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Civilian exposure to munitions-specific carcinogens and resulting cancer risks for civilians on the Puerto Rican island of Vieques following military exercises from 1947 to 1998

Hans Sanderson[a] [ID], Patrik Fauser[a], Ricky Steven Stauber[b], Jesper Christensen[a] [ID], Per Løfstrøm[a] and Thomas Becker[a]

[a]Department of Environmental Science, Aarhus University, Roskilde, Denmark; [b]Independent Consultant, Waldorf, MD, USA

ABSTRACT

Estimation of legacy public health risks from munitions residues near or at former military test ranges has for the past decades been a challenge to health authorities. Parts of the island of Vieques (PR) were for six decades used for military training, and these are now declared as a Superfund site. ATSDR has conducted site assessments there and found no cause for public health concerns. The reports and findings of ATSDR have since been heavily contested and disputed. This paper provides a case study on cancer risk screening of munitions-specific carcinogens for the full period of military training on Vieques. Added cancer risks and Margins of Exposure for the different carcinogens for each year were derived. We found that there is a potential for cancer risk concern related to BaP exposures. Furthermore, there were health risks from TNT exposures. The primary exposure route of these compounds was oral. The period 1992–1997 showed a significantly elevated lung and bronchus cancer incidence rate in Vieques compared to Puerto Rico mainland mainly among women <50 yr and men 50–64 yr. These correlate with high munitions exposures in the period 1977–1984.

Introduction

Estimating the human health risks from historical and legacy distributed munitions residues from military test ranges has for the past decades been a challenge to authorities (Phillips & Perry, 2002). This is because there generally is little information available regarding estimating the public health risks of military-unique releases to humans via environmental pathways from past activities (Phillips & Perry, 2002). The aim of this paper is therefore to provide an example of how a risk assessment can be developed to help prioritise further empirical risk research with an emphasis on cancer risks. We chose the Puerto Rican island of Vieques, which has been used as a military test area for more than six decades by the U.S. military. The Navy engaged two-thirds of the island's 9000 acres where military exercises tested live ammunition. Testing was open on average 180 days per year (AJPH, 2001). Meanwhile, between 9000 and 14000, inhabitants lived eight miles away from the ranges during the period. The first large-scale war games took place in 1948 involving more than 60 war ships, 350 planes and 50,000 troops from all branches of the military. In the early 1980s, an average of 3400 bombs were deployed, 158 days of naval bombardment, 200 days of air-to-ground combat exercises and 21 days of marines practising invasions per year on the island. Over 15 years from 1983 to 1998, the military deployed more than 17.7 million kg of munitions on Vieques (Davis, Hayes-Conroy, & Jones, 2007). In 2005, the United States Environmental Protection Agency (USEPA) listed the Vieques bombing range as a Superfund site (Davis et al., 2007). After the closure and the Superfund status, the US EPA commissioned an assessment of ecological and human health risks. The current conclusion regarding human health risks is that the exposure is not under control – meaning that; (1) contamination has been detected at a site at an unsafe level; and (2) a reasonable expectation exists that people may be exposed to the contamination (US EPA, 2016b). The remedial efforts have so far resulted in surface clearance of more than 10.25 km^2 cleared of munitions; over 38,000 munitions items have been removed and destroyed. Sub-surface clearance includes a total of 15.3 km of roads cleared (includes a 7.5 m buffer on either side) and 17 km of beaches cleared.

Figure 1(a–b). A: Detonation of a bomb; B: munitions recovered on Vieques. Source: AP Worldwide photos.

Over 7.7 million kg scrap metal processed and over 6 million kg recycled – see Figure 1(A) and (B) below (US EPA, 2016a).

The population on Vieques is concerned about what this means concerning safety of local food, as well how this relates to potential elevated cancer risks (Phillips & Perry, 2002). The *American Journal of Public Health* reported that from 1960 to 1979 the cancer rates on Vieques were lower than those on the mainland of Puerto Rico. However, that the rates for the period 1985–1994 increased and exceeded alert levels adopted by ATSDR, prompting the Puerto Rican Legislature to mandate an epidemiological study of the cancer rates in Vieques (AJPH, 2001). Hence, the objective of this paper is to provide a case study of how historical exposures and risks to carcinogens originating from munitions can be assessed; moreover, with this knowledge to prioritise compounds of concern and their exposure pathways in support of future site-specific risk assessments.

Methods

In the following is a brief contextualisation of the study area, the demographics, the history and use of munitions, as well as a presentation of the current ATSDR model and exposure parameterisation and findings, which we subsequently update in the following sections where we will assess the public health risk following the exposures of carcinogenic munitions residues. The terrain was observed during a site visit we conducted in 2014. The demographics were assessed based on US census data. The munitions used was assessed based on the historical records of all military activities on Vieques since the 1940s to 2000 from the US National Archives to derive a comprehensive overview of the loading of carcinogenics associated to the munitions used over time on to the island. The 2013 ATSDR report on Vieques was used to derive the state-of-the-art measured exposures and model parameterisation

used by the ATSDR in assessing the exposures and risk from the munitions on Vieques.

Vieques

Isla de Vieques is an island in the US Commonwealth of Puerto Rico and is located approximately 13 km east of the main island of Puerto Rico. It is approximately 34 km long and 6 km wide, and covers an area of 348 km^2 (Figure 2). The island has a relatively flat topography with rolling hills with the highest point at 300 m. The land vegetation is subtropical dry forest. The test areas were cleared sandy areas when in use. Today these are more or less covered with grass, scrubs and trees.

Until 1 May 2003, the United States Navy used half of the land on Vieques and conducted military training exercises on the east side of the island from 1947 to 2003. These air-to-land, ship-to-shore and land-based exercises, that included various types of bombing and shelling ordnances, took place at the three training areas Live Impact Area (LIA), Secondary Impact Area (SIA), Eastern Manoeuvre Area (EMA) and Eastern Conservation Area (ECA), see Figure 3, which are located at distances between approximately 4–13 km east of the two main residential areas Isabel Segunda and Esperanza.

Concerning the population living on Vieques, the 2000 USA Census Bureau profile for Vieques documents that the population was 9106 in 2000. The demographics of Vieques and Puerto Rico are shown in the Table 1 below.

Smoking prevalence in Puerto Ricans living in the US is the highest among the Hispanic community at 35% for men and 32.6% women (Kaplan et al., 2014) and higher in Vieques than in Puerto Rico (Department of Health PR, 1999). There is a difference in the percentage of older citizens in Vieques compared to Puerto Rico. The demographics show that adults in the working age typically leave the island to find jobs on the mainland and elsewhere, which was also confirmed to us during our fact-finding visit in 2014. The visit also determined that there is no
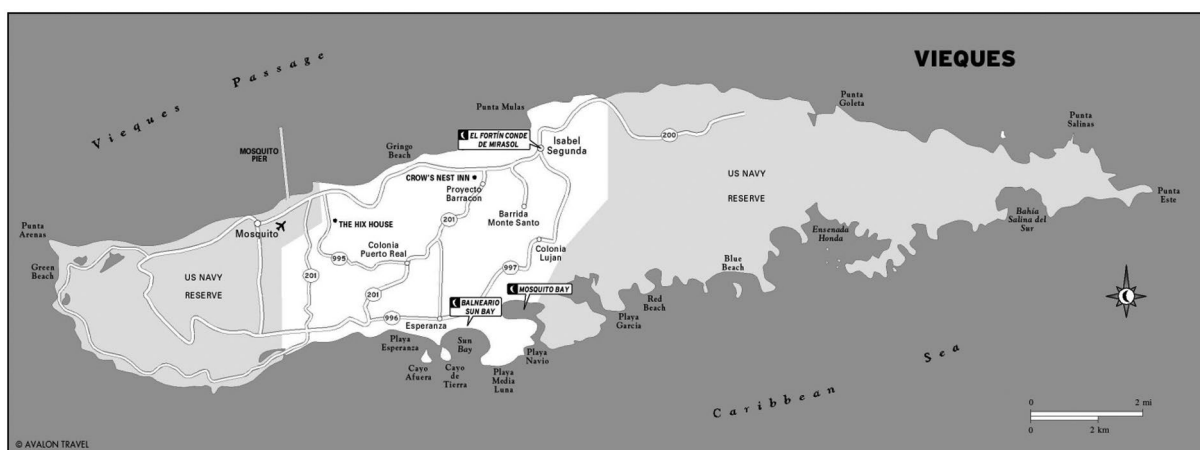
Figure 2. Vieques.
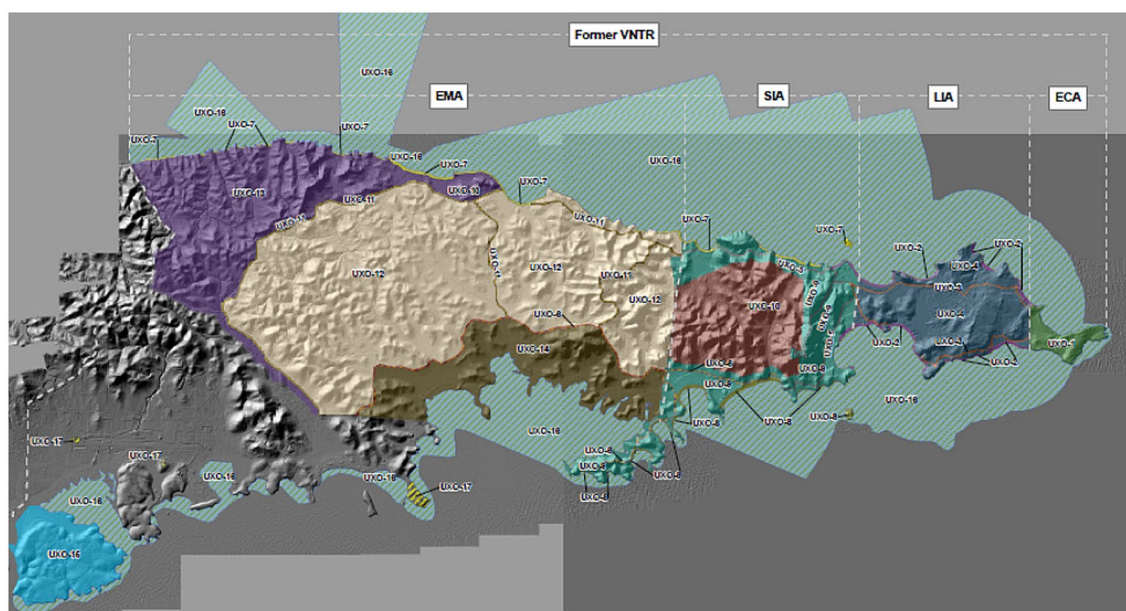
Figure 3. Former US Navy reserve on Vieques.

Table 1. Demographic data (Census, 2000).

Parameter	Vieques	Puerto Rico
Total population	9106	3.808.610
Mean age	34.5	32.1
<18 yrs	29.7%	28.7%
>65 yrs	13.9%	11.2%
Ethnicity	97.4% Latino	98.8% Latino
Family households	71.3%	79.6%
Smoking (ever)	38%	32.6%
Smoking (current)	46.4%	35.1%

heavy industry contributing with carcinogens. The island of Vieques does not have any additional unusual and identifiable environmental sources of cancer-causing materials arising from anthropogenic activities including industrial waste sites and soil contamination. Hence, any potential elevated cancer incidence rates observed on Vieques compared to Puerto Rico is conservatively, *ceteris paribus,* preliminarily attributed to the military activity, until otherwise refuted.

Munitions registry

To assess the amounts of munitions residues and carcinogens over the course of the entire training period, we retrieved munitions data from the US Marine Corps Training Exercises from the U.S. National Archive's in College Park, Maryland. All records of training exercises and manoeuvre reports on the island from 1941 to 2003 were retrieved (Munitions Registry, 2016) . In addition, annual and summary reports were retrieved of the activities (e.g. Final Draft, Preliminary Range Assessment

Report, Vieques Naval Training Range, Vieques Island, Puerto Rico: Naval Facilities Engineering Command, Atlantic Division, 1510 Gilbert St., Norfolk, VA 23451-2699, April 2003). Based on the reports in the munitions registry (2016), we retrieved the following information:

- Types of munitions used each year (e.g. projectile, rocket, bomb, grenade, missile)
- Classification (e.g. aircraft gun, naval gun, air launched)
- Caliber/size of munitions
- Nomenclature of the munitions (e.g. M-56)
- Fillers used (see list below)
- CAS No. of fillers
- Use location codes (LIA, SIA and EMA locations)
- When (year)
- Amount of each filler, casing and projectile material in each munitions type (kg)
- Amounts (number of total ordnances fired each year)

From 1999 to 2003, only practice bombs were used, without explosive fillers. Annual use amounts of the different munitions types for each year were retrieved. Annual uses are evenly distributed on single days, assuming that military training exercises occurred 200 days per year prior to 1999. Emissions are set to zero between 11:00 PM and 7:00 AM every day. This diurnal 16-h testing profile reflects the time of day when the Navy used live bombs prior to 1999. There are many different types of munitions – here are just a few examples: 1940s and 1950s: Mk-79 1000 lb napalm fire bomb (gasoline) on SIA; 1960 and 1970s: Mk-55 5″/54 COMP A-3 (91.7% RDX and 9.7% stearic acid) was often used both on SIA and LIA; 1980s and 1990s: Mk-82 general-purpose 500 lb bomb with tritonal (80% TNT and 20% Chaff) was often used; in 1981; 8467 Mk-82s was used on the LIA. There were also 263 rounds of 25 mm aircraft gun PGU-20 containing 150 g depleted uranium, which was used at LIA in the period (Munitions Registry, 2016). These data allowed us to determine the amount of carcinogens applied to the island for each year and in total.

Carcinogenic munitions-related pollutants

The IARC Monographs, Volumes 1–112 (IARC, 2015), was used to screen the complete list of explosive fillers from the munitions registry, chemical by-products of explosion and metals from soil and casings for carcinogenicity according to the Agents Classified. The following organic compounds and metals were thus included in our assessments (Table 2).

Destruction efficiencies for high explosives have not been measured for live bombing; however, emission factors (ATSDR, 2003) state that more than 99% of high

Table 2. Carcinogenic munitions fillers (high explosives charges).

Compound	CAS#
TNT	118-96-7
RDX	121-82-4
HMX	2691-41-0
HBX-1	118-96-7 & 121-82-4
Ethylene oxide	75-21-8
Gasoline	86290-81-5 & 8006-61-9

explosive organic chemicals are destroyed, thus making accounting of open detonation of unexploded ordnance unnecessary. A conservative estimate by ATSDR (2003) is that 10% of the organic chemicals in high explosive charges are not destroyed and thus emitted. Emission factors for the resulting chemical by-products of explosions are derived from various types of ordnance from air to ground (ATSDR, 2003). They are high explosives containing some combination of TNT, RDX and aluminium powder, which constitute approximately 95% of total amount of filler used. We assume that the remaining fillers emit equal amounts of by-products (Table 3).

The munitions also consist of metals and metalloids from ordnance casings, traces in explosives and soil crater ejecta. On impact, the following compounds are released. Emission rates for crater ejecta, traces in explosives and metal composition in casings were retrieved from ATSDR (2003). ATSDR (2003) conservatively assumes that the entire casings are vaporised in every explosion (Table 4).

ATSDR exposure assessment

In this section, we summarise the results that ATSDR has found on occurrence of carcinogens from modelling studies and from measuring campaigns (ATSDR, 2013). These data and information are the starting point for our modelling and are briefly summarised below for air; soil; produce and livestock; fish and seafood; drinking water. With respect to the air compartment, which is the driving media for transport of contaminants following explosions, a number of sampling campaigns were conducted by the ATSDR. However, few air samples were collected on Vieques between the early 1970s and 1998, the years when the Navy's military training exercises using live bombs were most intensive. The overall results from the air compartment analysis were that the uncertainties inherent in the modelling analyses had been adequately addressed using worst-case assumptions by overestimating the aspects of contaminant emissions. Despite this fact, the result was that airborne contaminants would have been essentially non-detectable in the residential areas, and would not have resulted in harmful effects (ATSDR, 2013).

Direct exposure of residents of contaminated soil through swallowing or touching was evaluated based on

Table 3. Carcinogenic explosive filler by-products.

Compound	CAS#
2,4-dinitrotoluene	121-14-2
2,6-dinitrotoluene	606-20-2
1,3-butadiene	106-99-0
1,4-dichlorobenzene	106-46-7
Benzene	71-43-2
Benzo(a)pyrene	50-32-8
Carbon tetrachloride	56-23-5
Naphthalene	91-20-3
Vinylchloride	75-04-1

Table 4. Carcinogenic heavy metals from casings, fillers and projectiles.

Compound	CAS#
Arsenic	7440-38-2
Beryllium	7440-41-7
Cadmium	7440-43-9
Chromium VI	18540-29-9
Cobalt	7440-48-4
Lead	7439-92-1
Mercury	7439-97-6
Nickel	7440-02-0
Selenium	7782-49-2
Strontium 90	10098-97-2
Titanium dioxide	13463-67-7
Vanadium pentoxide	1314-62-1
Aluminium powder (Chaff)	7429-90-5
Depleted uranium	7440-61-1

measurements and transport estimates. The only soil data available from the residential section of the island were the 1972 US Geological survey data, which were considered of unacceptable quality due to missing information on sampling depth and soil conditions. To address remaining uncertainties, ATSDR recommends surface soil sampling in residential areas (ATSDR, 2013).

With regard to indirect exposure via food, the conclusion by ATSDR (2013) is that the overall data are insufficient to quantify adequately human exposures or draw any valid health conclusions about whether consuming locally grown produce and livestock would result in harmful health effects or not. Analysis showed that some fish and shellfish from certain reefs surrounding Vieques had higher levels of some metals (e.g. arsenic and selenium) and lower levels of other metals, compared with other reefs surrounding Vieques. HMX and trace levels of RDX explosives compounds were found in the fiddler crabs from the LIA. ATSDR recommended that different measurements and model analysis were needed to clarify the occurrence, location and risk of pollutants in marine seafood (ATSDR, 2013).

Drinking water sources on Vieques is via a pipeline supply from the Puerto Rico main island, public and private wells and collection of rainwater. A lack of adequate historical data from the 1970s and 1980s' public supply wells and collection of rainwater prevents a conclusion of the extent of exposure to residents. ATSDR (2013) states that regarding private wells, the groundwater flow appears to preclude contamination of relevant aquifers.

Updated exposure modelling

We used the same assumptions and model parameters as the ATSDR study (ATSDR, 2013), e.g. regarding emission factors of metals and explosion by-products and fraction of unexploded ordnance; however, we have updated important parts, especially with respect to the munitions amounts, dispersion models and meteorological parameters. We used the European Technical Guidance Document (EU TDG, 2003) as the source for the modelling since this is in our view the most accurate and comprehensive set of exposure models used in global environmental assessment and regulatory affairs. The results of the ATSDR analysis indicate that there is a need to (a) derive the additional data to reduce the uncertainty and thereby get the exposure under control; (b) more urgent and importantly, to refine the environmental exposure pathway modelling to allow a prioritised sampling campaign; and (c) to focus on specific diseases and thereby exposures to target first (e.g. cancer and carcinogens as highlighted by the *American Journal of Public Health* (2001). There are some basic assumptions and data used primarily in the atmospheric transport modelling that should be improved in order to obtain a more correct and realistic exposure assessment. The atmospheric transport of airborne pollutants is the main driver for exposure to residents. Specifically, Puerto Rican meteorological data from surface measurements, upper air measurements and precipitation data were used in our models. Puerto Rico has a surface area of approximately 9000 km² and compared to the 348 km² of the adjacent Vieques, the local wind conditions, which are strongly influenced by sea-breezes, which again is governed by the size of land, may be considerably different to the wind directions and speed on Vieques. ATSDR reviewed nearly six years of range utilisation statistics to characterise the most intense bombing activity over a 24-h period. The data source is not considered exact and complete in terms of covering the use of ordnance types, their amounts and composition of explosive fillers and metals. With the identified munitions registry data, we can model the munitions residue transport through environmental exposure pathways from the firing ranges to the residents of Vieques. Pollutant doses to the residents in the two major cities on Vieques from exposure via the environment and ingestion of water and food are quantified. The specific conceptual exposure model for munitions on Vieques (Figure 4) illustrates how munitions residues are transported via different pathways from the explosion in the test area towards people in the civilian area.
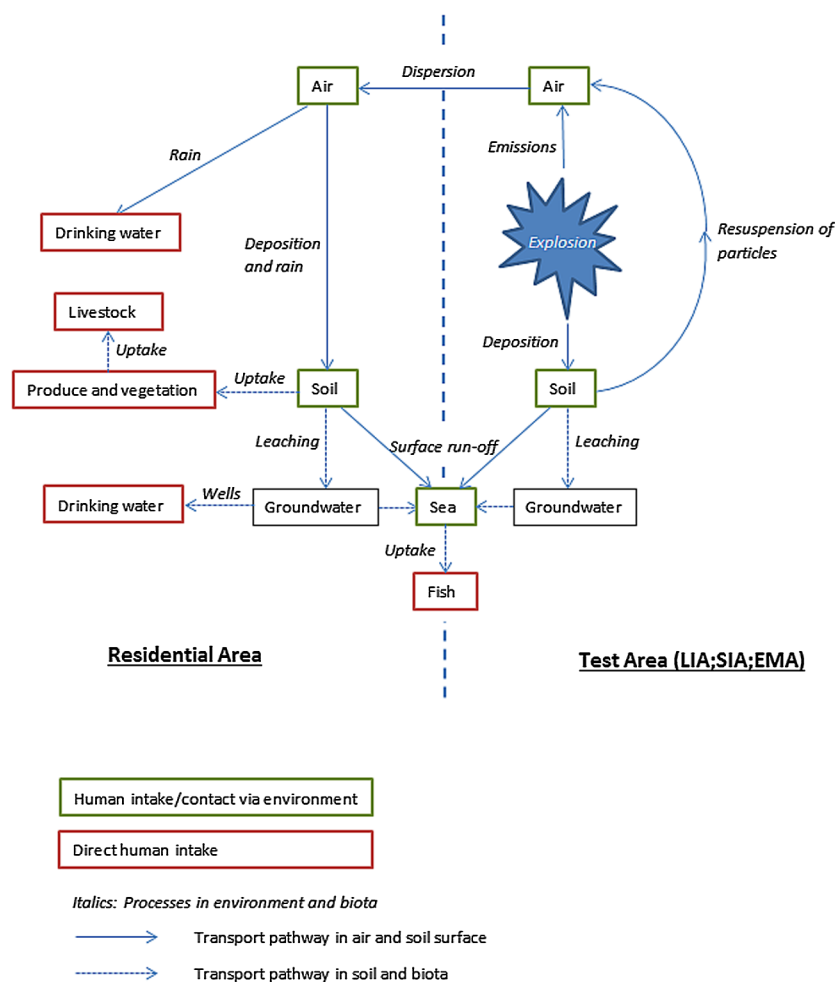
Figure 4. Vieques, munitions-specific conceptual pathway model for human exposure.

We considered the impact of carcinogens from the use of munitions ordnance from air-to-land, ship-to-shore and land-based exercises that took place in the years from 1947 to 1998 on Vieques. The exposure of carcinogens is calculated for residents in the two main populated areas on Vieques, i.e. Isabel Segunda on the northern shore and Esperanza on the southern shore both located approximately 4 km from the western border of the EMA. No other anthropogenic industrial or private activities have been found to release significant amounts of the considered carcinogenic compounds based on two independent site visits. To estimate the accumulated exposure and dose of carcinogens to the population during the 52-year period of munitions testing, a 52-year annual mean exposure is calculated from a model run of the total summed use for all years in one year, and dividing by 52. Additionally, a worst-case year is calculated separately for the year with maximum munitions use to illustrate the range of exposure.

Atmospheric compartment

Atmospheric dispersion modelling was performed with the OML-Multi model, a multi-source version of the atmospheric Gaussian plume dispersion model OML (Olesen, Berkowicz, Ketzel, & Løfstrøm, 2009; Olesen, Berkowicz, & Løfstrøm, 2007a, 2007b). It was used to assess air pollution from point and area sources and can be used at distances up to around 20 km from the source. The model accounts for plume rise due to gas temperature and exit velocity and the effects of nearby structures, and in this case, the emission plume was defined as a volume source. Information on emissions (in UTM coordinates) and meteorology on an hourly basis was needed, and is applied during one year of hourly calculations. The meteorological data were calculated by The Weather Research and Forecasting (WRF) Model (Skamarock et al., 2005), which uses global meteorological data (Dee et al., 2011). Worst-case meteorological conditions were derived based on a sensitivity analysis where hourly meteorological data

for an entire year are tested for the two residential areas, the centre of LIA and the centre of SIA. Explosions took place at many different sites within LIA, SIA and EMA. Therefore, emissions were allocated as volume sources, at the lower left corner in a $500 \times 500 \times 500$ m³ grid with constant and evenly distributed emission rates, found from fractions of different emission areas (LIA, SIA and EMA) in each grid area. The centre of the continuous elevated volume explosion cloud is between 285 and 424 m (ATSDR, 2003), and the lateral dimensions (44–66 m) which is small compared to the grid size and the distances to the receptor areas are set equal to the grid size. The emission height is assumed to range between 0 m and 850 m (divided in seventeen 50 m segments). These dimensions do not describe the entire cloud, and as a conservative assumption, the skirt, which deposits relatively quickly, is not considered. All pollutants are assumed to be associated to PM10 (in reality larger particles occur), which is a conservative measure, as deposition becomes lower and air concentration thus higher. The settling velocity is 0.3 cm/s. Over the course of an hour, or the time it generally takes the wind to blow from the LIA to the residential areas of Vieques, particles would be expected to have settled approximately 10 m, on average, which means that the entire 'skirt' of the emission cloud would have settled before reaching the residential areas. The use of a cloud with the above dimensions therefore represents a realistic worst-case situation. Complex terrain topography is not considered because the estimated initial cloud heights were greater than the elevations of the local terrain features (ATSDR, 2013). At the time of explosions, there was limited or no vegetation on the LIA; this minimises the terrain effect and enhances the suspension of soil particles (Figure 2). The total human exposure concentration after any number of years is additive according to each annual mean concentration.

Soil compartment

The airborne pollutants deposit on soil in the residential areas where residents can be exposed directly via swallowing or touching surface soil or household dust contaminated by past or ongoing (at the time) military activities. From the soil, the pollutants can leach to groundwater and enter drinking water wells, surface run-off to the sea with fish and marine predators being exposed and be taken up by crops and livestock. Assuming a homogeneously mixed 5-cm top soil layer with no macro-pore and symmetry along the horizontal plane, and assuming that the diffusive transport from the topsoil is negligible compared to deposition and vertical flow, the governing differential equation for the total ($C_{soil,tot}$) and dissolved pollutants in the soil pore water ($C_{soil,diss}$) in mg/m³ in the top soil is:

$$\frac{dC_{tot,diss}}{dt} = \frac{F_{dep}}{h}|_{z=0} - q \cdot \frac{dC_{tot,diss}}{dz} - k_1 \cdot C_{soil,diss} \quad (1)$$

where dt is the time step; F_{dep} is the annual mean atmospheric deposition of pollutant in mg/(m² × year); h is the top soil layer thickness (0.05 m); $R = (\theta + Kd \times \rho)$ is the retention factor; θ is the pore volume fraction in the soil 0.5; $Kd = foc \times Koc$ is the partitioning coefficient between dry matter and water in L/kg dw; foc = 0.02 is the fraction of organic carbon in particulate matter (kg OC/kg dw); Koc is the partitioning coefficient (sorption coefficient) between organic carbon and water (L/kg OC); and Xs is the density of soil 1.3 mg/kg dw/L; q is vertical flow of water from homogeneous top soil 20 cm/year; dz is step in vertical direction in m; k_1 is the first-order degradation rate of pollutant in soil in s⁻¹ (Lugo-Lopez, Bonnet, & Garcia, 1953).

Steady-state conditions ($dC_{soil,tot}/dt = 0$) can be assumed as we consider annual mean concentrations; this yields the following mean annual steady-state concentration of dissolved pollutant in the top soil pore water:

$$C_{soil,diss} = \frac{F_{dep}}{(q + k_1 \cdot h)} \quad (2)$$

Leaching to groundwater and surface run-off to sea

The fraction (X_L) of a (dissolved) pollutant that is leached from the top soil compared to the total deposited pollutant (sorbed + dissolved) is:

$$X_L = \frac{q}{(q + k_1 \cdot h)} \quad (3)$$

If the half-life $T_{1/2} = \frac{ln2}{k_1}$ of a pollutant is smaller than its Kd value, approximately all pollutants will be degraded in the top soil before it will be leached. Dissolved pollutants in vertical soil flow, q, can be divided in a fraction to groundwater and a fraction to the sea from surface run-off. Worst case for both is that the entire pollutant in q goes to the respective compartments.

Precipitation and drinking water

The annual mean concentration of pollutants in drinking water from rainwater collection at the residential sites, $C_{rainwater}$ (mg/m³), is calculated from F_{dep}:

$$C_{rainwater} = \frac{F_{dep}}{p} \quad (4)$$

where p is the annual mean precipitation of 1 m/year.

The drinking water is assumed to be composed of 50% rainwater and 50% groundwater from surface soil leaching. The fraction of rainwater is set high as worst case, and

using the surface soil pore water for groundwater is also a worst-case assumption. A complete removal of suspended particles from the groundwater, which corresponds with EU TGD (2015), but not from the rainwater, is assumed.

Marine compartment

In addition to surface run-off of deposited pollutant from land, direct atmospheric deposition to the sea constitutes the inflow of pollutants to the marine compartment. The annual mean concentration of bioavailable (dissolved) pollutants in seawater in the local area (1 km from the shore) around the island ($C_{sea,local}$ in $\frac{mg}{m^3}$) is:

$$C_{sea,local} = \frac{F_{dep}}{R \cdot d_{sea,local}} + \frac{q \cdot C_{soil,diss} \cdot A_{land}/A_{sea,local}}{d_{sea,local}} \quad (5)$$

where $d_{sea,local}$ = 30 m is the mean water depth within a 1 km distance of the shore of Vieques; $A_{land}/A_{sea,local}$ = 1 is a dilution factor accounting for the ratio between total land area and area of the sea that is the recipient to the surface run-off, assuming that all of q enter the marine waters as a worst-case scenario. R is the retention factor of pollutant in sediment, which is set equal to the retention factor in soil.

Fish caught by resident fishermen are assumed to be residing in the local contaminated sea water and consequently the predicted environmental concentration (PEC) of pollutant in fish meat is found from (EU TGD, 2003):

$$PEC_{fishment} = PEC_{sea,local} \cdot BCF_{fish} \cdot BMF_1 \quad (6)$$

where $PEC_{sea,local} = C_{sea,local}$ is the predicted pollutant concentration in the local deposition and run-off recipient area, BCF_{fish} is the pollutant bio-concentration factor in fish; and BMF_1 is the biomagnification factor. The latter two are found from the pollutant log Kow value according to EU TDG (2003).

According to EU TDG (2003), the direct uptake of pollutants from the environment, i.e. from water and sediment, is only of minor relevance to top predators like sharks and capitan, which are fish preferred by consumers. For a first tier (or trophic level) of predators, a worst-case assumption is that they obtain their prey equally from the local and regional areas, respectively. For the second tier (the top predators), it can be assumed that they obtain their prey mainly (approximately 90%, EU TGD 2003) from the larger scale regional marine environment. The regional scale marine environment, defined as 200 × 200 km², is assumed not to be influenced by the munitions testing activities due to dilution in the atmospheric and marine compartments. The concentration in top predator meat is found from (EU TGD, 2015) to be:

$$PEC_{toppered,meat} = PEC_{sea} \cdot BCF_{fish} \cdot BMF_1 \cdot BMF_2 \quad (7)$$

where PEC_{sea} is set to 10% of $C_{local,sea}$ and BMF_2 is the biomagnification factor for top predators, which is based on log Kow and BCF_{fish} (EU TDG, 2003).

Produce and livestock

Uptake and translocation from soil and gaseous uptake from air is accounted for, and only the concentrations in leaf and root tissue are estimated. The daily human intake amount of leaf includes fruit and cereals, and in order to calculate the total intake dose (amount × concentration) from leaf, fruit and cereals, the leaf concentration is used for all parts. Biotransfer factors are defined as steady-state concentrations in meat or milk divided by the animal's daily intake of the pollutant in media (air, grass, soil, drinking water). Fifty per cent of the grass intake is assumed to correspond to the leaf and root tissue concentrations, respectively. For all dairy products, the concentration in milk is used. All equations and default factors are taken from the EU TDG (2003) Part 1 Appendix III.

Total daily intake for residents

Standard daily intake values in L or kg per capita per day can be found for each source in EU TDG (2003) Appendix VII Tables 4 and 5. The daily dose ($Dose_{i,j}$) in mg/(kg body weight × day) of each pollutant is calculated for each intake medium from:

$$Dose_{i,j} = \frac{C_{i,j} \cdot IH_j}{BW} \quad (8)$$

where $C_{i,j}$ is the concentration of pollutant i in medium j in mg/(m^3 or kg), IH_j is the daily human intake value of medium j in (kg or m^3)/d and BW is the body weight of the considered human (default 70 kg).

Total dose of pollutant i is the sum of doses for all media. The annual mean dose is calculated by multiplying with 365 d/y. Physical/chemical parameters for pollutants are mainly obtained from the Hazardous Substances Data Bank (HSDB, 2017), US EPA (2014) and US Army Corps (2006) for TNT and RDX.

Cancer risk and margin of exposure

We used the US EPA integrated risk information system (IRIS. 2016) to derive acceptable exposure levels of the carcinogens and the ATSDR (ATSDR, 2016) data to complete the toxicity description of the compounds. We used three different types of values in the assessment of cancer risk and margin of exposure: (1) Cancer factors (Oral Slope factors; Inhalation Unit Risk factors; Drinking Water Unit Risk factors); (2) Reference concentrations (oral and inhalation) (RfC); and Minimum Risk Levels

Table 5. Total use of munitions carcinogens and release of by-products and metals onto Vieques 1947–1999.

Compound	Kg
TNT	8,094,242
RDX	907,025
HMX	5296
HBX-1	3415
Ethylene oxide	395,840
Gasoline	52238
2,4-dinitrotoluene	392
2,6-dinitrotoluene	0.490
1,3-butadiene	456
1,4-dichlorobenzene	0.351
Benzene	10007
Benzo(a)pyrene	532
Carbon tetrachloride	703
Naphthalene	16700
Vinylchloride	137
Arsenic	133
Beryllium	3.22
Cadmium	28000
Chromium VI	110
Cobalt	195
Lead	33,400
Mercury	3.34
Nickel	1900
Selenium	164
Strontium 90	2080
Titanium dioxide	28800
Vanadium pentoxide	1420
Aluminium powder (Chaff)	162,200
Depleted uranium	39

(MRLs) (oral and inhalation) (MRL). We did this is to provide the most conservative assessments. The relative toxicity estimations vary between these data types. We calculated the MoE for each compound by:

$$\text{MoE} = \frac{RfC \text{ or MRL}}{\text{Dose}_{i,j}} \qquad (9)$$

where $\text{Dose}_{i,j}$ is the exposure, and the RfC or MRL is the acceptable toxicity limit.

The MoE expresses the margin of relative safety for non-cancer diseases – the higher the MoE, the lower the risk of the exposure causing health impacts to the population. Typically, a MoE greater than 100 is accepted (US EPA, 2013).

Cancer risk is calculated as in Equation (10) below:

$$\text{LADD} \times \text{cancer factor} = 1/x \qquad (10)$$

where LADD = Lifetime average daily dose (mg/kg body weight/d = Dose i,j); cancer factor = Oral Slope factors; Inhalation Unit Risk factors; or Drinking Water Unit Risk factors; $1/x$ = one in x persons will develop cancer due to this exposure.

The cancer risk is determined by multiplying the exposure by the cancer slope factor for each compound. The result is hence x additional cancer cases due to the exposure. Typically, one extra case per one million persons is accepted (US EPA, 2013).

Cancer incidence rates

The cancer data were collected per request to the Puerto Rican Cancer Registry (PR CR, 2016). The quality of the PR-CR registry is high with an A2 > 75% rating by the WHO IARC. Age-specific incidence rates for all cancer sites by age group and sex were recorded, Puerto Rico and Vieques 1987–2011, to allow comparative analysis. The Incidence Case File of Puerto Rico from the Puerto Rico Central Cancer Registry (8 July 2014) was used for the analyses. The Population Source was Vintage 2012 estimates series from the Population Division of the United States Census Bureau to allow the calculation of the cancer rates by the PR-CR. Basal and squamous cell carcinomas of the skin, except when these occur on the skin of the genital organs, and *in situ* cancers except urinary bladder were excluded. Counts of cancer types < 20 in Vieques are too few to calculate a stable age-adjusted rate and are therefore not compared with Puerto Rico. It is a priori known that a key determinant of cancer incidence is age. The risk of cancer increases exponentially with increasing age. Hence, to compare the incidence of cancer over time and between populations, the summary incidence rates therefore need to be independent of age. The use of the standard population adjustment allows international comparison and evaluation of changes in incidence by comparing them to previous rates – the objective of age standardisation is essential to establish rates for comparison purposes. Rates in this analysis are adjusted to per 100,000 age-adjusted to the World Standard Population and adopted by the WHO (Segi, 1960). Trends were calculated with confidence intervals of 95% for the cancer incidence rates with Tiwari, Clegg, and Zou (2006) modification. The Puerto Rican Cancer Registry has high-quality data going back to 1987 – before that the data are less reliable due the technological development in diagnosis; moreover, data prior to 1987 are not electronically accessible and therefore not included in the direct comparative analysis. The overall cancer incidence rates for all types of cancer were moreover compared with the US rates and the Caribbean island Martinique rates from the IARC GLOBOCAN database (2012). The US data were included to give context to the rates, and Martinique was included because this cancer registry has the same quality as the Puerto Rican registry, and therefore is the most comparable Caribbean island in terms of cancer rates to Puerto Rico and Vieques. Lastly, the overall cancer rate for Vieques is compared to that of all the municipalities of Puerto Rico (2008–2012).

Results

Munitions loading

The point of departure for the assessment is the amounts of carcinogens dropped and fired on to the testing area of
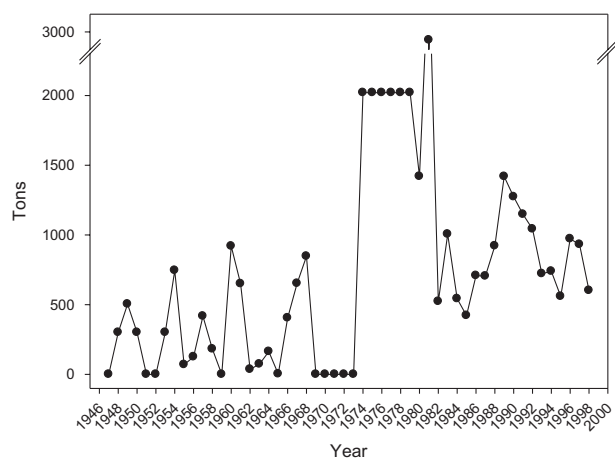
Figure 5. Total mass of carcinogens from munitions per year used on Vieques.

Figure 6. Use statistics of all explosives (kg/year) on LIA, SIA and EMA, respectively.

Vieques from all sources from the beginning of the testing in 1947 to the end in 1998. Figure 5 shows the total mass (fillers + casings and projectiles) of carcinogens over time. It is clear that the amounts were the highest from 1974 to 1982, with 1981 as the year with the highest loading at almost 3 million kg.

Table 5 shows the total use of the different identified munitions-specific carcinogens and total release of by-products and metals onto Vieques 1947–1999 . It is clear that TNT with > 8 mill kg and RDX with almost 1 mill kg used over the entire period were the most used materials.

Figure 6 illustrates where the loading mainly took place. It is interesting that for the first three decades from 1947 to 1973 the SIA location received the most loading and in the second period from 1973 to 1998 it was mainly the LIA area that was used for testing live munitions with carcinogenic fillers because the LIA is further away from the residential area than the SIA.

1981 was the worst-case year for TNT, ethylene oxide and depleted uranium, whereas gasoline use peaks in 1954. Organic explosion by-products and metals from casings and crater ejecta are emitted proportional to the use amounts and therefore also peak in 1981. As a worst-case assumption, we assumed that the particulate matter would remain airborne until reaching the residential receptor areas. Deposition of particles with adsorbed pollutants will be continuous and modelled on an annual scale.

Human exposure pathways

When applying the pathway models (Equation (1)–(8)) in accordance with the conceptual model (Figure 5), we were able to assess the annual mean pollutant concentrations in air, deposition to soil and concentration in top soil for the two receptor residential areas, or cities, Isabel Segunda
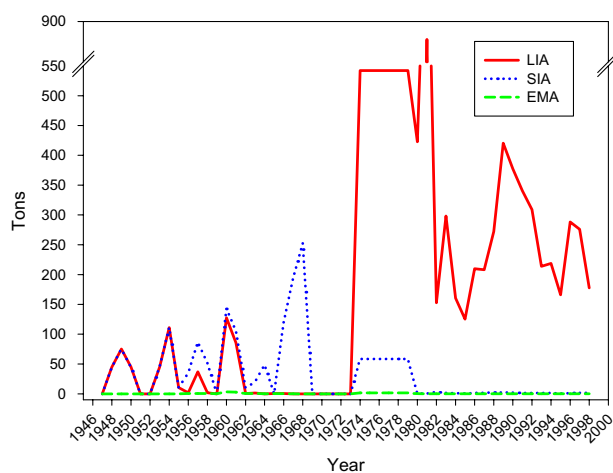
on the northern shore and Esperanza on the southern shore. Furthermore, the fraction of deposited pollutant that is leached to lower soil layers, which are potential drinking water sources, surface run-off to sea, pollutant concentration in sea water at local distances < 1 km from shore, concentrations in fish and top predators (sharks, capitan), concentrations in crop leaf and root tissue and concentrations in cattle meat and milk for the city with the highest concentrations (Isabel Segunda) were calculated. Finally, the annual average human dose of pollutant for each pathway and the sum of doses for all pathways were found. The resulting lifetime daily dose (LADD) was calculated as the 52-year average during the military activities on the island. Full tables can be found in the Supplementary Information Tables S1–S4, and in Tables 6 and 7 with the main results shown below.

We found that the highest concentrations of all pollutants in air, and consequently in all other media, occur at Isabel Segunda on the northern shore. The concentrations are however only a factor of 1.2 higher than Esperanza on the southern shore, and considering the uncertainties from worst-case assumptions, the pollutant levels in the environment can be considered approximately equal in the two cities. In Figure S1, the modelled annual mean air concentration profiles of TNT using annual mean munitions data for the entire period of testing (1947–1998) are shown using meteorology data for worst-case (maximum) concentrations at Isabel Segunda and Esperanze, respectively.

1981 was the year with the highest military activity; hence, this can be viewed as the worst-case year in terms of exposure and human intake of munitions-related carcinogens. TNT was the most used filler in 1981 at 664,900 kg (8.2% of the total TNT use for the entire period 1947–1998), and is therefore also the largest contributor to the total intake of carcinogenic filler via produce.

Table 6. Exposure and risk of munitions fillers.

Compound	CAS #	Maximum exposure pathway (oral) and total average daily dose (1947–1998) mg/kg/d (LADD)	Maximum exposure pathway (inhalation) and total average daily dose (1947–1998) mg/kg/d (LADD)	RfD Oral, mg/kg/d	RfC Inhalation, mg/m^3	Cancer class	Cancer site	Minimum MoE and 1/n person cancer risk
TNT[a]	118-96-7	Leaf (incl. fruits and cereals) 3.7E-5	Air and particles 8.0E-7	5E-4 OSF =3E-2 DW = 9E-10	NA	3	Bladder	**Oral 13** *Oral risk 1:1E6* *DW risk 1:3E14*
RDX[a]	121-82-4	Leaf (incl. fruits and cereals) 5.2E-5	Air and particles 9.1E-8	3E-3 OSF = 1.1E-1 DW = 3.1E-9	NA	3	Liver	**Oral 59** *Oral risk 1:5.7E6* *DW risk 1:1E13*
Ethylene oxide	75-21-8	Drinking water 2.1E-7	Air and particles 3.7E-8	NA	9E-2*	1	Leukemia	Inha 2.4E6
Gasoline	86290-81-5 & 8006-61-9	Root (vegetables and crops) 4.5E-7	Air and particles 5.5E-9	NA	7.1E-2	2B	Lung	Inha 1.3E7
Aluminium powder	7429-90-5	Drinking water 2.3E-5	Air and particles 1.5E-6	1*	10*	1**	NA	Oral 4.4E4 Inha 6.7E6
Depleted uranium	7440-61-1	Milk 8.2E-8	Air and particles 3.7E-11	2E-4*	4E-5*	3	NA	Oral 2.4E4 Inha 1E8

*Minimal risk level (MRL) ATSDR (2015); **Production; OSF = Oral Slope Factor (cancer); IUR = Inhalation Unit Risk (cancer); DW = Drinking water Unit Risk (cancer); aIncludes contribution from HBX-1, which comprises approx. 38% TNT and 40% RDX. Italic risks are added cancer risk in the population. The bold indicates that acceptable risks were exceeded.

Table 7. By-products from explosions and metal from explosives, crater ejecta and casings.

Compound	CAS #	Max exposure pathway (oral) and total average daily dose (1947–1998) mg/kg/d (LADD)	Max exposure pathway (inhalation) and total average daily dose (1947–1998) mg/kg/d (LADD)	RfD Oral, mg/kg/d	RfC Inhalation, mg/m³	Cancer class	Cancer site	Minimum MoE and 1/n person cancer risk
2,4-dinitrotoluene	121–14-2	Leaf (incl. fruit and cereals) 1.4E-9	Air and particles 3.8E-11	2E-3 OSF = 6.6E-1	1.5	2B	Bladder	Oral 1.4E6 Inhal 3.9E10 *Oral 1:9E10*
2,6-dinitrotoluene	606–20-2	Leaf (incl. fruit and cereals) 7.4E-11	Air and particles 4.8E-12	6.8E-1 OSF = 6.6E-1	1.5	2B	Bladder	Oral 9.2E9 Inhal 3E11 *Oral 1:5E11*
1,3-butadiene	106–99-0	Drinking water 6.2E-10	Air and particles 4.5E-11	NA	2E-3 IUR = 3E-8	1	Leukemia	Inhal 4.4E7 *Inhal 1:1E18*
1,4-dichlorobenzene	106–46-7	Root (vegetables and crops) 8.8E-10	Air and particles 3.4E-12	2.5E-3	8E-1	2B	Liver	Oral 2.8E6 Inhal 2.4E11
Benzene	71–43-2	Drinking water 5.4E-8	Air and particles 1.1E-8	4E-3 OSF = 1.5E-2	3E-2 IUR = 2.2E-9	1	Leukemia	Inha 2.7E6 *Oral 1:8E10* *Inhal 1:2E17*
Benzo(a)pyrene	50–32-8	Milk 8.2E-6	Air and particles 5.2E-11	NA OSF = 7.3 DW = 2.1E-4	NA	1	Stomach	**Oral 1:6E5** *DW 1:1.7E9*
Carbon tetrachloride	56–23-5	Root (vegetables and crops)4.5E-9	Air and particles 6.9E-11	1.4E-4 OSF = 7E-2	1.7E-3 IUR = 6E-9	2B	Liver	Oral 3E4 Inhal 2E7 *Oral 1:3E11* *Inhal 1:4E19*
Naphtalene	91–20-3	Root (vegetables and crops)4.6E-8	Air and particles 1.6E-9	2E-2	3E-3	2B	NA	Oral 4E5 Inhal 2E6
Vinylchloride	75–01-4	Drinking water 4.1E-7	Air and particles 2.7E-8	3E-3 OSF = 7.2E-1	1E-1 IUR = 4.4E-9	1	Liver	Inhal 5E11 *Oral 1:5E11* *1:5E11* *Inhal 1:6E20*
Arsenic	7440–38-2	Drinking water 3.7E-9 7.0E-11	Air and particles 1.3E-11 1.3E-10	3E-4 OSF = 1.5	NA IUR = 4.3E-6	1	Lung	Oral 8E4 *Oral 1:5E9* *Inhal 1:5E16*
Beryllium	7440–41-7	Drinking water 4.7E-11	Air and particles 3.1E-12	2E-3	2E-5 IUR = 2.4E-6	1	Lung	Oral 4E7 Inhal 6E6 *Inhal 1:7E18*
Cadmium	7440–43-9	Drinking water 4.1E-7	Air and particles 2.7E-8	5E-4	6E-4 IUR = 1.8E-6	1	Lung	Oral 1.2E3 Inhal 2.3E4 *Inhal 1:5E15*
Chromium VI	18540–29-9	Drinking water 1.6E-9	Air and particles 1.1E-10	5E-6*	8E-6 IUR = 1.2E-5	1	Lung	Oral 3.1E3 Inhal 7E4 *Inhal 1:1E15*
Cobalt	7440–48-4	Drinking water 2.9E-9	Air and particles 1.9E-10	3E-2 1E-2*	3E-5*	2B	Lung	Oral 3E6 Inhal 1E5

Compound	CAS	Pathway / concentration						
Lead	7439-92-1	Drinking water 7.1E-7	Air and particles 3.2E-8	NA	NA	2B	NA	NA
Mercury	7439-97-6	Fish 1.3E-9	Air and particles 3.2E-12	NA	3E-4	3	NA	Oral 9E7
Nickel	7440-02-0	Drinking water 5.1E-8	Air and particles 3.4E-9	NA	5.9E-5 IUR = 2.4E-7	2B	Lung	Inhal 1.7E4 / *Inhal 1:8E16*
Selenium	7782-49-2	Drinking water 2.4E-10	Air and particles 1.6E-11	5E-3	NA	3	NA	Oral 2E7
Strontium 90	10098-97-2	Drinking water 2.0E-9	Air and particles 2.0E-9	2*	NA	1	NA	Oral 6E7
Titanium dioxide	13463-67-7	Drinking water 4.5E-7	Air and particles 2.9E-8	NA	NA	2B	NA	NA
Vanadium pentoxide	1314-62-1	Drinking water 2.1E-8	Air and particles 1.4E-9	9E-2	1E-4*	2B	NA	Oral 4E6 / Inhal 1E13

*Minimal risk level (MRL) ATSDR (2015). Italic risks are added cancer risk in the population.

In summary, the carcinogenic explosives fillers, *i.e.* TNT, ethylene oxide and gasoline, represent 72.5, 3.5 and 0.47% of the total amount of explosive fillers used in the 1947–1998 period, respectively. Their use peaked in 1981 with the exception of gasoline, which peaked in 1954. The munitions filler concentrations are highest for Al powder and TNT in air, Al powder in soil, plant root, fish and predator, TNT and RDX in plant leaf, TNT, RDX and Al powder in sea water and Al powder and depleted uranium in meat and milk. The highest concentrations of chemical by-products from explosions occur for benzene in air and sea water, caused by a high emission factor from explosives. The highest leached fractions from top soil occur for carbon tetrachloride, B(a)P and 1,4-dicholorobenzene, caused by relatively low first-order degradation rates. In top soil, fish and predators, highest concentrations are for naphthalene due to a relatively high Koc value. In crop root, leaf, meat and milk, the highest concentrations occur for B(a)P due to a relatively high octanol–water (Kow) and high Kplant–water partitioning coefficients together with relatively high meat and milk biotransfer factors. Among the metals, cadmium, lead and titanium have highest concentrations in air, soil, sea water, crop leaf and root, caused by high emissions from explosives for cadmium and lead and high emissions from explosives and crater ejecta for titanium. In fish and predators, cadmium, mercury and lead have relatively highest concentrations due to high BCF values. Lead has the highest concentrations in meat and milk because of relatively high concentrations in air and soil, which the livestock are exposed to. In the drinking water scenario, groundwater from leaching of top soil pore water accounts for an approx. 65% increase in the drinking water concentrations of the metals compared to the contribution from deposited pollutant from the atmosphere alone. For TNT, ethylene oxide, benzene and vinylchlorid, >90% of the pollutant in drinking water originates from rainwater. For the other pollutants, >60% of the pollutant concentration in drinking water is from groundwater. In 1981, the year with maximum use of explosive fillers, the average annual concentrations in all media are approximately a factor of four higher than the annual average pollutant concentrations for the 1947–1998 period. See Supplementary Information for complete data.

All fillers are aggregated for the 1981 analysis and the total filler doses in 1981 for the different pathways were: Oral; Max exposure pathway is leaf (incl. fruit and cereals), annual average daily dose = 0.46 µg/kg/d. Inhalation; Max exposure pathway is air and particles, annual average daily dose = 0.0099 µg/kg/d. Daily doses for 1981 are a factor of 3.6 higher than the average daily 1947–1999 doses. Twenty-nine per cent of the total annual human dose of munitions-specific carcinogens is TNT. Here off, 88% of TNT exposure is from ingestion of crop leaf, including

fruit and cereals, and 10% is from drinking water. RDX constitutes 37% of the total annual dose, where 95% is oral via ingestion of vegetables. Al powder and B(a)P constitute 22 and 10% of the total annual dose, respectively, where Al powder is mainly via drinking water and B(a)P is via milk. See Figure S1 for a graphical presentation of the relative annual average human doses.

Cancer risk and MoE

As evident from the Tables 5 and 6, the primary compounds of concern from a cancer risk perspective are B(a)P with a cancer risk of 1:600,000 and TNT at 1: 1,100,000. For both of these, oral exposures were the most relevant route of exposure. (US EPA, 2013). From a non-cancer health point of departure, the MoE for TNT and RDX are 13 and 59, respectively, which suggest a potential risk, which should be reviewed. As with the cancer risk, the health risks are a factor of four greater for 1981 than the average risk resulting in MoEs of approximately 3 and 15, for TNT and RDX, respectively. A MoE greater than 100 is normally presumed safe if the toxicological and exposure data behind are comprehensive and of high quality (US EPA, 2013). In this study, the toxicity data are of high quality and assumed conservative as they are oral reference concentrations from an IRIS review process. The exposure data are however a result of conservative modelling efforts and could be supplemented with additional refined modelling and measurements. Non-cancer and cancer risks from all the other compounds are found in Tables 6 and 7 and are lower than the values of B(a)P, TNT and RDX, and are therefore at this point in time of lower public health concern via the exposure routes described in this paper. Below is a brief summary of the toxicological profile of TNT and RDX. B(a)P is not included as this is mainly a carcinogen.

TNT: Workers involved in the production of explosives who were exposed to high concentrations of 2,4,6-trinitrotoluene (TNT) in workplace experienced several harmful health effects, including anaemia and abnormal liver function. Similar blood and liver effects, as well as spleen enlargement and other harmful effects on the immune system, have been observed in animals that ate or breathed 2,4,6-trinitrotoluene. Other effects in humans include skin irritation after prolonged skin contact, and cataract development after long-term (365 days or longer) exposure. It is not known whether 2,4,6-trinitrotoluene can cause birth defects in humans. However, male animals treated with high doses of 2,4,6-trinitrotoluene have developed serious reproductive system effects. Toxic hepatitis, aplastic anaemia, methemoglobinemia, hemolytic anemia and cataracts have been reported after occupational exposure. Hemolytic anaemia has been described in workers with a

genetic glucose-6-phosphate dehydrogenase (G6PD) deficiency. Reduced haemoglobin values were seen with exposures as low as 0.48 mg/m³. Cataracts were induced in 6 of 12 workers exposed at 0.14–0.58 mg/m³ for 6.8 ± 4.7 years and in 7 of 9 workers exposed at 0.10–0.35 mg/m³ for 1–27 years (average of 14 years) https://www.atsdr.cdc.gov/toxfaqs/tf.asp?id=676&tid=125). TNT has the cancer classification C, possible human carcinogen. The basis for the classification is evidence of human carcinogenicity is inadequate. Urinary bladder papilloma and carcinoma were observed in female Fischer 344 rats. Mutagenic activity was observed in Salmonella with and without metabolic activation (https://toxnet.nlm.nih.gov/cgi-bin/sis/search2/f?./temp/~dffsUz:3).

RDX: RDX affects mainly the nervous system. Workers have experienced central nervous system effects including seizures after occupational exposure. It can cause seizures in humans and animals when large amounts are breathed in or ingested. Some people exposed to high amounts of RDX have had changes in blood pressure and in some parts of the blood. The effects of long-term exposure to low levels of RDX are not known. It is not known whether RDX affects reproduction in people. In sub-chronic feeding studies of dogs (50 mg/kg/day for 6 weeks), one of seven dogs died, and the others had weight loss and seizures. Animals in chronic studies show liver injury. RDX is classified as: Other CNS Neurotoxin and a Secondary Hepatotoxin (https://www.atsdr.cdc.gov/toxfaqs/tf.asp?id=411&tid=72). RDX cancer classification is C, possible human carcinogen; the basis for the classification is Hepatocellular adenomas and carcinomas in female B6C3F1 mice (https://toxnet.nlm.nih.gov/cgi-bin/sis/search2/f?./temp/~weQxE2:1).

Cancer rates

It is important to note the scale difference between Puerto Rico and Vieques in terms of total number of cancers as well as the difference in total population (approx. 10000 on Vieques and 3.7 mill on Puerto Rico). In the 25 years covered in this analysis, Puerto Rico had a total of 262.505 cancer cases (56% male and 44% female), while Vieques had 776 (58% male and 42% female). This makes the direct quantitative comparison difficult and in some cases impossible for other than large cancer types, large age groups and long periods, despite the standardisation, as the difference in sample size causes large variations in the Vieques rates. It is clear that the Vieques rate (total rate 1987–2011 = 220 ± 166 SD) is more variable than the rate for Puerto Rico (total rate 1987–2011 = 203 ± 16 SD), (the total rate standard variation is 10 times higher for Vieques relative to Puerto Rico), but also that the variation is decreasing over time and that the overall rate
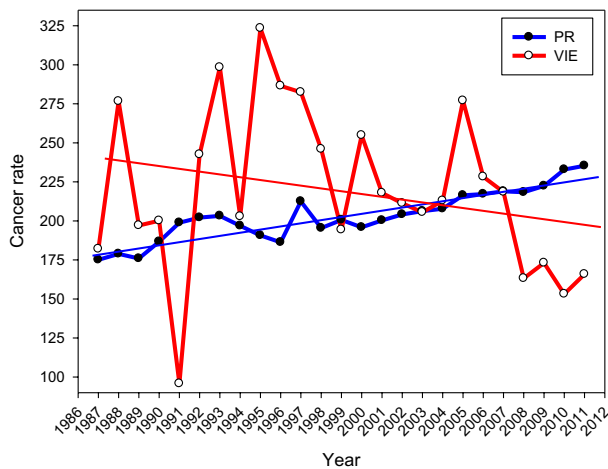
Figure 7. Overall age-adjusted cancer incidence rates and trend lines for Puerto Rico and Vieques.

Table 8. Overall cancer incidence rates in USA; Martinique (FR); Puerto Rico; and Vieques (GLOBOCAN, 2012).

Country/area	All sites, female	All sites, male
USA	284.6	363.4
Martinique	158.6	314.8
Puerto Rico	174.5	257.0
Vieques (2011)	157.5	178.7

until 2000 was higher in Vieques than in Puerto Rico and lower towards the end of the period following the trend lines (Figure 7).

To provide context to the rates, we have compared the overall cancer rates for U.S.A.; Martinique; Puerto Rico to Vieques in 2012 for females and males. It is clear that the rate was highest in USA, and that for the year 2012 the rate was lower in Vieques, which illustrates that all sites' rate in Vieques is lower than in the U.S. and, lower than for males, and on par for females, with regard to Martinique. In recent years, from 2008 to 2012, Vieques was among the 25thcentile of lowest cancer incidence rate among Puerto Rican municipalities PR CR (2016) (Table 8). However, the ratio between male and female rates is much lower in Vieques and since there is no biological reason for this difference, it suggests that the male rates are underreported.

Below, in Figure 8(a)–(c), all the major types of cancer and their distribution among women and men for three different overall age groups (<50; 50–64; >64 years) are normalised to Puerto Rico (red line = 100). There is of course as mentioned above great variation from year to year in the Vieques data due to the low numbers. However, there are two cancer types, which stand out relative to Puerto Rico in total, and for the most age groups: lung and bronchus; and colon and rectum. The most significant difference is for lung and bronchus cancer rates for women < 50 yr with a 280% higher rate in Vieques than in Puerto Rico, and for men 50–64 yr with a 200% higher rate than in Puerto Rico.

We use the five-year mean values for the overall and two major cancer types of concern where the Vieques data moreover have enough readings to allow a more data rich comparison. Figure 9(a)–(c) below illustrates that the in the period 1992–1997, the incidence rate was significantly higher in Vieques than Puerto Rico, and that the rate since

decreased – and in 2007–2012 the Vieques rate in total was significantly lower than Puerto Rico. The main cause of the elevated incidence rate in 1992–1997 was lung cancer and colon and rectum cancer. The statistically significant annual percent change (APC%) in incidence rates 1987–2012 for Puerto Rico and Vieques for total cancer, lung and bronchus; and colon and rectum, was: Total: 1.0 (PR) and −1.0 (VIE); lung and bronchus: −1.0% (PR) and NA (VIE); colon and rectum: 1.6% (PR) and 0.2 (VIE), indicating the trend in Figure 7.

We further investigated if any age groups had significantly higher incidence rates on Vieques compared to Puerto Rico, normalising the data to Puerto Rico (= 100). It is clear that the variation in age groups 0–19 years old is very varied due to very low numbers, very few cases (0–2 per year) make a big difference, e.g. girls 5–10 yrs had 0%, whereas girls 10–15 yrs had > 300% of the rate compared to Puerto Rico – making comparison difficult due to random effects. From 35 yrs and older, the total numbers increase and the statistical reliability in the comparison increases and the rates align with no statistically significant differences (Figure 10).

A more detailed picture of the direct cancer incidence rate for the total population, females and males, in Puerto Rico and Vieques is shown in the Figure 11 below. The overall trends are quite similar between the two locations, especially not in the centre of the age distribution where the numbers are greatest in Vieques.

Childhood cancer is a special concern as the incidence rates should be as low as possible – in U.S. in 2014 the annual rate for 0–19-year-old children was 18.6 per 100.000 (Ward, DeSantis, Robbins, Kohler, & Jemal, 2014). Figure 12 below highlights the trends for younger age groups, which follow the same trajectory and are within the normal level compared to the U.S. The rates among Viequenses are more variable than for Puerto Rico again due to low numbers. The results does not indicate a far greater than expected childhood cancer rate on Vieques and the differences are not statistically significant, nor in absolute numbers significantly different from the norm.

Discussion

Exposure assessment

The point of departure is that the training area on Vieques is a Superfund site and that the current conclusion
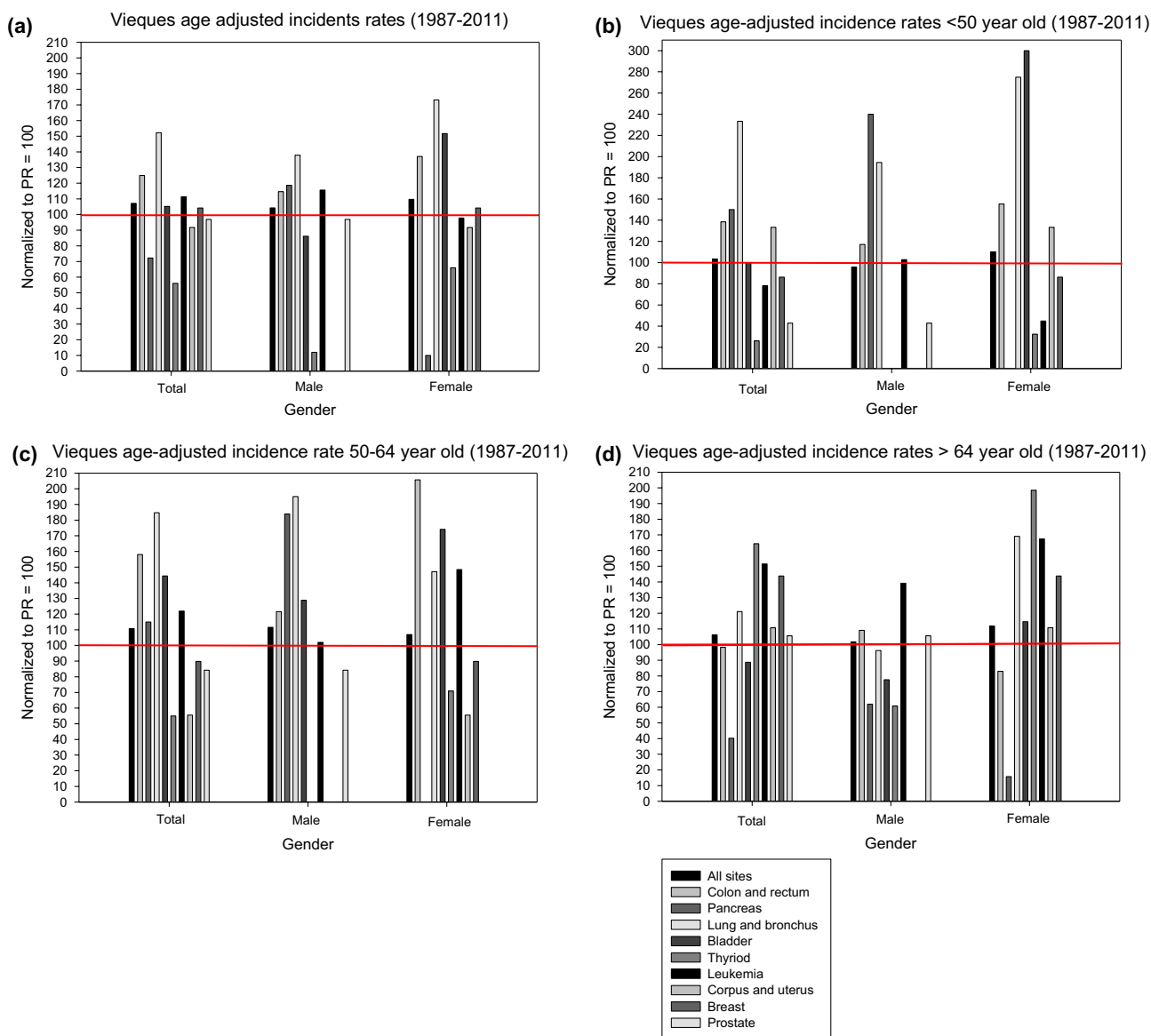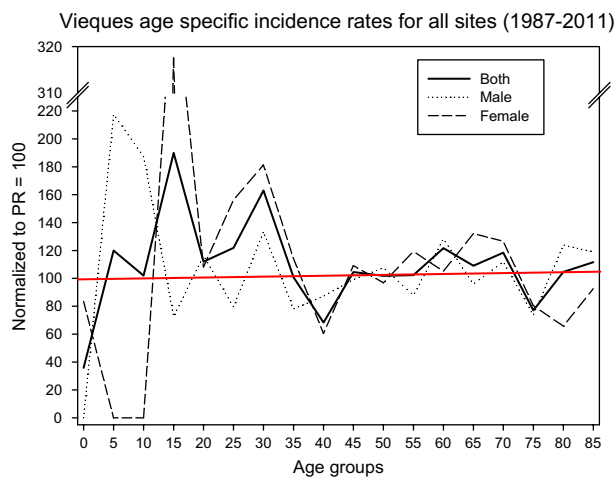
Figure 8(a–d). Mean age-adjusted cancer incidence rates for all major cancer types in total and split on three age classes.

regarding human health risks is that the exposure is not under control in such a way that: (1) contamination has been detected at a site at an unsafe level, and (2) a reasonable expectation exists that people may be exposed to the contamination. Evaluating the model results together with measurements that have been performed for local produce on Vieques, there is an indication that the explosive fillers concentrations in local produce need special attention as the measured data are very sparse and the modelled data suggest that this is a significant exposure pathway. The measurement campaigns that have taken place have focused on metals, and have been evaluated by ATSDR (2013) with the general conclusion that the overall data are insufficient to quantify adequately human exposure or draw any valid health conclusions whether consuming locally grown produce and livestock would

result in any harmful health effects. Specifically, there are indications of potentially critical concentrations of cadmium in pigeon peas, which stress the need to conduct further sampling (ATSDR, 2013). The conclusions and recommendations from this work are therefore in accordance with ATSDR (2013) that additional sampling of locally grown foods and milk is warranted. This includes metals but also specifically B(a)P, TNT and RDX. Sampling should represent the edible parts of leafy vegetables which accumulate pollutants more effectively, and milk for B(a)P. Surface soil samples should be taken at the same locations and time. Data from previous studies have been discarded to lacking of adequate high standards and quality assurance of sampling and chemical analysis; this must therefore be taken care of and included in the sampling campaigns.
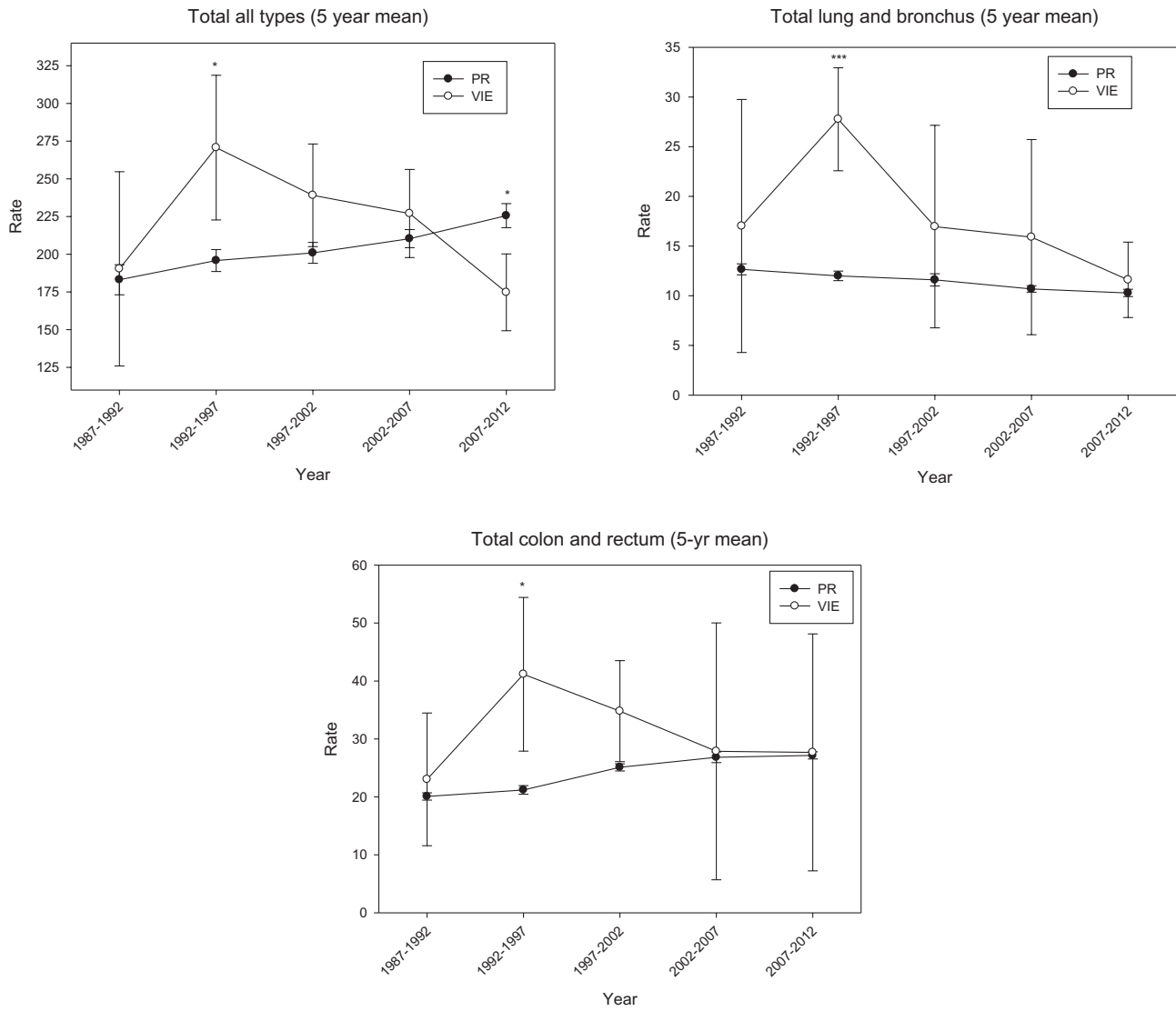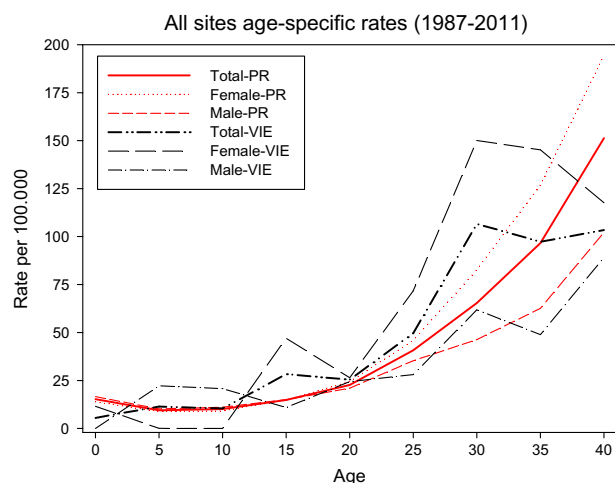
Figure 9 (a–c). Five-year mean cancer incidence rates for Puerto Rico (PR) and Vieques (VIE), 1987–2012.
Note: * = $p < 0.05$; *** = $p < 0.001$.

Figure 10. Relative overall cancer incidence rates for specific age groups per year normalised to Puerto Rico.
Note: Red line = 100.
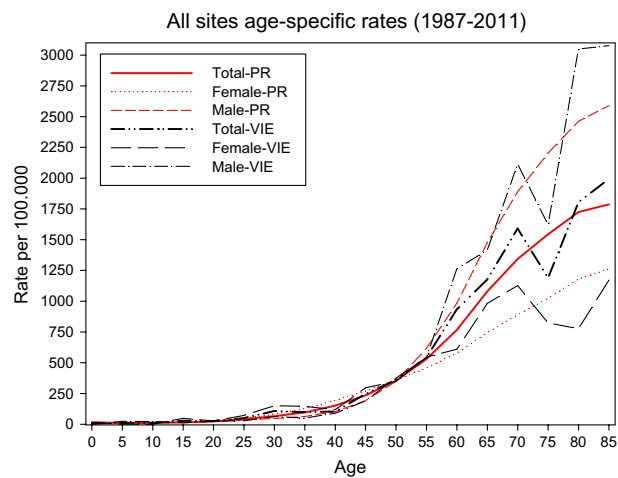
Figure 11. All site age-adjusted and age-specific absolute cancer rates for Puerto Rico and Vieques.

Figure 12. All site age-adjusted and age-specific absolute cancer rates for Puerto Rico and Vieques.
Note: 0–40 yrs.

Cancer risk

It is not possible based on the data to identify Viequenses, or sub-groups of Viequenses (e.g. children), as systematically having very high incidence rates compared to Puerto Rico or neighbouring areas in the study period (which is the only period with reliable data). There is one five-year period where the cancer risk is significantly elevated (1992–1997), where total lung and bronchus; colon and rectum; and total rates were statically significantly higher than in Puerto Rico (Figure 8(a)–(c)). However, the pattern is not consistent as it is the only period with significantly higher rates. Hence, following the Bradford-Hill (1965) criteria, this in not significant and not robust enough at this time to ascribe causality, but to warrant further investigation mainly among women < 50 yr and men 50–64 yr for these types of cancer. The potentially critical added cancer risk found in this study is due to B(a)P exposure via milk. Overall, the cancer incidence rate is lower on Vieques than in Puerto Rico for the period with a trend of APC of −1% vs.+1%, respectively, despite the fact that the portion of > 65 yrs old persons is 2.7% higher on Vieques and that the percentage smokers is 5–10% higher on Vieques. The overall cancer incidence rate on Vieques is in the 25thcentile lowest among the municipalities in Puerto Rico (2008–2012) and for men significantly lower than comparable countries, and on par for women in Puerto Rico and Martinique and lower than U.S.A., for 2011/2012 (Table 8). This assessment does not include non-accessible data on inter-municipal migration in Puerto Rico and Vieques as a potential cause of both under and overestimation of rates – i.e. exposed to carcinogens in one municipality and moved to another municipality and was diagnosed due to the exposure in the first municipality. It seems like at least the male reporting

is low as the ratio between male and female cancer is 1.1 vs. 1.47 in Puerto Rico.

The aetiology of cancer is multi-causal, complex and complicated by latency (the period between the exposure causing biological initiation of cancer to medical diagnosis). Figure 4 shows that 1981 was the year with highest use, and that the period 1974–1999 was the period with the highest use compared to the period from 1947 to 1973. Moreover, the usage in the earlier period was mostly gasoline, whereas TNT and RDX were more used in the second period. Latencies between exposure and diagnosis can range from months to decades. The amount of B(a)P tracks the amount of TNT and RDX as this is an explosion by-product. Historically, Nordling (1952) reported latencies of 9–40 years; more recently, Nadler and Zurbenko (2014) reported that 89% out of more than 1.6 million investigated cancer cases had a latency of more than 10 years. Following the 9/11 attack, the WTC Program Administration determined a minimum latency period of all cancer types (except mesothelioma, lymphoproliferative, thyroid and childhood cancers) of 4 years (Howard, 2013). Hence, the cancer registry starting in 1987 does cover the most critical usage period from 1983 to 1977 and onwards depending upon length of the latency period (minimum 4–10 years). The statistical latency period for lung and bronchus is 13.6 years (Nadler & Zurbenko, 2014); hence, the observed significant elevated level of this type of cancer in Vieques in 1992–1997 (Figure 7) could have been onset in 1979–1984. The lack of identified significant added cancer risk in the worst-case year of 1981 indicates that the exposures in the years before 1987 would not contribute to the overall cancer rate in the period 1947–1987 on Vieques.

Health effects

We calculated general health risks for the munitions-related compounds and derived MoEs (Table 6). MoEs greater than 100 indicate negligible risk for chronic exposures based on a No Observed Adverse Effect Level (NOAEL) or a MRL (US EPA, 2013). This suggests that TNT and RDX could be a potential risk to human health in the study period. Effects of TNT exposure include: anaemia and abnormal liver function; abnormal blood and liver effects; spleen enlargement; and harmful effects on the immune system, whereas RDX mainly affects the nervous system, see summary above. All other compounds had MoEs greater than 100 indicating low concern of risk. There is no central disease registry for Vieques where these diseases' frequency can be compared to background frequencies to determine if these are elevated in Vieques or how they may relate to munitions exposures.

Conclusions

We have demonstrated how a retrospective risk screening of carcinogens related to munitions training areas can be conducted in response to the challenges Phillips and Perry (2002) mentioned. This requires a truly multidisciplinary team approach to derive the munitions registry data and to translate this to public health risks; this provides a good guidance for further prioritisation of exposure pathways, compounds of concern and diseases and population groups of potential concern. Based on the munitions registry data and the conservative exposure modelling, we determined that there was a potential for elevated cancer risks with regard to B(a)P exposures in the 52 years of military activity. There was also a potential for concern of health risks from TNT and RDX exposures. No other exposures indicate a concern for risk. Both of these exposures' primary exposure route was oral via leafs, fruits and cereals. There is in general no significant difference in the cancer incidence rates between Vieques and Puerto Rico. However, the period 1992–1997 did show a significantly elevated lung and bronchus cancer incidence rate in Vieques compared to Puerto Rico, mainly among women <50 yr and men 50–64 yr, which could correlate with exposures in the period 1977–1984. These data are consistent with the assertion that military activity on Vieques was potentially contributory to public health risks based on these conservative assessments, warranting further measurements to be conclusive. The focus of this study was the direct carcinogenic risks and general health risk caused by emitted and transported carcinogens originating from the munitions used; however, both cancer, and other public health impacts and risks are multi-causal. Hence, the general stress impacts due to military training on Vieques on the public health of Viequenses would warrant a wider community-based public health and risk assessment with past, current and future potential epigenetic risks covering other diseases than cancer. In our view, the focus should be on a community-based epigenetic assessment to allow the assessment of the most relevant diseases and concerns to the citizens for the current and next generation. Since the vast majority of exposure form the munitions has decreased over time, epigenetic effects would be the most relevant future public health concern.

Geolocation information

Coordinates:18°07′N 65°25′W

Disclosure statement

No potential conflict of interest was reported by the authors.

Notes on contributors

Hans Sanderson, PhD, is a senior scientist at the Department for Environmental Science at Aarhus University, Denmark. He is also a member of the WHO Chemicals Risk Assessment Network. His research interests include environmental and public health of chemicals. He has conducted risk assessments of sea dumped chemical munitions in the Baltic Sea for more than a decade. He has moreover, recently conducted a public health cancer risk screening of background environmental chemicals and exposures for the government of Gibraltar. His research supports decision-making regarding chemical risk assessment as well as advances the science in this field.

Patrik Fauser, PhD, is a senior scientist at the Department for Environmental Science at Aarhus University, Denmark. His research interests include modelling environmental and public health exposures to chemicals. He contributed to risk assessments of sea dumped chemical munitions in the Baltic Sea for more than a decade in various projects. He has moreover, recently contributed to a public health cancer risk screening of background environmental chemicals and exposures for the government of Gibraltar. His research supports decision-making regarding chemical risk assessment as well as advances the exposure modelling science in this field.

Ricky Stauber is a retired Explosive Ordinances and Disposal (EOD) officer of the US military. He is now a private consultant. His research interests are in military history and the use and disposal of explosive ordinances. He is an expert after a full career in in the EOD field in the different weapon types and their composition and nomenclature.

Per Løfstrøm, PhD, is a senior advisor at the Department for Environmental Science at Aarhus University, Denmark. His research interests include modelling metrology boundary layer assessments of turbulence, deposition, concentration fluctuations and local scale dispersion modelling. He is responsible for the maintenance of the OML model for the Danish EPA.

Jesper Christensen, PhD, is a senior scientist at the Department for Environmental Science at Aarhus University, Denmark. His research interests include modelling atmospheric physics and the deposition, and concentration of chemicals in the environment with a special focus on human exposures. His research focuses on air quality in support of governmental policy developments.

Thomas Becker was a post-gradual fellow at the Department for Environmental Science at Aarhus University, Denmark. His research interests include GIS modelling of the distribution of atmospheric concentrations of particles and contaminants.

ORCID

Hans Sanderson http://orcid.org/0000-0002-6735-2444
Jesper Christensen http://orcid.org/0000-0002-6741-5839

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