The Incorporation of Radionuclides After Wounding by a “Dirty Bomb”: The Impact of Time for Decorporation Efficacy and a Model for Cases of Disseminated Fragmentation Wounds

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Objective: In the case of a terrorist attack by a “dirty bomb” there is a risk of internal contamination with radionuclides through inhalation and wounds. We studied the efficacy of a decorporation treatment depending on the initiation time and duration.

Approach: Based on biokinetic models, we simulated the impact of different diethylenetriaminepentaacetic acid treatments on the committed effective dose after the incorporation of plutonium-239.

Results: For the same level of radioactivity, the dose was higher after the fast absorption from the wound than after a slow invasion following inhalation. The impact of the treatment initiation time was particularly important in the case of the internal contamination through the wound. Ending the treatment at an early point in time was followed by an augmentation of radioactivity in the blood compartment, reflecting insufficient treatment duration. Treatment efficacy increased only marginally if extended over 90 days.

Innovation and Conclusion: For plutonium-239, the committed effective dose and the impact of the treatment initiation time on therapeutic efficacy predominantly depend on the speed of invasion, *i.e.*, the pathway and the physicochemical properties of the compounds involved. Thus, it is prudent to start decorporation therapy as soon as possible, as a loss of efficacy resulting from a delay in treatment initiation cannot be compensated later on. In the case of plutonium-239 incorporation, the treatment must be continued for several months. Multiple fragmentation wounds might be aggregated to a single wound model suited for internal dosimetry calculations by using the “rule of nine.”

Keywords: dirty bomb, radionuclide incorporation, plutonium-239, DTPA, decorporation treatment

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INTRODUCTION

IN THE CASE of a terrorist attack with a radiological dispersal device (RDD or “dirty bomb”), patients with a combination of conventional mechanical trauma, external radiation exposure, and external and internal

contamination must be expected. Explosion-related injuries are usually classified into five categories (Table 1).¹ Secondary and tertiary injuries are expected to be predominant, as explosive devices are often built to cause primarily damage from fragments

Table 1. Blast injury categories

Effect	Impact	Typical Injuries
Primary	Direct blast effects (pressurization)	Tympanic membrane rupture Blast lung
Secondary	Propelled projectiles (e.g., fragments of the bomb or casing)	Penetrating injuries Multiple fragmentation wounds Concussion
Tertiary	Propulsion of the body of the victim Burying after the collapse of infrastructure	Blunt trauma Crush syndrome
Quaternary	Heat or combustion fumes	Burns Inhalation injury
Quinary	Radioactive, toxic, or infectious additives	Depending on the agent

Adapted from NAEMT.¹

resulting from the breakup of the casing or embedded objects.¹ According to the principle “treat first what kills first,” life-threatening mechanical injuries must be managed as the top priority.^{1,2} After stabilization of the vital functions, the consequences of external irradiation and incorporation must be assessed. Data from past radiological incidents indicate that, except special cases (Litvinenko),³ the incorporation of radionuclides alone is usually not expected to induce acute radiation sickness.⁴ Internal irradiation of target organs may, however, cause stochastic health effects.⁵

Thus, one treatment goal should be to enhance the elimination of radionuclide(s) incorporated. Like for other metal intoxications, this may be achieved by chelating agents that exchange a less firmly bound ion (e.g., calcium) for the toxic metal ion.⁶ The principal agent to remove radioactive metals (transuranic metals, rare earths) is diethylenetriaminepentaacetic acid (DTPA).⁷ The radionuclides are bound by DTPA (e.g., Ca-DTPA exchanges the calcium ions for plutonium ions) to form a chemically stable complex that is water soluble and readily excreted in the urine by glomerular filtration.^{8,9} By enhancing the excretion rate, the radionuclide body burden and the committed effective dose are reduced.

The efficacy of decorporation treatment after radionuclide incorporation is best when started early, even if the committed effective dose has not yet been determined (“urgent approach”).^{10–12} However, the impact of time on decorporation efficacy has been shown to depend on the specific radionuclide(s), the speed of the invasion kinetics, *i.e.*, the invasion pathway, and the physicochemical properties of the compounds involved (Rump *et al.*, unpublished observations, Bundeswehr Institute of Radiobiology, 2015). Thus, it is not possible to give a definitive time limit for valid treatment ini-

tiation in all cases. The particular scenario must be considered with all details.

In the case of a “dirty bomb” (RDD) explosion, the primary invasion pathways will probably be through inhalation and wounds. Therefore, understanding the impact of the time of treatment initiation and duration in such a scenario would be useful. Although we cannot predict the precise nature of terrorist attacks and the materials that might be used in an RDD, radionuclides widely used in industry, medicine, or research are of particular concern.^{13–15} Among them is plutonium-239 because of its availability and high toxicity.

The radiotoxicity of plutonium is far more relevant than the chemotoxic heavy metal effects.¹⁶ Plutonium-239 emits high-energy alpha-particles and the transfer of energy to the surrounding tissues is the dominant mechanism of toxicity. The low range of the alpha-radiation explains the coincidence of plutonium accumulation and toxic effects. Animal experiments as well as epidemiological studies on workers at plutonium production and/or processing facilities provide evidence for an association between cancer mortality, in particular lung, bone, and liver cancer.¹⁷ Following inhalation, plutonium-239 may also cause a radiation pneumonitis, observed as the primary cause of early death in animal experiments, depending on the activity inhaled. Non-neoplastic liver lesions (degeneration, necrosis) have also been described in experimental studies.¹⁸

CLINICAL PROBLEM ADDRESSED

In the present work, we studied the decorporation kinetics of plutonium-239 entering the body through the lungs or a wound as may be expected after a “dirty bomb” attack. We focused on the impact of the initiation time and the duration of a DTPA decorporation therapy as there is a lack of concrete data on this topic. We assumed plutonium to be integrated in the bomb as a soluble compound, *e.g.*, plutonium (IV) nitrate,¹⁹ essentially used in the chemical processing of plutonium, and thus contaminating and entering the wound as such. We moreover assumed that plutonium was further oxidized to poorly soluble plutonium dioxide, due to the heat developed during the explosion, before contaminating the atmosphere and being inhaled. As in the case of a “dirty bomb” attack, victims involved in the explosion would be expected to be hit by a large number of fragments of different sizes, we further developed a proposal for a simple method that may be used by clinicians, how to aggregate disseminated multiple fragmentation injuries into a single wound model suited for internal dosimetry calculations.

MATERIALS AND METHODS

Biokinetic models and simulations

The computations are based on the National Council on Radiation Protection and Measurement (NCRP) wound model^{20,21} and models developed by the International Commission on Radiological Protection (ICRP)^{22,23} and by Legget *et al.*^{24–26} The models were further adapted by Bailey *et al.*²⁷ and Davesne *et al.*²⁸ (respiratory tract model), as well as by Stricklin *et al.*²⁹ and Konzen and Brey,³⁰ to study decorporation biokinetics.

A simplified wound model used in this work included two basic compartments: one for a soluble state and another that represents both colloid and intermediate states. In the model, the portion of the fragment that is soluble can transfer directly from the wound site into the blood compartment of the systemic ICRP model. However, soluble particles from the wound may also form colloids, which undergo intermediated states before fully dissolving and entering the blood compartment. Konzen and Brey³⁰ provide rates for the transition of these compartments, which feed into the ICRP biokinetic model. Other compartments described by the NCRP model were not included since rates for their transitions were not readily available at the time of our work. The transfer of plutonium occurred directly from the soluble state compartment. The rate constants of the wound model were as follows: from the soluble state into the blood 0.241 day^{-1} ; from the soluble state into the colloid state 0.094 day^{-1} and back into the soluble state 0.026 day^{-1} .

The committed effective dose was determined for a period of 50 years. The dose reduction factor was calculated by dividing the committed effective dose absorbed under treatment by the dose without treatment. The efficacy of the treatment was estimated by subtracting the dose reduction factor from one (efficacy = $1 - [\text{dose with treatment}]/[\text{dose without treatment}]$).

Scenarios

To assess the impact of the treatment initiation time, the simulated DTPA treatments were started at different times ranging from 1 h to 180 days after acute inhalation of a poorly soluble plutonium-239 compound or injection of a soluble compound into a wound. In both cases, the incorporated activity was set at $1 \mu\text{Ci}$ and the duration of the treatment at 180 days. To study the impact of treatment duration on efficacy, the treatment start time was set at 1 h after incorporation and treatment duration was varied from 14 to 180 days. The time course of the total radioactivity in the blood was studied in more

Figure 1. Time course of radioactivity in different organs and tissues after the injection of $1 \mu\text{Ci}$ of plutonium-239 as a soluble compound into a wound. Simulations are based on a simplified wound model linked to the ICRP systemic model of plutonium, further adapted to study decorporation biokinetics by Stricklin *et al.*²⁹ ICRP, International Commission on Radiological Protection.

detail depending on the initiation time of the treatment and its duration.

RESULTS

The committed effective dose after the injection of a soluble plutonium-239 compound into a wound was calculated to be 823 mSv, whereas inhalation of the same activity of a poorly soluble plutonium-239 compound resulted in a dose of only 260 mSv.

The maximum activity in the blood is reached at the end of the 2nd day following incorporation through the wound (1 day and 22 h) and amounts to $0.165 \mu\text{Ci}$ (Fig. 1). Plutonium-239 accumulates principally in the liver and to a lesser extent in the bone. The biokinetic models estimate that activities in these tissues reach a plateau around the 10th day. At the same time, the remaining activity in the wound asymptotically approaches the abscissa (Fig. 1).

If starting a DTPA treatment early, *i.e.*, within 12 h, the absorbed dose could be reduced by more than 97% after incorporation through inhalation as well as through the wound. In the further time course, therapeutic efficacy decreased more rapidly in the case of incorporation through the wound up to 90 days, when decorporation efficacy became quite similar for both invasion pathways (Table 2). The efficacy of treatment increases with its duration (Table 3). For treatment durations shorter than 90 days, decorporation appears to be more efficacious in the case of a soluble plutonium-239 injection into a wound than after the inhalation of a poorly soluble compound.

Table 2. Effect of decorporation treatment with diethylenetriaminepentaacetic acid after inhalation of a poorly soluble plutonium-239 compound or injection into a wound of a soluble compound ($1\mu\text{Ci}$) depending on the treatment initiation time

Treatment Start Time										
	None	1 h	2 h	6 h	12 h	1 Day	2 Days	10 Days	30 Days	180 Days
Committed effective dose (mSv)										
Inhalation	260	4.7	4.7	5.0	5.8	8.3	15	70	150	214
Wound	823	9.7	10	14	24	57	143	501	581	663
Therapeutic efficacy										
Inhalation	0	0.98	0.98	0.98	0.98	0.97	0.94	0.73	0.42	0.18
Wound	0	0.99	0.99	0.98	0.97	0.93	0.83	0.39	0.29	0.19

A treatment duration of 180 days is assumed. The calculations of the committed effective dose for 50 years are based on the wound model of Konzen and Brey³⁰ and the ICRP systemic model for plutonium.^{22,23} For each treatment start time, the dose reduction factor was calculated by dividing the committed effective dose absorbed under treatment by the dose without treatment and the efficacy was estimated by subtracting the dose reduction factor from one.

ICRP, International Commission on Radiological Protection.

The impact of the treatment start time on therapeutic efficacy after plutonium incorporation through a wound is also reflected by the time course of radioactivity in the blood compartment depending on the time treatment is initiated (treatment duration of 14 days is assumed) (Figs. 2 and 3). If starting the DTPA treatment together with the plutonium injection, a first lower maximum activity in the blood compartment is reached immediately after injection and amounts to $0.00240\mu\text{Ci}$, and a second peak in activity follows the treatment termination and amounts to $0.0062\mu\text{Ci}$ on the 17th day (Table 4). If the initiation of treatment is delayed to the 7th day after incorporation or later, the maximum activity in the blood is the same as without treatment (0.165 at 1 day and 22 h). At that time, activity is roughly 300 times higher than in the case in which treatment had been started immediately ($5.5 \times 10^{-4}\mu\text{Ci}$).

The time course of radioactivity in the blood compartment depending on the duration of the treatment is shown in Fig. 4. If DTPA treatment is started 1 h after plutonium-239 injection into a wound and continued for 14 or 21 days, the maximum activity in the blood compartment is reached just before treatment starts and amounts to $0.0117\mu\text{Ci}$. Discontinuation of the treatment leads

to an increase of activity in the blood, but the amounts reached stay below the value just before treatment starts. In the case treatment duration is only 7 days, the activity in the blood will increase after treatment has been stopped from a minimum of $5.3 \times 10^{-5}\mu\text{Ci}$ and reach its highest value of $0.0193\mu\text{Ci}$ 2 days later (Table 5).

The procedure proposed to handle the radiological assessment of multiple fragmentation wounds is described in the next section following the discussion of the previous results.

DISCUSSION

Biokinetic models for decorporation have been developed as under treatment the disposition of a radionuclide is modified, so that the original biokinetic model cannot be used any more. Hall *et al.*³¹ developed an empirical model to describe the modified urinary excretion of plutonium under decorporation therapy. The pharmacokinetics of DTPA itself has been studied in humans and animals and can be described by a 3-compartment model³² used to develop a DTPA decorporation model.^{33,34} In this study, we used a simpler approach, assuming an effective residence time of 24 h for DTPA during the treatment and a complete

Table 3. Effect of decorporation treatment with diethylenetriaminepentaacetic acid after the inhalation of a poorly soluble plutonium-239 compound or injection into a wound of a soluble compound ($1\mu\text{Ci}$) depending on the duration of the treatment

Treatment Duration						
	None	14 Days	30 Days	90 Days	120 Days	180 Days
Committed effective dose (mSv)						
Inhalation	260	152	87	15	8.2	4.7
Wound	823	199	149	50	29	10
Therapeutic efficacy						
Inhalation	0	0.42	0.67	0.94	0.97	0.98
Wound	0	0.76	0.82	0.94	0.97	0.99

The initiation of treatment was assumed to be 1 h after acute inhalation or injection into the wound. The dose reduction factor was calculated by dividing the committed effective dose absorbed under treatment by the dose without treatment. Efficacy was estimated by subtracting the dose reduction factor from one.

Figure 2. Time course of radioactivity in the blood compartment depending on the time DTPA treatment is initiated after the injection of $1\mu\text{Ci}$ of plutonium-239 as a soluble compound into a wound. Treatment duration is assumed to be 14 days. Simulations are based on a simplified wound model linked to the ICRP systemic model of plutonium, further adapted to study decorporation biokinetics by Stricklin *et al.*²⁹ DTPA, diethylenetriamine pentaacetic acid.

binding of plutonium in the blood compartment.²⁹ This seems justified in view of the large DTPA concentration excess and the binding constant of the DTPA-plutonium complex ($\log K=23.4$ at physiological conditions).³⁵ In this model, the chelated plutonium amount is added to the amount excreted in the urine.²⁹ We used this model to focus on the impact of the initiation time and duration of a decorporation treatment after plutonium incorporation by inhalation or through a wound, as up to now

Figure 3. Time course of radioactivity in the blood compartment when DTPA treatment is started immediately at the same time as the injection of $1\mu\text{Ci}$ of plutonium-239 as a soluble compound into a wound. Treatment duration is assumed to be 14 days.

there is no consensus on the concrete time frame in which a decorporation treatment should be started.

Our simulation results confirm that the efficacy of a decorporation treatment depends on the initiation time. This is not surprising from a theoretical point of view, as distribution between the compartments and elimination processes in the systemic biokinetic model for plutonium are described as first-order processes. If DTPA treatment is started immediately, at the same time as the activity injection of a soluble compound into a wound, the maximum activity in the blood compartment never reaches the peak values observed without treatment at the end of the day following incorporation ($0.165\mu\text{Ci}$ at 1.93 days) (Figs. 2 and 3 and Table 4). As shown previously (Rump *et al.*, unpublished observations), the impact of the treatment initiation time is particularly important when the invasion speed is high. Our data actually show that the loss of efficacy is more pronounced if the initiation of treatment is delayed after the incorporation of a soluble plutonium compound through a wound, compared to the inhalation of a poorly soluble compound. However, starting treatment 90 days after the incorporation or later does not seem to make a significant difference in efficacy. It may be assumed that at this time point, a large part of the plutonium of the central compartment has already entered the main target organs (bone, liver), which are deep compartments releasing plutonium into the central compartment only at a very slow rate. Although DTPA has been shown to enter the intracellular spaces in small amounts,³⁶ it distributes mainly in the extracellular space,³² *i.e.*, the central compartment, and plutonium trapped in the bone and liver tissues is very difficult to remove.

The second factor affecting decorporation efficacy is the duration of the treatment, as previously shown in different studies.^{29,37,38} When ending the DTPA treatment, a low activity in the blood compartment again starts to increase, and if started early (1 h) and limited to 7 days after the incorporation of a soluble compound through a wound, even reaches higher values ($0.0193\mu\text{Ci}$ on day 9) than on the 1st day shortly after incorporation ($0.0117\mu\text{Ci}$ at 1 h) (Fig. 4 and Table 5). Our data also show that prolonged treatment seems to be more important after the incorporation by inhalation than through a wound. Extending treatment duration from 15 to 30 days improves efficacy by 25% (from 42% to 67%) after inhalation, but only by 6% (from 76% to 82%) after absorption through a wound. This is not really surprising, as the invasion of the poorly soluble plutonium compound

Table 4. Maximum activity in the blood compartment (A_{max}) and time this maximum is reached (T_{max}) depending on the initiation time of diethylenetriaminepentaacetic acid treatment after injection of $1\mu\text{Ci}$ of plutonium-239 as a soluble compound into a wound

	No Treatment	Treatment Initiation Time			
		Immediately	7 Days	14 Days	21 Days
A_{max} (μCi)	0.165	0.0062	0.165	0.165	0.165
T_{max} (days)	1.93	17.35	1.93	1.93	1.93

Treatment duration was set at 14 days.

from the lungs into the blood is more protracted in time, so that maintaining a sufficient DTPA concentration in the central compartment for a longer time period permits binding and elimination of a larger amount of plutonium before redistribution into the target organs. Our results also show that for a treatment initiated early after incorporation (1 h) and lasting 90 days, the efficacy has reached 94% for both invasion pathways, and so may not be substantially improved by further extending therapy. Avoiding an unnecessary treatment does not only permit the reduction of costs and adverse effects, which are quite slight in the case of DTPA at the recommended dosages,¹² but may also be an ethical imperative in such cases where a large number of patients must be treated and DTPA stockpiles are limited. It must, however, be emphasized that limiting treatment duration to 90 days is not a general rule and among other factors may depend on the specific radionuclide(s) and chemical forms involved as well as the incorporated activities (Rump *et al.*, unpublished observations).

Figure 4. Time course of radioactivity in the blood compartment depending on the duration of DTPA treatment when treatment is initiated 1 h after the injection of $1\mu\text{Ci}$ of plutonium-239 as a soluble compound into a wound. Simulations are based on a simplified wound model linked to the ICRP systemic model of plutonium, further adapted to study decorporation biokinetics by Stricklin *et al.*²⁹ For reasons of clarity, the activity time course for a treatment duration of 14 days is not shown.

Therapeutic efficacy, as defined in this article, provides a means to normalize the effect of a decorporation therapy and is useful for the purpose of comparison. However, to assess the reduction of the stochastic health risks, it is important to know by how many mSv the effective dose has been reduced by. The dose depends not only on the total activity incorporated, the radionuclide(s), its physical half-life, and the energy of the emitted radiation but also on the invasion kinetics and therefore the invasion pathway and the physico-chemical properties of the compounds involved (Rump *et al.*, unpublished observations). Although the absorption of a soluble plutonium-239 compound through a wound is obviously slower than in the case of an intravenous bolus injection, the invasion kinetics may nevertheless be considered as fast (in our simulation 0.241 day^{-1}) compared to the transfer rates into the target organs (blood–liver 0.1941 day^{-1} , blood to bone cortical surface 0.1294 day^{-1}), and very much faster in comparison to the absorption of a poorly soluble compound from the lungs (the final dissolution rate for an S-compound is estimated to be 10^{-4} day^{-1}). This might explain why although the incorporated activity is similar ($1\mu\text{Ci}$), the resulting committed effective dose without treatment is several times higher after the fast incorporation through the wound (823 mSv) than after the slower absorption of a poorly soluble compound by inhalation (260 mSv). Moreover, the insoluble form deposited into the lung is removed from the respiratory tract through, for example, the mucociliary action, which clears some of the internal contamination through the GI

Table 5. Maximum activity in the blood compartment (A_{max}) and time this maximum is reached (T_{max}) depending on the duration of a diethylenetriaminepentaacetic acid treatment started 1 h after injection of $1\mu\text{Ci}$ of plutonium-239 as a soluble compound into a wound

	No Treatment	Treatment Duration		
		7 Days	14 Days	21 Days
A_{max} (μCi)	0.165	0.0193	0.0117	0.0117
T_{max} (days)	1.93	9	0.042	0.042

tract. These results confirm our previous findings (Rump *et al.*, unpublished observations) that in the case of a faster invasion kinetic, there is a combination of a higher committed effective dose without therapy with a more rapid loss of efficacy if decorporation treatment is delayed.

This leads to the general question of the validity of the biokinetic models used for the computation of the committed effective doses, as these cannot be directly measured. These models have been conceived for a “standard” person, and default parameters are based on human data, animal experiments, and the properties of well-known elements showing biochemical similarities, as far as data are available.³⁹ The wound model possesses several particularities.^{20,21} The compartments represent different physicochemical states and not anatomical structures (1. soluble state, 2. colloid and intermediate state, 3. particles, aggregates, and bound state, 4. trapped particles and aggregates, 5. fragments, 6. lymph nodes, and 7. blood). In our simulations, we have used a simplified model accounting for the soluble, colloid and intermediate states, and absorption occurred directly into the blood as the central compartment of the systemic model for plutonium. Thus, the wound model was reduced from seven to three compartments. Moreover, the parameters used in our model are based on the data of Konzen and Brey,³⁰ and the resulting retention function shows elimination rates that are much lower than the NCRP values as follows. In our function $[A(t) = 0.976 \cdot e^{-0.3427 \cdot t} + 0.024 \cdot e^{-0.0183 \cdot t}]$ with t in days, the shortest half-time corresponds to 48 h 30 min ($\ln 2 / 0.3427$), whereas for a soluble “weak” compound in the NCRP model, the highest elimination rate corresponds to a half-time of 15 min. Thus, it is not surprising that the committed effective dose calculated with our model is much lower than the values given by commercial software that uses NCRP parameters as default values. Another big issue is that the wound model does not take into account the different types of wounds from a clinical perspective (*e.g.*, cuts vs. burns), although differences regarding the absorption of contaminating material might be expected.⁴⁰ It seems that the speed of absorption of radioactive material into the blood is decreasing in the following order: stab wounds > deep lacerations involving muscles > lacerations > abrasions > chemical burns > heat burns.⁴¹ As stated in the introduction, in the case of a “dirty bomb” attack, victims will probably be hit by a large number of fragments and particles of different sizes. From a practical point of view, it does not seem feasible to represent each particle impact by an own invasion pathway. That is why

we propose a practical method that can be used at a very early point of time in the emergency room and permits to aggregate multiple fragmentation injuries to a single wound for an early approximate assessment of the radiological risks by internal wound contamination.

A model for cases of disseminated multiple fragmentation wounds

The total area covered by small wounds can be easily assessed with the “rule of nine” or the “rule of palms” (the palm + the fingers of the patient correspond roughly to 1% of the body surface), well known to emergency physicians and used for burn size estimation.¹ Results are expressed as percentage of the total body surface but can be transformed to a surface unit (*e.g.*, cm²) assuming an average total body surface of 1.7 m² for an adult (body surface = $0.01667 \times [\text{height in cm}]^{0.5} \times [\text{weight in kg}]^{0.5}$).⁴² After identification of the radionuclide(s) involved, the activity per unit area (Bq/cm²) on the surface of the patient can be monitored using a commercial contamination monitor. In the next step, the highest measured activity per unit area (“worst case” principle) is multiplied with the total area covered by multiple fragmentation wounds, to give the best early estimate of the total activity contaminating the wounds. After deciding on assumptions regarding the physicochemical state of the contaminating material (liquid, small particulates, larger fragments) based on surgical inspection, this activity could be used for a first approximate computation of the resulting committed effective dose in the case in which no decorporation treatment is initiated. It must be emphasized that this procedure might be useful for radionuclides emitting gamma-radiation, but can very easily lead to erroneous results in the case where only beta- or alpha-radiations are emitted because of their absorption in the surrounding tissue. As this method permits just a very rough assessment, further examinations (*e.g.*, whole body counting, excretion measurements) must be performed later on to give more precise results on the health risks.

INNOVATION

In the case of a “dirty bomb” attack, compounds with different physicochemical properties may be involved. Thus, it is prudent to start decorporation therapy as soon as possible and to continue it for several months, unless the activity incorporated is shown to be insignificant afterward. A loss of efficacy by a delayed treatment start cannot be

compensated later on. In the case of multiple contaminated fragmentation wounds, measuring the activity on the body surface and applying the “rule of nine” will permit to approximate the radiological health risks by internal dosimetry at an early point in time.

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AUTHOR DISCLOSURE AND GHOSTWRITING

A.R., A.L., S.E., M.A., and M.P. are employed at the Institute of Radiobiology of the Bundeswehr in Munich. D.S. is employed at Applied Research Associates, Inc. in Arlington. All authors contributed to this study and no ghostwriter was used.

KEY FINDINGS

- After an attack with a “dirty bomb” and an internal contamination with radionuclides, the resulting committed effective dose predominantly depends on the speed of invasion, *i.e.*, the pathway (*e.g.*, inhalation and/or through a wound) and the physicochemical properties (solubility) of the compounds involved.
- After the incorporation of radionuclides, decorporation treatment should be initiated as soon as possible because delays result in a loss of therapeutic efficacy.
- Multiple fragmentation wounds that are radioactively contaminated might be aggregated to a single wound model suited for internal dosimetry calculations by using the “rule of nine.”

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Abbreviations and Acronyms

Ci = Curie
 DTPA = diethylenetriaminepentaacetic acid
 ICRP = International Commission on Radiological Protection
 NCRP = National Council on Radiation Protection and Measurement
 RDD = radiological dispersal device
 Sv = Sievert