Hot Particles in the Environment: Assessment of Dose and Health Detriment

Werner Burkart and Hanspeter Linder

Biologie & Umwelt, Abt. SU, EIR, CH-5303 Würenlingen

Introduction

In weapon production and also during catastrophic events such as Chernobyl, mechanically produced particles containing a core of highly insoluble radioactivity may become airborne and pose an inhalation hazard. The analysis of the fallout from Chernobyl in Switzerland, more than 1300 km away from the source, showed the presence of such hot particles with activities of up to 3 kBq ^{103}ruthenium or up to 0.3 Bq ^{242}curium in single particles. By autoradiography, by α- and γ-spectrometry, both fission product and transuranium hot particles were found to be deposited in Switzerland. The α-spectrograph in *Figure 1* shows the presence of actinides found in nuclear fuel with a high burn up typical for commercial power reactors. Although identification and determination of size of the hot particles in the scanning electron microscope was not achieved so far, the means of transport exclude a diameter above few micrometers.

A theoretical dose calculation using ICRP models [1] yields quite small values for one single particle deposited in the lung. However, it is evident that, given the limited range of α- and β-radiation, a dose value averaged over the whole organ will give a poor description of the real situation arising from the highly concentrated radiation around the particle. Insoluble radioactivity deposited in the tracheobronchial region will be removed by the mucociliary ladder to the gastrointestinal tract where the speciation would prevent a potentially hazardous absorption. Deposition of locally irradiating hot particles in the pulmonary region with its slow clearance mechanisms will result in extremely high dose rates to only few hundred cells.

Already in the seventies, the extreme spatial inhomogeneity of the dose after the inhalation of insoluble particle with short-ranged particle radiation gave rise to hypotheses about the deadly radiotoxicity of hot particles [2]. However, these specific claims were never substantiated and clearly refuted with experimental evidence by *Albert* [3]. A review taking into account theoretical considerations [4, 5], results from animal experiments [6–9] and from epidemiological studies on former Manhattan Project workers [10] yields a very complex picture. A realistic model of the biological effects of radiation in the vicinity of hot particles is not available because of the complex dynamics of the afflicted tissue in response to the non-stochastic microlesions. However, the effects can be compared with the health detriments from the same amount of radioactivity distributed homogeneously in the lung [11].

Hot Particles from the Chernobyl Accident

A very conservative upper estimate of the hot particle concentration in the Chernobyl fallout in Western Europe is given by the total activity of insoluble radionuclides. The ^{103}ruthenium concentration on the Swiss Plateau was about 2 to 4 kBq/m^2. Since wet deposition was the major contributor, air concentration was only 2–5 Bq/m^3 for about two days. Time-integrated air concentrations were quite constant all over Switzerland. *Table 1* shows the maximal particle concentrations resulting from two different assumptions on particle size, i.e. particle activity, for Northern Switzerland and the more heavily contaminated Southern Tessin area.

Only activity deposited in the pulmonary region will lead to a long residence time and hence to considerable doses. Particles deposited in the tracheobronchial region of the lung will be cleared to the gastrointestinal tract by the mucociliary ladder. Assuming an initial dose rate of 100 Sv per hour in a distance of 50 μm (mucus layer thickness) from a 10 kBq ^{103}ruthenium particle, the total dose to the tissue along the clearance

Tab. 1. ^{103}Ruthenium particle concentrations in the soil, in the air during the fallout period, and in the lung assuming all ^{103}ruthenium activity in particles with the specified activity.

	Number of particles with activity of 10 kBq	100 Bq
Soil Swiss Plateau (1.85 kBq/m^2)	0.2/m^2	20/m^2
Soil Southern Tessin (18.5 kBq/m^2)	2/m^2	200/m^2
Air (5 Bq/m^3)	0.0005/m^3	0.05/m^3
Inhaled during fallout	0.02	2.3
Pulmonary deposition[a]	0.008	0.46

[a] Deposition probabilities for particle diameters of 2 and 0.43 μm are 0.3 and 0.2, respectively.

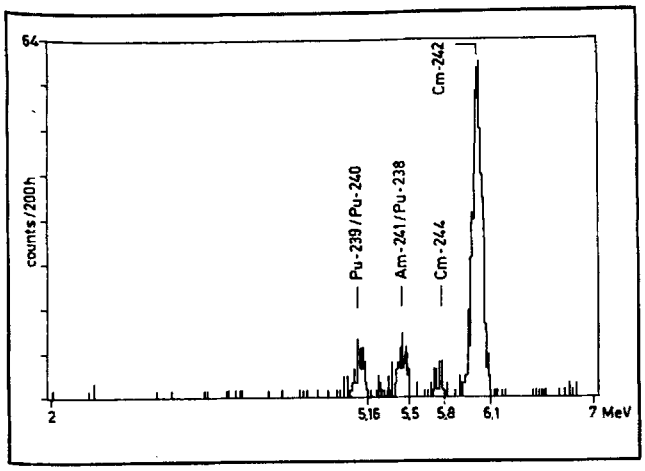


Fig. 1. α-Spectrometry of a hot particle collected on a plastic foil in Baar, Switzerland, in the wake of Chernobyl. The transuranic curium nuclides typical of spent reactor fuel are clearly present.

path is in the range of 1 Sv. Although this dose is considered too low to impair the ciliary movement in the tracheobronchial tree, poor clearance in smokers or in pathological states could theoretically lead to an immobilization of particles, for example at bifurcations. Then non-stochastic, acute effects could lead to a microlesion and possible long-term immobilization. The gastrointestinal transfer factor for monomeric ruthenium is very small. ICRP assumes a value of 0.05 [1]. For an insoluble ruthenium particle the value is practically zero. Given the continuous movement of the contents of the gastrointestinal tract and its protective coatings, the risk for local non-stochastic effects in the digestive tract is very small.

Conventional Dose Calculation

The annual limit of intake (ALI) of ICRP publication 30 [1] for insoluble inhaled ^{103}ruthenium activity amounts to 20 MBq. This implies that, over the next few months, the inhalation of 20 MBq ^{103}ruthenium will lead to an effective dose equivalent (H_{eff}) of 50 mSv, the annual exposure limit for occupational exposure. Even for finely dispersed or monomeric ruthenium, the exposure of the body will be quite inhomogeneous with most of the dose committed to the lung. Using the same ICRP model for particulate radioactivity in a worst case scenario with all the airborne ^{103}ruthenium activity in 10 kBq particles *(Table 1)*, only one in hundred persons was afflicted in Switzerland by the ruthenium fallout in air, receiving only an insignificant effective dose equivalent. A particle of 10 kBq ^{103}ruthenium deposited in the pulmonary region yields a "macroscopic" effective dose equivalent of:

(50 mSv/20 MBq) × 10 kBq × 3.3 = 82.5 μSv H_{eff}

50 mSv: occupational exposure limit
20 MBq: annual limit of intake for inhalation of ^{103}ruthenium; one ALI leads to an exposure of 50 mSv
3.3: inverse of probability of deposition in the pulmonary region

Therefore, in conventional dosimetry, the concentration of the fallout ruthenium radioactivity in few particles has no other effects than a highly skewed dose distribution in the population. ICRP dose limit recommendations for the general public of 1 mSv or 5 mSv are not surpassed by the hottest ^{103}ruthenium particle found in the wake of Chernobyl [12].

Microdosimetry of Hot Particles

Figure 2 shows the extremely high committed doses around single hot particles. The dashed line depicts the radial dose for a monoenergetic β-source of only 1 Bq activity and a β-energy equal to β_{max} of ^{103}ruthenium. Due to the shorter penetration depth of α-particles, the localized doses from α-hot particles are still higher and more confined than in the case of β-point sources. Dose assessments published in the wake of Chernobyl [13] are, however, misleading by up to two orders of magnitude for doses at larger distances from the particle because of the erroneous assumption that β-decay yields monoenergetic electrons of energy β_{max}. *Figure 2* gives both the published estimate [13] and a more realistic relationship which takes the lost neutrino energy into account. This distinction is quite important for a discussion of the biological relevance of hot par-

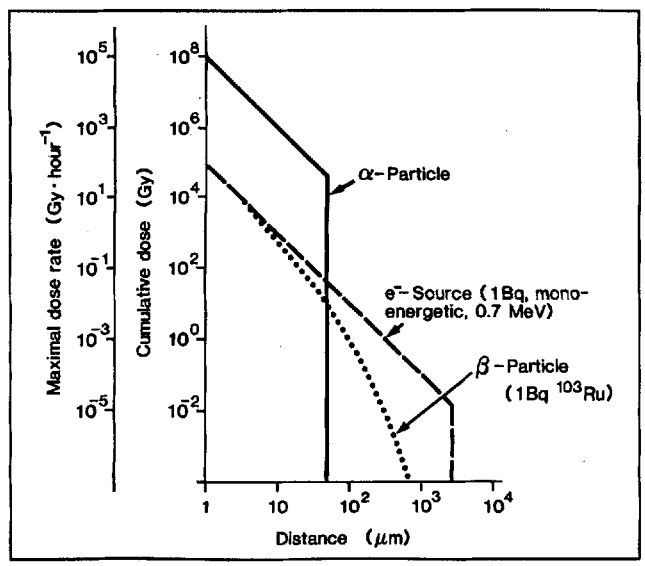


Fig. 2. Cumulative dose and maximal dose rate in the vicinity of an immobilized hot particle. The β-dose/distance-relationship is given both for a point source of 1 Bq ^{103}ruthenium and for a 1 Bq source of monoenergetic electrons with an energy of 0.7 MeV. The steeper decrease of the β-dose is caused by the fact that the average β-energy is only about one third of β_{max}.

ticles and the quantitative differences between α- and β-particles. The steeper dose decrease with distance for β-emitting fission products than for monoenergetic electrons removes some of the postulated differences in the dose distribution around α- and β-particles, respectively. This is especially relevant because this decrease takes place in the area irradiated with sublethal doses. The biological relevance of local megadoses from particulate α-emitters was discussed and studied in the seventies [2, 4, 6, 8]. Experimental findings from animal studies and theoretical considerations [5] 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 α-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 *(Fig. 2)*. Hence, almost all ionizations and excitations occur in dead tissue. Since the radicals formed during the passage of charged particles only travel very short distances in the range of nanometers, i.e. a small fraction of a cell diameter, transformation events would only arise in sublethally irradiated, proliferation competent cells. The wasting of a large fraction of the radiation dose on dead cells is thought to cause the relative innocuousness of α-hot particles.

For β-particles, the dose distribution looks quite different *(Fig. 2)*. Over a much larger activity range of the particle, considerable tissue areas receiving sublethal dose rates are expected.

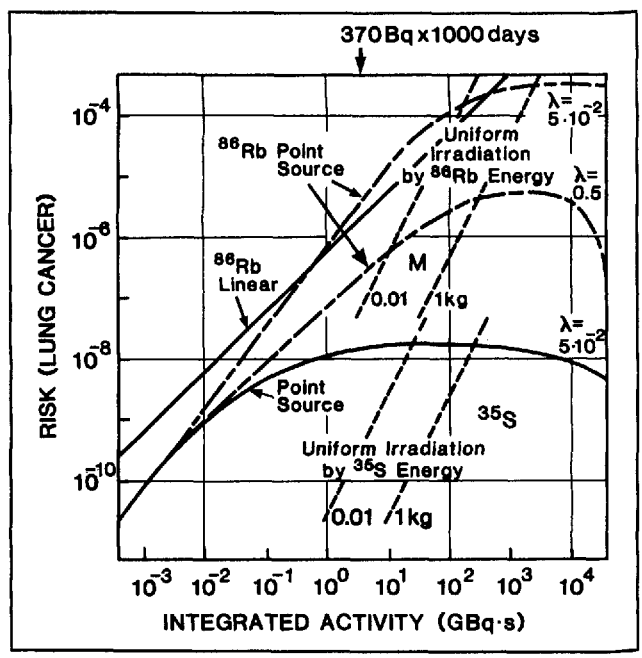
Besides the dose distribution, the distance of critical tissue areas and cell populations relative to the radiation source is crucial. Only the basal cells and mucus cell layer of the tracheobronchial epithelium and in the alveolar tissue are the critical cell mass for the late somatic effects, i.e. lung cancer, of ionizing radiation. Any energy deposition in a mucus layer or another extracellular matrix will have no biological effect at all, whereas irradiation of the differentiated postproliferative epithelium will only lead to non-stochastic effects. These acute effects are caused by gross cellular damage and show a clear threshold in the range of sieverts. Depending on the respiratory region, the contribution of mucus, extracellular structures, and of non-transformable cells to the tissue may vary considerably.

Dose, Dose Rate, and Theoretical Effects

With a half-life of 40 days for ^{103}ruthenium, the particle delivers initially about 0.1 % of the total dose per hour. Therefore dose rates are very high and may reach values up to 15 kSv/h for a 10 kBq particle. In this dose area, a non-stochastic microlesion arises. After the destruction of the cells in the direct vicinity, dose fractions delivered later on become biologically ineffective due to increasing deposition in non-viable structures.

Adjacent to the lethally irradiated zone, groups of cells will receive sublethal doses. For a cumulative dose in the range of 10 Gy, the dose rate drops to about 10 mGy per hour. Due to cellular repair processes such a rate will have only slight effects on survival and proliferation. For small organ doses, a quadratic dose/effect-model for late somatic effects [14] will predict largely increased biological effects for inhomogeneously distributed activity as compared to homogeneous doses. The theoretical difference may amount to several orders of magnitude. *Figures 3* and *4*, based on raw data from *Mayneord* and *Clarke* [5], show this drastic effect for β- and α-emitters, respectively. At an integrated activity of 10 kBq × sec ^{239}plutonium, a point source irradiation is 8 orders of magnitude more dangerous than uniform irradiation *(Fig. 4)*. It has to be noted, however, that the risk ratios between inhomogeneously and uniformly distributed doses are only high at a very low total risk. Above an absolute life-time risk of about 10^{-8} from ^{239}plutonium or from ^{35}sulfur deposited in the lung, the risk from uniformly distributed radioactivity becomes higher. The ratio change is caused by the overkill effect, the steep increase with dose of radiation wasted on dead tissue. For ^{86}rubidium with its high β_{max}-energy of 1.8 MeV, the tissue area affected around a point source is quite large. The β-range is about 6 mm in soft tissue as compared to less than 0.5 mm for ^{35}sulfur. Therefore cell-killing effects become dominant only at a much higher theoretical life-time cancer risk of 10^{-6} to 10^{-4} *(Fig. 3)*.

Fig. 3. Theoretical excess lung cancer risk from a single β-particle assuming a quadratic dose/effect-relationship with an exponential cell killing function $0.0025\ D^2 \times e^{-\lambda D}$ [20]. The dashed straight lines show the risk from the same activity uniformly distributed over a tissue mass of 10 g and 1 kg, respectively.



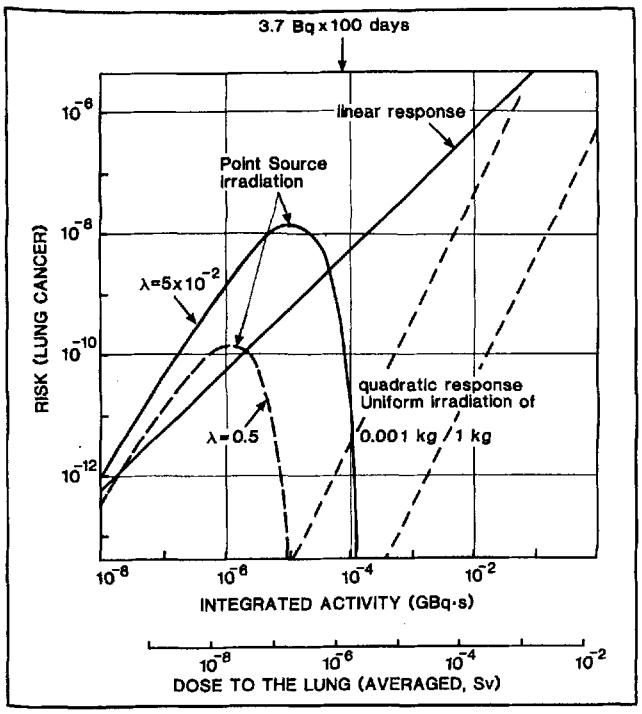


Fig. 4. Theoretical excess lung cancer risk from a single α-emitting ^{239}plutonium particle assuming a quadratic dose/effect-relationship with an exponential cell-killing function 0.0025 $D^2 \times e^{-\lambda D}$ [20]. The straight line gives the lung cancer risk for the linear function 0.0025 D. The straight dashed lines show the risk based on the quadratic risk function from the same activity uniformly distributed over a tissue mass of 1 g and 1 kg, respectively.

If a linear dose/effect-relationship for cancer induction is assumed, dose distribution has no effect on the risk from ionizing radiation. For comparison, the quadratic and linear risk functions in *Figure 3* and *4* are assumed to intersect at a lung dose of 1 Sv. The resulting lifetime risk to contract lung cancer from this dose is taken to be 0.0025. In the linear model non-uniform irradiation will be as effective as a corresponding uniform irradiation as long as the cell-killing effect does not contribute to the outcome. As soon as local doses are high enough to induce cell killing, the particulate radioactivity becomes less effective than the uniformly distributed activity. Since almost all radiation biologists prefer a linear model for α-irradiation and since health physicists use this model even for the estimate of risk from low-LET β-doses, the above theoretical considerations provide no logical argument to consider particulate radioactivity more dangerous than uniformly distributed radionuclides. In other words, the extreme risk ratios between the two modes of irradiation are a result of the extremely low risk from small doses in the quadratic dose/effect-model.

Because a dose/effect-model with risk based solely on dose and dose square functions cannot predict the outcome of dynamic biological processes induced by the microlesion in the vicinity of a hot particle, experimental studies are needed to assess the effect of particulate radioactivity. This holds especially for the complex proliferation and differentiation processes occurring in a sublethal radiation field around and in response to the lesion. Only experimental data from animal studies or human epidemiology can yield information about the possibility that higher tissue sensitivity around the lesion could overcompensate for the overkill effect.

Animal Experiments and Human Epidemiology

Several animal studies were conducted, notably by *Little* et al. [6], *Lafuma* et al. [15], and *Richmond* [4]. Dogs, rats, mice, and hamsters were used.

Richmond [16, 17] implanted large zirconium oxide particles containing ^{238}plutonium and ^{239}plutonium in an amount corresponding to pure plutonium oxide particles with diameters of 0.1 to 1 μm. At a concentration of about 2000 particles per hamster, none of 700 animals showed visible tumors upon disection. This is in marked contrast to the hypothesis of *Tamplin* and *Cochran* [2], which would predict a tumor rate of about 2 per animal.

Direct comparisons of the effects of inhomogeneous and uniform lung distribution were made by *Grossmann* et al. [18] and *Little* et al. [6]. ^{210}Polonium was injected into the lungs of two groups of hamsters in solution or bound to iron oxide particles, respectively. The average, macroscopic dose to the lung was about 200 Sv. Tumor incidence was higher by a statistically significant factor of 2.7 in the animals with uniform dose distribution. Non-uniform irradiation also lead to longer latency periods, i.e. increased the time between irradiation and the emergence of detectable tumors.

Lafuma et al. [8, 15] studied the effects of inhaled α-emitting transuranium elements in rats. Soluble ^{244}curium nitrate was used for soluble, uniformly distributed activity and particulate, insoluble ^{239}plutonium oxide as a model for hot particles. Both the life shortening effect and the tumor incidence was shown to be correlated with the activity distribution. Uniformly distributed activity being up to five times more toxic than particulate ^{239}plutonium oxide [8].

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 μm. 1972, more than 25 years after the contaminations, the average body burden still amounted to about 3.7 kBq ^{239}plutonium with about 5 to 10% residing in the lung. No health detriments traceable to these extremely hot particle exposures have been found so far [10].

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 [19]. 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.

Conclusions

Contrary to the impression at first glance, radiation from hot particles was never shown to be more 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 [20]. Therefore, the general conclusion stressed also in review papers by *Bair* et al. [7] and *Feinendegen* et al. [21] is that, with increasing inhomogeneity, the detrimental effects of ionizing radiation to the lung tend to decrease.

Although additional studies on β-hot particles are needed to improve our knowledge, the present state of comprehension, derived mainly from α-hot particles, is sufficient to refute most of the highly publicized hot particle theories. Given the small dose contribution from inhalation, it can be stated that at least in Western Europe, hot particles did not contribute significantly to the dose and the possible health detriment from the Chernobyl fallout.

Summary

Hot particles, highly radioactive particles made up of fission products or actinides and being small enough to become airborne, defy many of the dose models and risk concepts in use by the health physics community. At environmental exposures, both on the level of populations and tissues, dose distributions become very skewed; only few persons or tissue cells being exposed at correspondingly higher levels.

Fallout from Chernobyl showed a numerically small subfraction of the aerosol particles containing activities of up to 10 kBq ^{103}ruthenium or up to 0.3 Bq ^{242}curium in single particles. The implications of these hot particles on the dosimetry after uptake are great since the locally accumulated dose, for example in the pulmonary region, may reach up to 10 MSv.

For inhalation as well as for ingestion, theoretical considerations predict a decrease in the effectiveness of particulate radioactivity as compared to monomeric activity. This hypothesis is also supported by experimental and epidemiological evidence from the examination of the radiation effects of particulate α-emitters deposited in the lung.

Zusammenfassung

Heisse Teilchen in der Umwelt: Abschätzung der Dosis und des Lungenkrebsrisikos

Bei der Untersuchung der Speziation der Tschernobyl-Fallout-Aktivität mit Hilfe der Autoradiographie, der Alpha- und Gamma-Spektrometrie fanden sich einzelne Aerosolpartikel mit Aktivitäten von bis zu 3 kBq ^{103}Ruthenium bzw. bis 0,3 Bq ^{242}Curium. Die Exposition der Lunge durch solche unlösliche «hot particles» kann durch die etablierten Dosiskonzepte nicht realistisch modelliert werden, da die kumulierte Strahlenbelastung zwischen mehreren MSv in nächster Nähe des Teilchens und praktisch null für das restliche Gewebe schwankt. Die Abschätzung der gemittelten, makroskopischen Dosiserwartung für einen inhalierten ^{103}Ruthenium-Partikel von 3 kBq ergibt eine effektive Äquivalentdosis von 25 μSv. Die extreme inhomogene Exposition führt jedoch dazu, dass nur eine kleine Gewebefraktion betroffen ist und dass der hochbestrahlte mikroskopische Gewebebezirk in direkter Nachbarschaft zum Partikel abstirbt. Dadurch wird ein grosser Teil der Dosis in absterbenden Zellen, die nicht mehr transformierbar sind, deponiert und somit biologisch wirkungslos. Partikuläre Radioaktivität liegt deshalb in ihrer biologischen Wirksamkeit auch unter Berücksichtigung der Gewebsbereiche mit subletaler Bestrahlung mit grosser Wahrscheinlichkeit unter derjenigen von monomerer Aktivität. Auch tierexperimentelle und humanepidemiologische Befunde aus der Untersuchung der Strahlenwirkung von in der Lunge deponierten partikulären α-Strahlern stützen diese Annahme, die jedoch für β-Strahler der experimentellen Nachprüfung bedarf.

Résumé

Les particules chaudes dans l'environnement: jugement des doses et effets sur la santé

Des examens destinés à déterminer la forme physique des retombées radioactives de Tchernobyl ont montré que certaines particules contenaient jusqu'à 10 kBq d'activité de produits fissiles, essentiellement du ^{103}Ru, ou jusqu'à 0,3 Bq d'activité alpha avec une partie évidente de ^{242}curium. Etant donné que l'oxyde et l'hydroxyde de ruthénium sont difficilement solubles et, qu'après inhalation, de longs séjours dans les poumons ont été décelés, la question des effets de telles «particules chaudes» se trouve donc posée. L'extrême nonhomogénéité dans l'espace de la dose radioactive après inhalation de particules insolubles fortement actives, avec un rayonnement de courte portée (alpha ou béta), a conduit à des hypothèses relatives à une radiotoxicité très élevée des «hot particles». Un aperçu des réflexions théoriques sur les résultats émanant d'expériences animales, ainsi que d'études épidémiologiques faites sur des collaborateurs du projet Manhattan, présente un tableau très complexe. Bien qu'une modélisation des effets de radiations dans le domaine des «particules chaudes» ne soit guère possible à cause de la dynamique des tissus affectés, les répercussions peuvent être comparées en ce qui concerne les effets à une activité pulmonaire répartie de manière homogène. La faculté de transformation de l'activité des particules est, selon toute probabilité, plus petite que celle de la même activité bien réparti dans les poumons. Cela est induit par l'absorption d'une grande partie de la dose par des tissus qui s'atrophient.

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SIDA, santé publique et éthique: La problématique des barrières

Jean Martin

Service de la santé publique et Institut universitaire de médecine sociale et préventive, Cité-Devant 11, CH-1014 Lausanne

Pour qui suit l'histoire rapidement évolutive du SIDA, notamment dans les médias, il est clair que la question des mesures autoritaires qui pourraient être prises est soulevée fréquemment, de plus en plus fréquemment semble-t-il.

De milieux plus ou moins crédibles et plus ou moins orientés politiquement, on voit venir par exemple des suggestions de tests sérologiques obligatoires, pour toute la population ou des groupes en son sein, et de mesures d'ostracisme vis-à-vis des séropositifs et malades. Par exemple leur exclusion de certains endroits (crèches, écoles, autres groupes organisés) et de certaines activités professionnelles. Sans compter ceux qui proposent que les personnes infectées soient identifiables de manière définitive (tatouage!).

Une réflexion sereine quant aux modalités d'application pratique de telles propositions dans notre société montre qu'elles sont irréalisables. Qui plus est, alors même que le gain de santé, les bénéfices de santé publique à attendre seraient nuls ou quasi nuls, les effets secondaires négatifs, eux, seraient graves: des discriminations et surtout le fait de pousser dans la clandestinité les personnes qui se sentiraient visées. D'où des difficultés très accrues de les atteindre pour les intervenants des secteurs sanitaire et social qui s'attachent à trouver des manières efficaces de les aider et d'implanter de véritables comportements de prévention dans la population.

En tant que médecin de santé publique, notre rôle est d'étudier tout ce qui peut apporter une contribution utile à la lutte contre l'extension du SIDA. L'observation de la situation actuelle nous amène à la constatation que, trop souvent, on se trompe de «barrières». Qu'on a tendance à envisager celles qui sont inutiles et «contre-productives», peut-être parce qu'elles paraissent plus simples et plus fermes. Et qu'on ne met pas suffisamment l'accent sur d'autres, qui peuvent, elles, être efficaces mais dont l'application demande un engagement personnel et souvent du courage.

Ce qui est inutile

C'est le cas des propositions tendant à élever des barrières pour isoler certains groupes (que ce soit les homosexuels, les toxicomanes, les prostituées, les immigrants, les réfugiés, etc.). Avec l'idée de les «empêcher de nuire» comme on le faisait dans d'autres