms of dose (or dose equivalent) to the lung or other relevant tissues. These risk estimates are then applied to the doses resulting from high-LET alpha radiation from transuranic elements. One difficulty with this approach is that there may be characteristics of specific exposures that are not fully reflected in a single dose estimate, but that may affect resulting health effect risks.

ICRP has suggested using a rate of 500 fatal cancers and 100 non-fatal cancers per 10^4 person-Sv (ref. 25). To obtain the number of cancer deaths per unit mass of inhaled plutonium (taken to be of Type S), we use the age dependence of the coefficients in Table 1 to first compute an age-weighted EDC.

The fraction of the population under the age of 15 in India is 39.3% (ref. 26). The data for Pakistan are similar[27]. We will assume that this population is uniformly distributed over the age range of 0–15 years and we will linearly interpolate between the EDCs at ages 3 months and 15 years. This results in an average of 3.0×10^{-5} Sv/Bq for children under 15 years. Convoluting this with the population distribution of 39.3% below 15 years of age and using the adult EDC for the remaining 60.7%, we obtain an age-weighted EDC:

$$\text{Weighted EDC} = (0.393 \times 3.0 + 0.607 \times 1.6) \times 10^{-5} = 2.15 \times 10^{-5}\ \text{Sv/Bq}.$$

To convert this to a dose per unit mass of inhaled plutonium, we use the figure of 2.65 Mbq/mg obtained earlier to get:

$$\text{Dose per unit mass} = 2.15 \times 10^{-5}\ \text{Sv/Bq} \times 2.65 \times 10^{6}\ \text{Bq/mg} = 57\ \text{Sv/mg}.$$

Using the ICRP risk estimate, we obtain a figure of:

$$\text{Cancer deaths per unit mass} = 2.85\ \text{cancers/mg}. \quad (11)$$

There are other estimates of the number of cancer deaths per unit of radiation. The US National Research Council Committee on the Biological Effects of Ionizing Radiation (BEIR) proposes that specifically for plutonium, the risk estimates are 700 lung cancer deaths, 80–1100 bone cancer deaths and 300 liver cancer deaths per 10^4 person-Sv on the basis of human studies (see *High Risks of Radon and other Internally Deposited Alpha-emitters*, ref. 15). This would lead to an estimate of 6–12 cancer deaths/mg of inhaled plutonium. Similarly, Fetter and von Hippel[13] extrapolate from the risk of pulmonary neoplasia (a cancer) to beagle dogs subjected to high doses of plutonium and obtain 12 cancer deaths/mg of weapons-grade plutonium inhaled. Both these estimates are significantly higher than the figure suggested by the ICRP, which we use.

Estimated casualties in South Asia

We now apply the results of the wedge model and the health impact risk estimates to possible accidents involving nuclear weapons in South Asia, where the HE detonates and disperses plutonium (i.e. category 3). We look at two examples.

An urban case

Suppose a category 3 accident takes place at some air-force base or cantonment, which happens to be at the edge of a major city like Delhi, Karachi or Lahore. These cities will, within the next few years, have a population of about 10 to 12 million people in an area of about 600 to 800 km^2. Let us consider the worst-case scenario where such a city happens to be downwind at the time of the explosion and has an area large enough for all the plutonium to be deposited.

Note that using a mixing height $H = 300$ m a wind velocity $u = 1$ m/s and a deposition velocity $v = 0.01$ m/s in eq. (2) gives a contamination range L of about 30 km, which is about the width of our assumed cities[28].

For such cases one can use the uniform density formula eq. (7) with a population density P_0 of 15,000 people per km^2 or 1.5×10^{-2}/m^2. It is widely accepted that a single warhead contains a few kilograms of plutonium. Given the estimates cited earlier that about 20% of the plutonium is converted to respirable aerosol, we assume that $Q_0 \approx 1$ kg $= 10^6$ mg. Using the breathing parameters suggested by the ICRP, we take the age-weighted average breathing rate for a South Asian population to be $b = 3.3 \times 10^{-4}$ m^3/s (using data for breathing rates in ref. 21). We take the aerosol deposition velocity v to be 0.01 m/s. Inserting these values into eq. (7) gives the total amount of plutonium inhaled by the population:

$$M_u = \frac{bQ_0P_0}{v} = 495 \text{ mg}.$$

This number represents just the effect of plutonium inhalation during the passage of the plume. In addition, there will be further contamination from inhaling plutonium that is resuspended from what was initially deposited on the ground during plume passage. It is simply not feasible to decontaminate or evacuate a major South Asian city. What is likely is that there will be panic and flight, creating an unprecedented disaster in its own right. However, in the context of subcontinental urban poverty, most people are in fact likely to stay on or return within a short period of time to their original homes. Therefore, we have to multiply the number of deaths estimated above with the resuspension factor in eq. (10). We use the values $K_0 = 10^{-5}$ m^{-1}, $s = 1.96$ (ln 2) years^{-1} derived from the leading term in the resuspension formula suggested by the US Nuclear Regulatory Commission in 1990 (ref. 29). We also use $v = 0.01$ m/s to obtain a total inhalation amount of:

$$(M_{\text{tot}})_u = 495[1 + (10^{-5})(0.01)(3.15 \times 10^7)/(1.96)(\ln 2)] \approx 1640 \text{ mg}.$$

The final step is to get an estimate of cancer fatalities from such a dosage of inhaled plutonium. As discussed in the previous section, estimates range from 2.85 to 12 cancers/mg of inhaled plutonium. To be conservative, we use 3 cancer deaths/mg of plutonium inhaled, close to the lower limit of what is plausible. Therefore, the number of cancer deaths in this scenario is:

$$N = 1640 \text{ mg} \times 3 \text{ cancer deaths/mg} = 4920.$$

This has been a conservative estimate. The BEIR and Fetter and von Hippel estimates for fatal cancers/mg would suggest four times as many deaths. It must be remembered that these 5000 deaths (and possibly more) result not from a nuclear explosion with a fission yield, but only from the 'worst case' of a category 3 accident, i.e. the detonation of the chemical HE in the weapon, leading to plutonium dispersal.

A semi-urban case

The urban case estimate above hypothesizes the detonation of the HE in a nuclear weapon in the immediate vicinity of (or inside) a major metropolis like Delhi or Lahore. It may be argued that nuclear weapons are unlikely to be deployed on missiles or bombers so close to a major metropolis. Actually there *are* military bases and cantonments in the subcontinent right next to big cities. Nevertheless, let us also estimate the damage done in a scenario where a category 3 accident takes place in a military base at some distance from any city. We consider a case wherein there is a middle-sized city within 50 km downwind from the site of the accident. Jallandhar, Agra and the twin cities of Rawalpindi and Islamabad are examples of such cities.

For this situation schematically sketched in Figure 2, there will be plutonium absorption by both the people in that city and the people in the rest of the rural countryside. These two contributions will be additive. For the former we can apply the result in eq. (8) reproduced here for convenience:

$$M = M_u \frac{\theta_c}{\theta}[e^{-R/L} - e^{-(a+R)/L}].$$

When resuspension effects are included for the same reasons as in the worst case example, this amount gets enhanced by the same resuspension factor in eq. (10):

$$M_{\text{tot}} = M\left[1+\frac{vK_0}{a}\right] = (M_u)_{\text{tot}}\frac{\theta_c}{\theta}[e^{-R/L} - e^{-(a+R)/L}], \quad (12)$$

where $(M_u)_{\text{tot}}$ gives the mass of plutonium inhaled by a population of uniform density, including the effect of resuspension that was obtained earlier. For the medium-sized city that we are considering, assuming a population of 0.75 million people in an area of 10 km × 10 km, the population density will be half of the 15,000 people per km^2 that was used in the earlier scenario. Therefore, $(M_u)_{\text{tot}}$ will be halved to 820 mg.

If we take this middle-sized city to be at a distance of 50 km, it will subtend an angle $\theta_c = 10/50 = 0.2$ radians at the point of explosion. The wedge angle θ in the wedge model can take values between 0.1 and 0.3. Let us take the mean of these numbers and use $\theta = 0.2$. The width of the city is $r = 10$ km. Lastly the range L is given by Hu/v. Taking a wind velocity $u = 1$ m/s (≈ 2 miles per h), and typical values for the mixing height of $H = 500$ m, and the deposition velocity $v = 1$ cm/s, yields $L = 50$ km. This shows that it is not at all unreasonable that winds carry the plutonium aerosol to distances of the order of 50 km. Inserting these values into the above equation we get for the total plutonium breathed in by the population of that city:

$$M_{\text{tot}} = \frac{(M_u)_{\text{tot}}}{e}(1-e^{-0.2}) = \frac{820}{e}(1-e^{-0.2}) \approx 55 \text{ mg}.$$

To estimate the plutonium inhaled by the people in the countryside we can use the uniform density result obtained earlier in the 'worst case' context, but vastly scaled down by an assumed rural population density of about 200 people per km^2, instead of the 15,000 assumed in the metropolis. This gives for rural inhalation a value of $1640 \times 200/15000 = 22$ mg. Along with the urban contribution of 55 mg obtained earlier, this gives a total of 77 mg, which translates, using the same risk rate of 3 cancer deaths/mg into a total of about 230 fatalities. Although far less than in the earlier example, this number is comparable to major air-crash disasters. Again, using the BEIR and Fetter and von Hippel risk estimates for fatal cancers/mg would lead to about four times as many casualties.

In addition to the fatalities, there will be the medical costs of treating the fatal and non-fatal cancers resulting from inhalation of plutonium, as well as the clean-up costs for the immediate neighbourhood of the explosion where the contamination levels would be very high.

Discussion

We have described a tractable model of plutonium dispersal in the event of a nuclear weapon accident in which the HE in the weapon detonates. This was followed by a quantitative discussion of the health hazard posed by inhaled plutonium in terms of potential cancer deaths. We then applied these results to hypothetical accidents of this type in India or Pakistan to get an estimate of nearly 5000 cancer fatalities caused in a worst case scenario of this type. To this human cost has to be added the massive financial cost of even limited decontamination of just the immediate neighbourhood of the accident, which could be at least hundreds of crores of rupees (see Chenin and Murfin[12]). This would clearly be a major catastrophe.

Should such a category 3 plutonium dispersal accident take place in South Asia, we believe our estimates of damage are quite conservative. We are well aware that the phenomenon of plutonium dispersal and its health hazards are exceedingly complex and that there is considerable variation in the models and parameters used to characterize its different facets. Nevertheless, given the gravity of the problem, one cannot shirk from obtaining at least an order of magnitude estimate of the overall damage. To be useful, this should be done in a manner that is transparent, in both its assumptions and its methodology. This permits any scientific disagreement about the conclusions to focus on individual contributory factors.

In this spirit, we have used the wedge model since it captures the essential physics of plutonium dispersal in simple analytical fashion. It is easy to apply to different situations and the dependence of its results on different contributing factors is easy to pinpoint. While the simplicity of the model means that it cannot reliably give detailed distributions of plutonium contamination, especially in the high-dosage region near the accident site, it is known to give results in agreement with those from more detailed models and numerical codes at larger distances. In the no-threshold linear model of radiation-induced cancer risk, which is generally accepted, it is the low-dosage contamination over large areas that contributes most to cancer fatalities. If, however, there turns out to be a threshold radiation dose below which radiation-induced cancer risk vanishes, then the number of fatalities would be reduced. But, in the absence of concrete evidence for a threshold, caution demands that, like BEIR and ICRP, public health risk estimates be made using a no-threshold model.

The wedge model offers a good technique for making an order of magnitude estimate of the total number of cancer fatalities from such accidents. Even within the wedge model some of the input parameters like the aerosol deposition speed v, the mixing layer height H and the wedge angle θ can have widely varying values. The biological factors that determine the cancer risks of plutonium inhalation also vary in the literature, depending on the basic data used to arrive at them. We have taken care to point out in this article the range of likely

values of all these different parameters, and used conservative values. Admittedly, if in a given incident all the parameters conspire to take extreme values, they can upset even order of magnitude estimates. Notice, however, that the crucial formula (eq. (7)) for mass of inhaled plutonium in the uniform population density case, which we have used for our worst case estimate, is independent of most of the uncertain parameters.

It may be argued that this worst case estimate of 5000 (or perhaps as many as four times more) predicted cancer deaths could take a few decades to occur and that it will form only a small fraction of all cancer fatalities during that period due to other causes. There is limited reliable data on cancer mortality in South Asia[30]. One estimate in 1985 reported a crude cancer mortality rate of about 54 per 100,000 in South Asia[31]. Total cancer incidence in India for the year 1990 was estimated to be 612,300, while cancer mortality was estimated to be 4,33,000 (ref. 32).

But 5000 deaths are still 5000 deaths. That they happen quietly over decades among a largely unsuspecting public does not mitigate the tragedy. If it can be avoided, it must be.

Finally let us reiterate that our estimate of casualties is not for a nuclear explosion, but only for plutonium dispersal and inhalation from the detonation of the HE in a weapon. The possibility of detonation of the HE triggering in turn the detonation of the nuclear weapon cannot be ruled out: the 4th category in our list of possible accidents. Should such an accident take place, the nuclear yield could be as large as the design yield of the nuclear bomb or warhead. If this were to happen in the vicinity of a large South Asian city, several hundreds of thousands of people would die[33]. In addition, such an explosion, especially in times of crises, might be assumed to be a nuclear attack and lead to a nuclear response. Thus an accidental nuclear explosion may even initiate a nuclear war.

In conclusion, even if such a catastrophic accidental nuclear explosion does not occur, we have seen that just the dispersal of plutonium due to a HE detonation could lead to several thousand fatalities. Thus, prudence, if nothing else, dictates that India and Pakistan do not deploy nuclear weapons. They should also store them far away from missiles and aircrafts carrying potentially explosive fuel. A further level of safety may be gained by keeping the weapons disassembled, so that the HE is not close to the fissile material pit. All these steps would not only reduce the danger of accidental explosions, but also reduce the risk of a nuclear weapon being launched through error, panic or miscalculation.

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5. Gregory, Shaun, *The Hidden Cost of Deterrence: Nuclear Weapons Accidents*, Brassey's, London, 1990, pp. 184–190.
6. According to most reports, India's *Prithvi* missile's oxidizer is inhibited red fuming nitric acid (IRFNA) and the fuel is a 50 : 50 combination of xylidine and triethylamine; see, for example, Mama, Hormuz, *Int. Def. Rev.*, 1 August 1992, 784. When combined, this mixture is hypergolic, i.e. self-igniting and highly volatile. It has been suggested that Pakistan's *Ghauri* is based on the North Korean *No-Dong* missile and fuelled by unsymmetrical dimethylhydrazine (UDMH) with inhibited red fuming nitric acid as an oxidizer, see Wright, David, *Sci. Global Security*, 1998, **7**, 227–236; Bermudez, Joseph, S., Monterey Institute of International Studies, Center for Non-proliferation Studies, Occasional Paper No. 2, 1999; Wright, David and Kadyshev, Timur, *Sci. Global Security*, 1994, **4**, 129–160. However, it has also been suggested that *Ghauri* may use RP1 (kerosene) as fuel; see Chandrashekar, S., *Curr. Sci.*, 1999, **76**, 280–285.
7. *The Defence Monitor*, Center for Defense Information, Washington DC, 1981, **10**.
8. In case of accidents that disperse fissile material, plutonium-based weapons have significantly more severe health effects when compared to those using highly enriched uranium, and we concentrate on these.
9. The International Commission on Radiological Protection (ICRP) estimates that only 0.05% of ingested plutonium is absorbed by the gastrointestinal system. ICRP, *Age-dependent Doses to Members of the Public from Intake of Radionuclides: Part 2 Ingestion Dose Coefficients*, ICRP Publication 67, Pergamon, New York, 1994, p. 127.
10. Stephens, D. R., Lawrence Livermore National Laboratory Report UCRL-1D-119303 (undated); Haschke, John, M., Los Alamos National Laboratory Report LA-1231, 1992.
11. In the case of Palomares, 2.26 km^2 was contaminated with high plutonium ground concentrations, in excess of 11.8 kBq/m^2. Iranzo, E., Salvador, S. and Iranzo, C. E., *Health Phys.*, 1987, **52**, 453–461.
12. Sutcliffe, W. G., Condit, R. H., Mansfield, W. G., Meyers, D. S., Layton, D. W. and Murphy, P. W., Lawrence Livermore National Laboratory Report UCRL-JC-118825, 1995; Stephens, D. R., ref. 10. However, in one experiment at the Nevada test site, it was found that 99% of aerosol plutonium particles produced by the HE detonation of a nuclear explosive test device, found within a 16 km (10 miles) radius of ground zero, had physical diameter of less than 2.5 microns, indicating that essentially all of the fallout particles were in the respirable size range. Dick, J. L. and Baker, T. P., Report WT-1512, Air Force Special Weapons Command, Kirtland AFB, NM, 1967, cited in Chanin, David and Murfin, Walter, Sandia National Laboratory Report, SAND96-0957, May 1996 (available on the internet at http://plutonium-erl.actx.edu/restoration.htm.)
13. Fetter, Steve and von Hippel, Frank, *Sci. Global Security*, 1990, **2**, 21–42.
14. *Rev. Mod. Phys.*, 1975, **47**, supplement no. 1.
15. A linear model requires that there be no minimum threshold dosage for health risks to exist. It is believed that for non-stochastic phenomena like cancers and other genetic effects, there is no threshold. See *Health Risks of Radon and Other Internally Deposited Alpha-Emitters (BEIR IV)*, National Academy Press, Washington DC, 1988, p. 177. The ICRP also asserts that 'there are no adequate grounds for assuming a real threshold' and uses a simple proportional relationship at low doses. ICRP, *1990 Recommendations of the International Committee on Ra-*

diological Protection ICRP Publication 60, Pergamon Press, New York, 1991, p. 18. Public health criteria in the US and many other countries use such a no-threshold linear model. There is an ongoing controversy about the validity of the linear no-threshold model to arbitrarily low doses of radiation. See, for example, articles and letters in *Phys. Today*, September 1999, April and May 2000. However, there is significantly less disagreement in the case of heavily ionizing radiation such as alpha particles emitted by plutonium decay than for lightly ionizing radiation such as gamma or beta radiation.

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20. Even though Pu-241 has a much shorter half-life, it is a beta emitter. As Table 1 shows, the radiation dose per unit activity of Pu-241 is much smaller and we will ignore that contribution. However, Pu-241 decays into Am-241, which *is* an alpha emitter and that would contribute about 10% of the dose after one half-life of Pu-241, namely 14.4 years, from the time of manufacture of the weapon plutonium core. To be conservative, we restrict our analysis to weapons that have not aged much.
21. We use the most recent ICRP publication available that deals with plutonium; ICRP, *Age-dependent Doses to Members of the Public from Intake of Radionuclides: Part 4 Inhalation Dose Coefficients*, ICRP Publication 71, Pergamon, New York, 1996.
22. Due to the greater solubility, plutonium of Types M and F is cleared faster from the lung and accumulates in the bone surfaces and the liver. Hence, even though the radiation dose to the lung from these is much smaller than for Type S, the radiation dose to the whole body is much greater.
23. See ref. 15, ICRP publication 60, RBE is defined as the ratio of doses from two different radiations that produce equal levels of biological damage in the same biological system, i.e. RBE = dose of reference radiation/dose of test radiation for same level of effect. Reference radiation is usually 250 kV X-rays or Cs-137 or Co-60 gamma rays.
24. The rate at which ionizing particles impart energy locally to a medium is known as the linear energy transfer, commonly abbreviated as LET. The damage to living tissue by absorption of a given amount of energy is generally greater as the distance over which this energy is imparted decreases, i.e. as the LET increases. Jacob Shapiro, *Radiation Protection: A Guide to Scientists and Physicians*, Harvard University Press, Cambridge, USA, 1990, p. 34.
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