Environmental Research 180 (2020) 108816

Contents lists available at ScienceDirect

Environmental Research

journal homepage: www.elsevier.com/locate/envres

One-Decade-Spanning transgenerational effects of historic radiation dose in wild populations of bank voles exposed to radioactive contamination following the chernobyl nuclear disaster

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ARTICLE INFO

Keywords:
Chernobyl
Radiation exposure
Radiotoxicity
Low-dose radiation
Bank vole
Embryonic lethality
Cytogenetic aberrations
Dose reconstruction
LNT model
Radiobiology
Historic dose
Genomic instability
Transgenerational effects

ABSTRACT

The concept of historic radiation doses associated with accidental radioactive releases and their role in leading to radiation-induced non-targeted effects on affected wild animals are currently being evaluated. Previous research studying Fukushima butterfly, Chernobyl bird and fruit fly populations shows that the effects are transgenerational, underlined by the principles of genomic instability, and varied from one species to another. To further expand on the responses of and their sensitivity in different taxonomically distinct groups, the present study sought to reconstruct historic radiation doses and delineate their effects on bank voles (*Clethrionomys glareolus*) found within a 400-km radius of the Chernobyl Nuclear Power Plant meltdown site. Historic dose reconstruction from the whole-body dose rates for the bank vole samples for their parental generation at the time of radioactive release was performed. Relationships between the historic doses and cytogenetic aberrations and embryonic lethality were examined via graphical presentations. Results suggest that genomic instability develops at the historic dose range of 20–51 mGy while a radioadaptive response develops at the historic dose range of 51–356 mGy. The Linear No-Threshold (LNT) relationship was absent at historic doses of lower than 356 mGy at all generations. However, LNT was apparent when the very high historic dose of 10.28 Gy in one sampling year was factored into the dose response curve for the bank vole generation 21–22. It is worth being reminded that natural mutation accumulation and other environmental stressors outside the realm of dose effects could contribute to the observed effects in a multiple-stressor environment. Nevertheless, the consistent development of genomic instability and radio-adaptive response across generations and sampling sites unearths the utmost fundamental radiobiological principle of transgenerational non-targeted effects. As a result, it calls for better attention and regulation from global governing bodies of environmental health protection.

1. Introduction

Chernobyl was the place of the largest radioactive release in history with total radioactive release reaching magnitudes over 10^{15} Bq (Steinhauser et al., 2014). Although this event occurred over 30 years ago, its effects are still being seen today. High levels of embryonic loss along with physical and chromosomal abnormalities have been seen in many non-human biota. The frequency of these phenomena is not what might be expected for the radiation doses found in the habitats of these organisms and do not follow the extrapolation of high dose effects (Garnier-Laplace et al., 2013; Goncharova and Smolich, 2002). Specifically, fruit fly and bird populations in Chernobyl have been observed to have consistently high rates of chromosomal aberrations and physical abnormalities (Omar-Nazir et al., 2018; Hancock et al., 2019; Galván et al., 2011; Bonisoli-Alquati et al., 2010). Similarly, after the radioactive release caused by the accident at the Fukushima Dai-ichi Nuclear Power Plant, increasing levels of physical aberrations and embryonic loss were found in pale grass blue butterflies around Fukushima (Hancock et al., 2018). Additionally, Goncharova team has revealed that for bank voles in sites contaminated by the Chernobyl fallout, there is a strong dependence of chromosome aberration frequency, embryonic mortality levels and Micronucleated Polychromatic Erythrocytes (MNPCEs) on whole body dose and dose rates (Goncharova and Smolich, 2002; Ryabokon et al., 2000, 2005;

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https://doi.org/10.1016/j.envres.2019.108816
Received 7 August 2019; Received in revised form 8 October 2019; Accepted 8 October 2019
Available online 09 October 2019
0013-9351/

Ryabokon and Goncharova, 2006a).

This variance from the expected dose effects from high dose extrapolation have been linked to a number of theories. While several research groups posit that the stress and other factors are the causes, others point to poor dose estimation techniques (Garnier-Laplace et al., 2013; Liess and Beketov, 2011; Klok and Kraak, 2008). Based on previous observations with irradiated human and animal cells in a laboratory setting (Seymour et al., 1986; Vo et al., 2017, 2019; O'Reilly et al., 1994; Cohen et al., 2019), our group argues that the increased frequency of these effects could be due to origination of genomic instability. What this implies in the context of radioecology is that the non-irradiated descendent offspring of initially irradiated progenitor survivors carry a higher load of mutations, that would result in either a higher lethality rate or more morphological abnormalities (Kadhim et al., 2004; LITTLE, 1998; Aghajanyan et al., 2011). This has been tested on native birds from Chernobyl and local butterflies from Fukushima (Omar-Nazir et al., 2018; Hancock et al., 2018). There is a strong positive correlation between the dose the progenitor organisms receive and the level of aberrations shown in the offspring (Goncharova and Smolich, 2002; Bonisoli-Alquati et al., 2010).

Other dose effects such as radioadaptive response (RAR) can occur in descendent generations. The RAR occurs when the initial exposure to low-dose radiation activates protective mechanisms that prevent the progression of harmful radiation effects caused by subsequent doses (Hancock et al., 2019; Vo et al., 2017; Cohen et al., 2019; Kadhim et al., 2004; Zainullin et al., 1992; Farooqi and Kesavan, 1993).

This paper hypothesises that genomic instability caused by historic radiation dose in bank voles (*Clethrionomys glareolus*) within a 400-km radius of the Chernobyl Nuclear Power Plant (ChNPP) meltdown disaster site could possibly be the driving mechanism leading to the observed elevated levels of chromosomal and chromatid aberrations and embryonic mortality. Owing to its short lifespan, short generation time and non-migratory lifestyle, the bank vole was a suitable candidate for this study (Ryabokon and Goncharova, 2006a; Ostfeld, 1985; Ryabokon et al., 2003). Chromosomal and chromatid aberrations are all part of possible cytogenetic damages caused by genomic instability (Hancock et al., 2018; Fenech, 2002; Celeste et al., 2002) and are known, in turn, to cause embryonic mortality and foetal death (Hancock et al., 2019). To test this theory, historic radiation doses associated with the ChNPP meltdown disaster were calculated and relationships between dose and cytogenetic aberrations and embryonic mortality in similar descendent generations at different contamination sites post-ChNPP accident were compared.

2. Methods

Ryabokon and Goncharova supplied the data on the mean percentage embryonic lethality and mean percentage cellular aberrations in populations of bank voles (*Clethrionomys glareolus*) in Belarus within a 400-km radius of the Chernobyl Nuclear Reactor. Within the 400-km radius, 5 sites with varying distances were the focus of their research with measured dose rates for some of the years between 1986 and 1996 (Ryabokon and Goncharova, 2006a). For details on methodology on the surveying methods, methods of capture, determination of cellular aberrations and summary of findings, we refer to Ryabokon and Goncharova (2006) (Ryabokon et al., 2005).

The release from the Chernobyl Nuclear Reactor contained many different radionuclides (Steinhauser et al., 2014; Drozdovitch et al., 2013). Gamma spectrometry, alpha spectrometry and other methods were used to determine the presence of certain radionuclides in collected soil samples (Ryabokon et al., 2005; Ryabokon and Goncharova, 2006a). Cs-137, Cs-134, Ru-106, Ce-144, Sr-90, Pu-238, Pu-239 and Pu-240, Pu-241 and Am-241 were the primary radionuclides found in the samples (Ryabokon et al., 2005; Ryabokon and Goncharova, 2006a). For whole-body absorbed dose rate estimations, Ryabokon and Goncharova used the absorbed fraction model and the local absorption method for gamma, and alpha and beta emitters respectively.

In order to obtain a historical dose for the parental generation of bank voles at the time of ChNPP accident, the estimated dose rates for each site were corrected for 1986 using the equation below.

$$\dot{D} = \dot{D}_0 e^{-\lambda t} \quad (1)$$

t signifies the time of decay, λ signifies the decay constant, $\dot{D}_0$ signifies the dose rate at the beginning of the decay while $\dot{D}$ signifies the dose rate at the end of the decay time.

The absorbed doses came from a number of radionuclides. Although the accident released various radionuclides, Cs-137, Cs-134, Ce-144 and Ru-106 were the primary radionuclides taken into account due to their significant soil contamination levels. In order for decay calculations to be performed, a set decay constant is needed which is usually derived from the half-life of the investigated radionuclide. Since many radionuclides were involved with primarily large half-lives, a decay constant was estimated by comparing the dose rates of sites 1,3 and 4 from 1986 to 1988 following equation (1) to obtain the estimated decay constant for each site. An average of the decay constants was used.

From the estimated decay constant, the cumulative dose over the lifespan of the progenitor bank vole populations in all 5 sites were found. Since bankvoles have a generational time of 4–6 months and an average lifespan of 2 years (Ryabokon and Goncharova, 2006a; Ostfeld, 1985; Ryabokon et al., 2003), the cumulative dose was calculated over a 2-year span.

The doses were then applied to the mean cytogenetic aberrations and mean embryonic mortality frequencies of each generation for each site.

The reconstructed doses were then compared to the mean cytogenetic aberration and embryonic mortality frequency of cells that have either chromosomal or chromatid aberrations. The cytogenetic aberration analysis was performed on bone marrow cells of captured animals from all five sites. Embryonic mortality was determined for only female rodents that could be captured at sites 2, 3 and 4 and accessed between 7 and 22 days of pregnancy (Ryabokon and Goncharova, 2006a; Mothersill et al., 2017.). Detailed information on the methods of determining cytogenetic aberration and embryonic lethality frequency was discussed in Ryabokon and Goncharova (2006) (Ryabokon and Goncharova, 2006a) and Ryabokon et al. (2000) (Ryabokon et al., 2000).

A GraphPad Prism 6 software (GraphPad, San Diego, CA, USA) was used for statistical analysis. Pearson correlation coefficients and their associated P-values were calculated to determine a possible correlation between the reconstructed historic radiation dose and cytogenetic aberration/embryonic lethality.

3. Results

The reconstructed historic radiation dose at sites 1, 2, 3, 4, and 5 were determined to be 3.80 ± 0.08, 20 ± 2, 51 ± 3, 356 ± 4, and 10280 ± 200 mGy, respectively (Table 1). The further the site is away from the ChNPP meltdown location, the smaller the dose is (Table 1). This follows the classic dose-distance relationship.

Fig. 1 shows the relationship between the historic whole-body doses (3.8–356 mGy) and the frequency of cytogenetic aberrations in sites 1, 3 and 4 for generations 1–2 and 5–6. No correlation is seen between the historical doses and cytogenetic aberrations for generation 1 (R (Garnier-Laplace et al., 2013) = 0.01; p = 0.9404) and generation 5 (R (Garnier-Laplace et al., 2013) = 0.6; p = 0.4343). Fig. 2 shows the relationship between the historic whole-body doses (20–356 mGy) and the frequency of cytogenetic aberrations in sites 2, 3 and 4 for generations 11–12 and 21–22. No correlation is also seen between the historic dose and cytogenetic aberrations seen for generation 11–12 (R^2 = 0.004; p = 0.9560) and generation 21–22 (R^2 = 0.4; p = 0.5541). However, there were clearly dose-dependent increases in the levels of cytogenetic aberrations at very low doses (from 3.8 mGy to

Table 1
Reconstructed historic doses and cytogenetic aberration frequency in bank voles at five sites within a 400-km radius of the Chernobyl nuclear power plant meltdown accident. Generations along with their year of capture are listed. Errors reflect the standard deviation of the range of aberrant cell frequencies for the site.

Year	Reconstructed Historic Dose (mGy)	Animal generation since the accident	Aberrant Cells (%+SD)
Site 1			
1986	3.80 ± 0.08	1–2	0.40 ± 0.22
1988	–	5–6	0.64 ± 0.32
Site 2			
1983	4.0 ± 0.3	Pre-event	0.41 ± 0.12
1986	20 ± 2	1–2	–
1991	–	11–12	1.22 ± 0.37
1992	–	13–14	1.09 ± 0.24
1996	–	21–22	1.17 ± 0.53
Site 3			
1986	51 ± 3	1–2	1.99 ± 0.53
1988	–	5–6	1.51 ± 0.03
1991	–	11–12	2.49 ± 0.76
1996	–	21–22	1.95 ± 0.38
Site 4			
1986	356 ± 4	1–2	1.21 ± 0.10
1987	–	3–4	1.10 ± 0.22
1988	–	5–6	1.86 ± 0.04
1991	–	11–12	1.84 ± 0.36
1996	–	21–22	2.04 ± 0.78
Site 5			
1996	10280 ± 200	21–22	5.10 ± 0.78

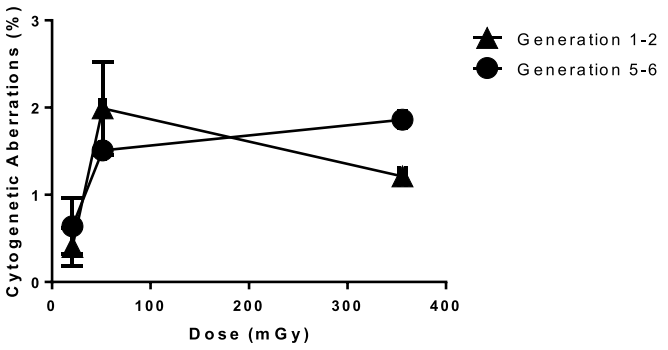

Fig. 1. Relationship between reconstructed historic whole-body radiation doses at sites surrounding the ChNPP accident location and mean cytogenetic aberration rates for bank voles of generations 1–2 and 5–6. Aberrant cell frequencies from three sites (sites 1, 3 and 4) within a 400-km radius of the ChNPP were plotted against the reconstructed historic whole-body radiation doses for those sites. The p value for generation 1–2 is 0.9404 and for generation 5–6 is 0.4343. Error bars reflect the standard deviation of the range of aberrant cell frequencies for the site.

51 mGy in early descendent generations 1–2 and 5–6, Fig. 1; from 20 mGy to 51 mGy in much later descendent generations 11–12 and 21–22, Fig. 2). By contrast, the levels of cytogenetic aberrations remained similar at doses between 51 and 356 mGy (Figs. 1 and 2). These observations show that the levels of cytogenetic aberrations in descendent generations of bank voles surviving the ChNPP meltdown did not follow the linear no-threshold model (LNT) at low doses (< 356 mGy).

The availability of field data from one year (1996) from site 5, where the historic dose was 10.28 Gy, made it possible to examine the relationship between dose and cytogenetic aberrations in descendent generation in the high-dose radiobiology scenario. The level of cytogenetic aberrations in generation 21–22 at site 5 was significantly higher than those in the same generation at the other three sites (2, 3, and 4) where the historic doses were very low (20–356 mGy). When including the high dose from site 5 in the dose response curve, the

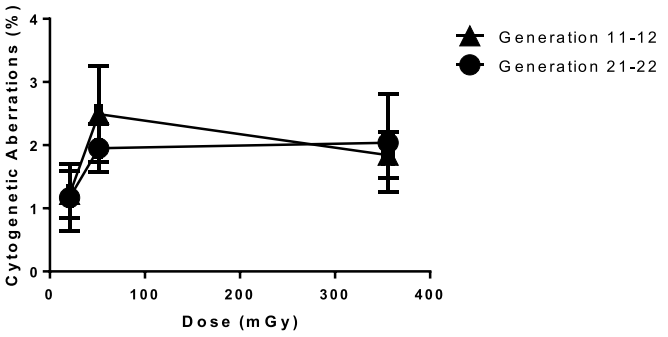

Fig. 2. Relationship between reconstructed historic whole-body radiation doses at sites surrounding the ChNPP accident location and mean cytogenetic aberration rates for bank voles of generations 11–12 and 21–22. Aberrant cell frequencies from three sites (sites 2, 3 and 4) within a 400-km radius of the ChNPP were plotted against the reconstructed historic whole-body radiation doses for those sites. The p value for generation 11–12 is 0.9560 and for generation 21–22 is 0.5541. Error bars reflect the standard deviation of the range of aberrant cell frequencies for the site.

relationship between dose and cytogenetic aberrations follows the LNT model (R (Garnier-Laplace et al., 2013) = 0.9782; p = 0.0218).

Fig. 4 shows the relationship between the historic whole-body dose (20–356 mGy) and the embryonic lethality frequency in sites 2, 3 and 4 for generation 11–12 and generation 21–22. Similar to the cytogenetic aberration endpoint, there is low correlation between embryonic lethality frequency and historic whole-body doses in both generations 11–12 (R^2 = 0.9; p = 0.2385) and 21–22 (R^2 = 0.07; p = 0.8262). Large variations in the data collection accounted for the low correlation (Table 2) and were commonly expected for this type of field work. Despite this challenge, it was clear that the mean embryonic lethality frequency increased in later generations, compared to the earlier generation, at each contaminated site (Table 2) and also increased in a dose-dependent relationship for generations 11–12 from sites 2, 3 and 4 (Fig. 4). For generation 21–22, the mean embryonic lethality frequency associated the historic dose of 51 mGy was higher than that with the 20 mGy dose but was similar to that with the 356 mGy dose (Fig. 4).

4. Discussion

Disasters like those at the ChNPP and at Fukushima allow researchers to examine the real-time effects of ionizing radiation on

Table 2
Reconstructed historic dose and embryonic lethality in bank voles at sites 2, 3 and 4. Generations along with their year of capture are listed.

Year	Historic Dose (mGy)	Animal generation since the accident	Mean Embryonic Lethality (% (range))
Site 2			
1981	4.0 ± 0.3	pre-event	7.23 (4.34–11.18)
1986	20 ± 2	1–2	–
1991	–	11–12	5.36 (1.10–14.87)
1992	–	13–14	4.35 (1.20–10.76)
1996	–	21–22	11.36 (3.79–24.56)
Site 3			
1986	51 ± 3	1–2	–
1988	–	5–6	0 (0–7.98)
1989	–	7–8	6.52 (3.03–12.08)
1991	–	11–12	9.86 (4.06–19.26)
1996	–	21–22	26.67 (7.74–55.10)
Site 4			
1986	356 ± 4	1–2	–
1988	–	5–6	4.76 (0.99–13.29)
1989	–	7–8	5.97 (3.12–10.20)
1991	–	11–12	15.53 (9.15–24.00)
1996	–	21–22	21.57 (11.29–35.32)

environmental health and to refine theories and concepts of the mechanisms involved. What could be learned from studying wild mammals such as bank voles is invaluable to gain a more profound understanding on the realistic effects of environmental radiation contamination on mammalian health. These effects can later be extrapolated to humans and then compared to survivor data to confirm fundamental radiation biology theories. By studying these effects, the accuracy of the LNT model for low-dose radiation can be refined and may thus lead to appropriate modifications in environmental radioprotection guidelines.

Although the historic doses were extrapolated from the recorded ambient dose rates, they may not be entirely representative of the doses received by the progenitor generation of bank voles. Firstly, due to changes in bioavailability of radionuclides like Cs-137 in the bankvoles herbivorous diet and movement of radionuclides through soil layers (Ryabokon et al., 2005), the true total absorbed dose of the bank voles may be slightly higher or lower than our estimated absorbed doses. Secondly, this dose estimation does not take into account shorter lived radionuclides that would have had some significant contribution to the absorbed doses.

It was assumed that bank voles found at the same site throughout the years 1986–1996 would be from the same parental generation due to these animals' non-migratory lifestyle. For cytogenetic aberrations, the LNT relationship was not found at low historic doses (lower than 356 mGy); however, the LNT relationship becomes more apparent only when the only field-available high dose 10.28 Gy was included.

In ionizing radiation-induced non-targeted effects (IR-NTEs), genomic instability is characterized as the higher-than-normal increase in genetic aberration burdens that are potentially lethal in progeny of organisms surviving the initial radiation exposure. In the present paper, increases in both cytogenetic aberrations and embryonic lethality occurred only between 20 and 51 mGy historic doses. This observation was consistent across the studied generation(s) or all sampling sites, further reaffirming the conservation of the classic radiobiological signature of genomic instability at this dose region. Figs. 1 and 2 also show the plateauing effect seen at doses between 51 and 356 mGy for all descendent generations. This displays the contrary of the LNT model and may signify a dose region that elicits radioadaptive effects (Farooqi and Kesavan, 1993; Bonner, 2003; Calabrese et al., 2007). This type of plateau effect was previously seen in two similar studies performed on birds and *Drosophila* in the ChNPP area (Omar-Nazir et al., 2018; Hancock et al., 2019). Generally speaking, IR-NTEs predominate at low doses (Mustonen et al., 2018). Genomic instability and radioadaptive response are two important IR-NTEs and here seemingly developed at different dose intervals pertaining to the low-dose region transgenerationally in Chernobyl bank voles. The acquirement of a radioadaptive response is ultimately essential for survival fitness of an animal population to cope with heightened radiation stress. This is particularly important for the survival fitness of bank voles even 60 generations after the ChNPP meltdown (Mustonen et al., 2018; Ryabokon and Goncharova, 2006b).

Fig. 3 shows the progression of cytogenetic aberrations through generation 21–22 in sites 2, 3, 4 and 5. All areas experienced different levels of historic dose. From Fig. 1, the non-LNT relation is seen for doses below 356 mGy. This is expected in the low dose range. However, for site 5, the dose extends to greater than 10 Gy. It can be deduced that within the range of 1 mGy to 1 Gy, the various low-dose phenomena that prevent the visualization of an LNT relationship between dose and their effects occur. Outside of this range, an LNT relationship is seen (31.). Additionally, the ambient dose that the specimen at site 5 receive is high enough to induce *de novo* mutations that might be passed on and skew the plausible compounding mutation frequencies due to historic doses. For this reason, the historic-dose effects for site 5 cannot be reasonably claimed (31.).

Despite the evidence that seems to disprove the LNT model for the recorded dose effects and proves radioadaptive response and genomic

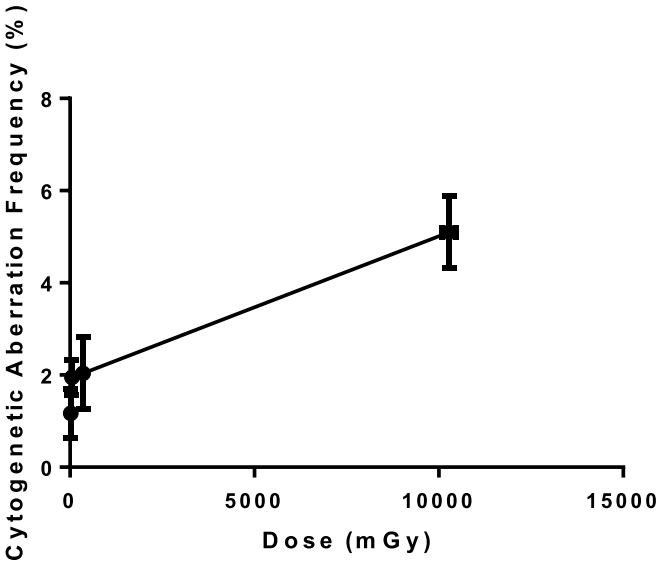

Fig. 3. Relationship between historic whole-body radiation doses from the Chernobyl Nuclear Power Plant and mean cytogenetic aberrations for bank voles of generation 21–22. The cytogenetic aberrations found for 4 sites (n = 4) (Sites 2, 3, 4 and 5) within a 400-km radius of the ChNPP were plotted against the derived historic whole-body doses for these sites. p = 0.0218. Error bars in this figure reflect the range of cytogenetic aberrations for each site.

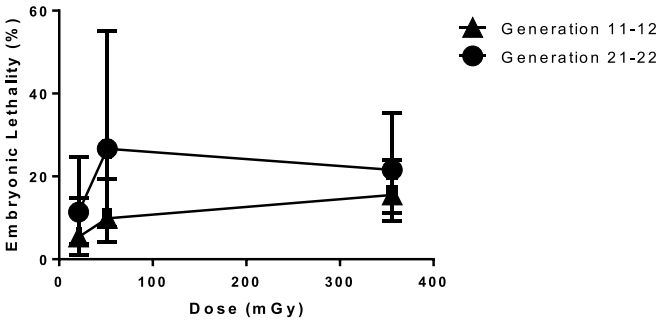

Fig. 4. Relationship between reconstructed historic whole-body radiation doses at sites surrounding the ChNPP accident location and mean embryonic lethality for bank voles of generation 11–12 and generation 21–22. Embryonic lethality frequency from 3 sites (n = 3) within a 400-km radius of the Chernobyl Nuclear Power Plant were plotted against the derived historic whole-body radiation doses for those sites. For generation 11–12 and generation 21–22, the p values reflect insignificance (p > 0.1). Error bars in this figure reflect the range of embryonic lethalities for each site.

instability, a number of technical challenges could occur. Firstly, natural mutation accumulation from the chronic low dose received may be the reason for the rise in embryonic lethality and cytogenetic aberrations. Secondly, environmental stressors outside the realm of dose effects may be behind the unviability of bankvole embryos which would later lead to the rise in embryonic lethality seen. In a similar paper by Ryabokon and Goncharova (2006), it is suggested that in addition to transgenerational genomic instability, the specimen's individual response to the current irradiation levels may play a role in some of the cellular aberrations seen (Ryabokon and Goncharova, 2006b). Another limitation of this study is the scarcity of datapoints that makes it impossible to see the progression of genomic instability throughout each generation. Additionally, specimen with very high frequencies of cellular aberrations may have missed collection due to a shortened lifespan. This shortened lifespan may have also led to them not being able to mate after the accident and therefore, not being able to pass on their acquired genetic instability.

Although, current radiation dose levels may be responsible for some

of the cellular aberrations and embryonic losses seen, similar increases in mutation rates can be seen among offspring of specimen reared in a laboratory environment (Bonisoli-Alquati et al., 2010; Ryabokon and Goncharova, 2006b). This shows that transgenerational genomic instability does play a role in the increased cellular aberrations and embryonic losses seen in biota environmentally exposed to radiation.

Acknowledgements

Funded by:
- Canada Research Chairs Program (Grant no. 950–221284).
- NSERC Collaborative Research and Development Grant (Grant no. RGPIN293153-12).
- CANDU Owners Group (Grant no. CRDPJ484381-15).

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