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LONG TERM EFFECTS OF CHERNOBYL CONTAMINATION ON DNA REPAIR FUNCTION AND PLANT RESISTANCE TO DIFFERENT BIOTIC AND ABIOTIC STRESS FACTORS

I. Boubriak[1], T. Akimkina[2,3]. V. Polischuk[4], A.Dmitriev[5], S. McCready[2] ,D. Grodzinsky[5]

[1]Dunn School of Pathology, Oxford University, Oxford, UK

Email: ivan.boubriak@path.ox.ac.uk

[2]Oxford Brookes University, Oxford, UK

[3]Pharmstore, Moscow, Russia

[4]Department of Virology, Kiev State University, Kiev Ukraine

[5]Institute of Cell Biology and Genetic Engineering, the National Academy of Sciences of Ukraine, Kiev, Ukraine

Email: dmgrod@gmail.com

Abstract

Thirty years after the Chernobyl explosion we still lack information regarding the genetic effects of radionuclide contamination on the plant population. For example, are plants adapting to the low dose of chronic ionising irradiation and showing improved resistance to radiation damage? Are they coping with changing/increased pathogenicity of fungi and viruses in the Chernobyl exclusion (ChE) zone? Are plant populations rapidly accumulating mutational load and should we expect rapid micro-evolutionary changes in plants in the Chernobyl area? This review will try to summarise the current knowledge on these aspects of plant genetics and ecology and draw conclusions on the importance of further studies in the area around Chernobyl.

I.   Introduction

The explosion at the Chernobyl nuclear power station on 26 April 1986 was the most severe nuclear accident in the history of nuclear power since 1954, when the Obninsk nuclear power plant (NPP) became the first in the world to generate electricity for a power grid. Release of radioactivity from the damaged Unit 4 of the Chernobyl NPP during the first ten days after the explosion included radioactive gases, condensed aerosols and large amounts of fuel particles. The total release of radioactive particles was about 14 EBq, including 1.8 EBq of ^{131}iodine, 0.085 EBq of ^{137}Cs, 0.01 EBq of ^{90}Sr and 0.003 EBq of plutonium radioisotopes. The noble gases contributed about 50% of the total release [1]. More than two hundred thousand square kilometres of Europe received levels of ^{137}Cs radioisotope above 37 kBq·m^{-2}; more than70% of this area was in Belarus, Russia and Ukraine. The deposition was extremely varied, as it was enhanced in areas where it was raining when the contaminated air masses passed over [2]. In addition to wide-scale dispersion of contamination, scientists were faced with some special pollution problems, such as the presence of numerous "hot spots", different solubilities of radionuclides, interaction with chemical compounds etc. [3, 4]

The highest levels of contamination for plant populations were observed in the 30 km exclusion zone around Chernobyl and the response of plants here was dependent on the radiation dose, dose rate, type of irradiation, variation in radio-sensitivities of different species and indirect effects from other events. It is crucial to frame any investigation of the consequences of Chernobyl to a specific period of time after the accident because radiation pressures and the resulting effects present now, after almost 30 years, and during the first days and weeks after exposure are dramatically different. Radiation-(radionuclide contamination) induced effects on plants can be framed into three broad time periods: 1) an acute exposure period during the first 30 days after the accident, caused by large quantities of short-lived radionuclides from passing clouds

(^{99}Mo, ^{132}Te/I, ^{133}Xe, ^{131}I,^{140} Ba/La); 2) a second phase that extended through the first year of radiation exposure, during which short-lived radionuclides decayed and long-lived radionuclides were transported to different components of the environment by physical, chemical and biological processes; 3) the third and continuing phase of chronic [5] irradiation derived mainly from ^{137}Cs contamination [6]. Radiation effects on plants in the first (doses in the vicinity of damaged reactor around 20 Gy·d^{-1}) and second phases of irradiation (<10% of initial values) have been well studied and reviewed [7-9] [10, 11] [12] so this paper will mainly concentrate on the long-term radiation effects of Chernobyl from phase three (<1% of initial values) and will attempt to look at possible mechanisms of plant adaptation and resistance to biotic and abiotic factors.

II. Radiation Damage to Plants and DNA Repair Pathways

Irradiation of plant cells by ionising radiation activates a number of events occurring between the original absorption of energy and the final biological injury. Both direct (when energy is deposited directly to the target molecules) and indirect effects are known. In the latter case, energy is absorbed by the external medium, which leads to the production of diffusible intermediates, further attacking the targets [13]. Irradiation leads to water radiolysis in plant cells, producing the most reactive primary radical, ˙OH, which could be responsible for numerous lesions in cells as well as cell death. Other reactive oxygen species (ROS), such as hydroxide, peroxide or less active organic radicals, are also responsible for genetic abnormalities in plant cells [14]. Free radicals have damaging effects in cells, both to membranes and to DNA, but damage to DNA is believed to be more important for the formation of genetically important lesions and for the fate of the cell.

A number of DNA lesions are known to be formed after ionising radiation and the action of free radicals, the most common being single-strand

breaks (SSBs) and double-strand breaks (DSBs), hydrolytic depurination, apyrimidinic sites, and oxidative damage to the bases and the phosphodiester backbone of DNA [15-17] . DSBs are a strikingly genotoxic form of DNA damage; hence, they present a unique challenge to the cell. The failure to repair such breaks in DNA can result in translocations, deletions, and other chromosomal abnormalities, including loss of a chromosome arm [18]. In addition, unrepaired breaks in dividing cells can initiate cell cycle checkpoints, halting division, and, in consequence, slowing the growth of the organism [16, 19]

DNA repair, in coordination with DNA replication, is essential for the maintenance of genome integrity and the survival of plants and mechanisms of DNA repair in plants are very complex. Of the multiple DNA repair pathways, repair (both light and dark) of UV lesions has been well studied, and photo-repair mutants are available [20, 21]. Much less detail exists about different repair pathways for ionising radiation-induced (and oxidative) damage in plants [13, 15, 22].

Plants possess base excision repair (BER), nucleotide excision repair (NER), and mismatch repair (MMR) mechanisms and all of these can play a role in dealing with radiation/oxidative damage. Most of the plant DNA glycosylases that initiate the first steps of BER of oxidative damage are bifunctional (glycosylase/lyase). They not only hydrolyse the glycosylic bond to release the damaged base, but also cleave the DNA backbone at the resulting abasic site [15]. A number of enzymes that are able to work on different types of gamma-irradiated DNA substrates are found in *Arabidopsis* [23, 24]. The enzymes, AtNTH1 and AtNTH2, are able to release both urea and thymine glycol from double-stranded polydeoxyribonucleotides. The genes for enzymes specific to the most common form of oxidative damage, 8-oxoG, (AtMMH and AtOGG1) have been cloned in *Arabidopsis* [23, 25, 26]. Other proteins of the

BER pathway, including those involved in gap tailoring (AP endonucleases: AtARP, AtAPE1, AtPE2), DNA synthesis and ligation (AtPOLL, OsPOLP1, AtLIG1) are also found in plants [27-30].

Although the majority of oxidative damage in plants is repaired by BER, the versatile NER pathway is sometime considered as a backup option for repairing oxidative damage of irradiation origin [31]. The first stage of NER is DNA unwinding and cleavage of the damaged DNA strand either side of the lesion (to release the damaged section as an oligonucleotide), followed by filling the gap by DNA polymerases and sealing the DNA by ligases. The key components of NER in plants were found by genetic analysis of *Arabidopsis* mutants. These include endonucleases AtRAD1, AtRAD2/XPG and AtERCC1, helicases AtXPB1 AtXPB2, and ligases AtLIG4 and AtLIG6 [18, 32-35]. Similar proteins (homologues) for NER are found in other plant species such as rice (*Oryza sativa*), grape vine (*Vitis vinifera*) and moss (*Physcomitrella patens*) [15].

The most abundant DNA lesions after gamma irradiation of plants are single-strand breaks. Those that have damaged termini (produced by ROS), such as $3'$ phosphate or phosphoglycolate end groups, cannot be repaired in one step by DNA ligase and are, broadly, repaired by either BER or NER overlapping mechanisms. For all eukaryotic cells, including plants, proteins from the PARP superfamily are known to be putative SSB sensors. PARP functions as a nick sensor, attracting proteins needed for end processing, gap filling and ligation of DNA [5, 36]. Irradiation of *Arabidopsis* plants that results in the production of SSBs also leads to accumulation of plant AtPARP1 and AtPARP2 proteins with (ADP-ribose) polymerase activity, which promotes DNA repair [37, 38]. Plants also have another unique group of DNA repair proteins that sense DNA breaks and catalyse the removal of $3'$ end blocking lesions, thus playing an important part in the repair of SSBs [39, 40].

Despite plants having so many different mechanisms to deal with irradiation/oxidative damage, some lesions may escape repair and these can severely impair/block DNA replication. To enable duplication of DNA in the presence of oxidative damage, plants possess DNA polymerases that are able to bypass damage and conduct trans-lesion DNA synthesis (TLS). One example of such a TLS DNA polymerase is a protein found in *Arabidopsis*, AtPOLK, which is able to insert a nucleotide opposite 8-oxoG (a damaged nucleotide) [41, 42]. Such mismatches can further be dealt with by mismatch repair, which is known to function in plants [43, 44].

Repair of oxidative damage and SSBs is important in plants and failure to repair these properly may lead to the formation of DSBs. The latter breaks can also be produced directly by clusters of hydroxyl radicals generated by ionising irradiation of plants. As mentioned above, repair of DSBs is crucial both for prevention of the majority of genetic abnormalities, and for survival of plant cells. We now have considerable knowledge of DSB repair in plants, which can be performed both by homologous recombination (HR) and non-homologous end-joining (NHEJ) [45-48].

In homologous recombination the first step – recognition of DSBs – is achieved by the MRN (MRX) and CtIT recognition complex together with BRCA1 and BARD1, homologues of all of which are found in plants [49, 50] [51]. Plants also possess a number of RecQ-type helicases that are essential for full resection [52, 53]. At the next stage, single-stranded 3′ overhangs are coated with RPA1, which is eventually replaced by RAD51 to facilitate invasion into the homologous template. Three RPA1 homologues have been found in *Arabidopsis* [54] and at least four proteins similar to RAD51 and the function of some of these is not totally clear [47, 55, 56]. Further steps in homologous recombination are less well studied in plants but two HR models –

either double-strand break repair (DSBR) or synthesis-dependent strand annealing (SDSA) – are both considered [57].

In plants, HR and NHEJ mechanisms compete with each other depending on the type of ends at the DSB. In addition to this, the selection of repair pathways by plant cells is influenced by cell type, stage of development and the phase of the cell cycle [58, 59], although NHEJ is still thought to be the predominant repair mechanism for DSBs [57]. In the first step of the NHEJ pathway, a Ku70-Ku80 heterodimer binds to broken DNA ends to prevent their degradation. End processing of breaks is carried out in the presence of RAD50 by MRE11, with further ligation by DNA ligase IV and XRCC4. *Arabidopsis* mutants, Atku70 and Atku80, are sensitive to DSB-inducing agents and a direct interaction between AtKU70 and AtKU80 proteins at the DNA ends has been confirmed [60, 61]. Both At*rad50* and At*mre11* mutants have been described in plants and the ligase complex AtLIGIV and AtXRCC4 has been isolated and found to be induced after irradiation [62-65].

At least four different NHEJ pathways exist in plants [48] and, because all of them can lead to short deletions or insertions of DNA as a result of joining broken ends without regard for absolute DNA sequence fidelity, these pathways are responsible for the accumulation of genetic changes after ionising irradiation that will be described later in this review.

After irradiation of plants, complex signalling cascades are triggered by cells to minimise the deleterious consequences of the accumulation of DNA damage. The resulting effects of such signalling include delaying cell cycle progress to allow DNA repair as well as activation of genes required for repair and cellular protection. In plants, as in mammalian cells, two protein kinases, AtATM and AtATR, are involved in the early signalling process and AtATM activation occurs with DSBs accumulation whereas AtATR primarily depends on single-stranded DNA at collapsed replication forks or excision repair [66,

67]. Downstream factors that are activated by plant stress kinases, have recently been cloned and their action described [68, 69]. Of these suppressors of gamma response 1, protein AtSOG1 plays a crucial role similar to that of p53 in mammalian cells, and protein kinase AtWEE1 is involved in the control of cell cycle checkpoints [70] [57].

The most up-regulated gene after acute gamma irradiation of plants is AtBRCA1 [66]. Induction of this gene by several hundred-fold points to the importance of DSB repair in plants. Because RAD51 protein is also essential to the HR machinery it is no surprise that it is also highly induced in plants after irradiation, as in many other species [47, 71, 72] . Other highly up-regulated genes include ligase AtXRCC2 and helicase AtRECQ3, AtPARP genes from the BER pathway, checkpoint regulator AtWEE1, AtRPA proteins and many others [57, 73].

Most of the damage/repair responses of plants have been investigated using acute gamma irradiation. There is limited information available on the dynamics of DNA damage accumulation and molecular mechanisms for recovery from radiation injury as a function of dose rate and/or type of the irradiation used [74]. *Arabidopsis thaliana* plants exposed to chronic or acute radiation exhibited differential regulation of genes for the DNA repair machinery. The absence of gene up-regulation observed in low dose rate(LDR)-treated cells suggests that DNA damage might not be properly signalled [75]. Exposure to high dose rate radiation (HDR), which leads to significant DNA damage, is perceived by plants as a severe stress and forces them to up-regulate their defence mechanisms and this is non-specific and includes genes common to different abiotic stresses [76]. In contrast, chronic irradiation at the same total dose of 1.0 Gy but delivered in 21 days ($0.3{\times}10^{-4}$ Gy·min^{-1}) did not alter the expression profiles of DNA repair or other stress genes [75], supporting the

hypothesis that a defined damage threshold is needed to switch on the cell defences [77, 78].

Gamma irradiation at different dose rates also induces different DNA damage responses in *Petunia hybrida* cells. Significant fluctuations in accumulation and repair of DSBs were observed between HDR (5.15 Gy·min^{-1}) and LDR (0.33·Gy·min^{-1}) treatments in the 0–4h time period following irradiation [74]. Finally, differential expression of miRNA regulating expression of some DNA repair genes was found in response to low and high dose-rate gamma-ray exposure of rice plants [79].

All these data suggest that acute and chronic exposure have different effects on genome integrity, which would be expected to have implications for plants growing in radionuclide-contaminated environments as they are mainly exposed to low dose-rate chronic gamma irradiation. Moreover in the case of radionuclide contamination of plant populations, as occurred after the Chernobyl accident, the situation is even more complicated because of the presence of different types of radiation with different linear energy transfer (LET). The genetic consequences for contaminated plants from such a mixture of radiation on the basis of dose-only measurements may be difficult to predict.

III. Genetics effects of Chernobyl contamination on plants

The response of plant biota to the Chernobyl radio-contamination is the complex interaction between radiation dose, dose rate, temporal and spatial variation, varying radiation sensitivities of the different taxons and indirect effects from other events [6]. Because of the complexity of the effects and interactions, estimation of genetic risk to the plant populations growing under different levels of radionuclide contaminations and the particular conditions at any given contaminated sites becomes a major task.

Various methods are available for screening mutation processes in somatic plant cells. These range from the analysis of chromosome aberration frequencies and analysis of micronuclei formation to more advanced techniques, such as measuring mutation frequencies in the repetitive DNA sequences termed "expanded simple tandem repeats" (ESTR) or using reporter genes in transgenic plants to follow mutations, homologous recombination rates and genome stability [80-82].

Cytogenetic studies at Chernobyl can be grouped into those conducted during the first few years post-accident, with higher dose-rate irradiation, and those conducted during the later decades which were characterised by chronic exposure to a declining dose rate. Most results from the early studies clearly showed an increased mutation burden in plants. Results from the last decade have been more controversial [6].

Early cytogenetic studies in Chernobyl, using winter rye and winter wheat from the1986 harvest, demonstrated a dose dependency in the number of aberrant cells recorded. A significant increase over the control level of aberrations was observed at an absorbed dose of 3.1 Gy. Analysis of three successive generations of winter rye and wheat on the most contaminated Chernobyl plots revealed that the rates of aberrant cells in intercalary meristems, in the second and third generations, were higher than in the first and, thus, an accumulative effect of irradiation over generations was evident [83]. Later studies on wheat plants that were descended from two genetically identical populations (derived from the same homogeneous parental line) and grown in soil with comparable agrochemical characteristics in the ChE zone at high (900 Ci·km^{-2}) or low contamination (<Ci·km^{-2}) level, confirmed the increased mutation rate from contamination. If the spontaneous mutation rate was 1.03×10^{-3} per locus, then in the exposed group this was 6.63×10^{-3} -

more than a six-fold increase in the mutation rate over the single generation of exposure to ionising radiation [84]. It was estimated that, in this experiment, the total exposure dose for the wheat plants did not exceed 0.3 Gy, with external and internal components of 0.2 and 0.1 Gy, respectively. Such a low-level radiation dose should not cause such a large increase in the mutation rate, suggesting that chronic exposure to the ionising radiation of Chernobyl has component(s) that are as yet unknown.

Arabidopsis, a very useful plant model system, was also used for genetic studies because *Arabidopsis* plants were found in all the contaminated areas around Chernobyl. Mutation dynamics in *Arabidopsis* was studied from 1986 to 1992 at different levels of contamination and the mutation rate showed a correlation with the accumulated radiation dose. Although, in later years, the mutation rate tended to decline, even in 1992 it was between 4–8 times higher than the spontaneous level [85]. Another native plant in the ChE zone that was widely studied, not only in the first acute phases of Chernobyl contamination but also in later years, is the Scots pine, which is also known to belong to very radiosensitive woody species[86]. In the first years after the accident, the mutation frequencies at enzyme loci in pine populations from the Chernobyl exclusion zone were 4–17 times higher than in the reference population [87, 88]. The cytogenetic effects of Chernobyl contamination of Scots pine were also checked during from 2003–2009 and the frequency of aberrant cells as well as more severe types of cytogenetic alterations (e.g multipolar mitoses, lagging chromosomes) were recorded in exposed pine trees in every year of the study [89] .The occurrence of cytological abnormalities increased with the dose absorbed and ^{137}Cs activity levels in the soil [90]. Finally, in experiments conducted on pine cones that were collected in December of 2009, a significant increase in the rate of enzymatic loci mutations was found with a radiation exposure at dose rates as low as 0.8 μGy·h^{-1}. Even more than 20 years after the
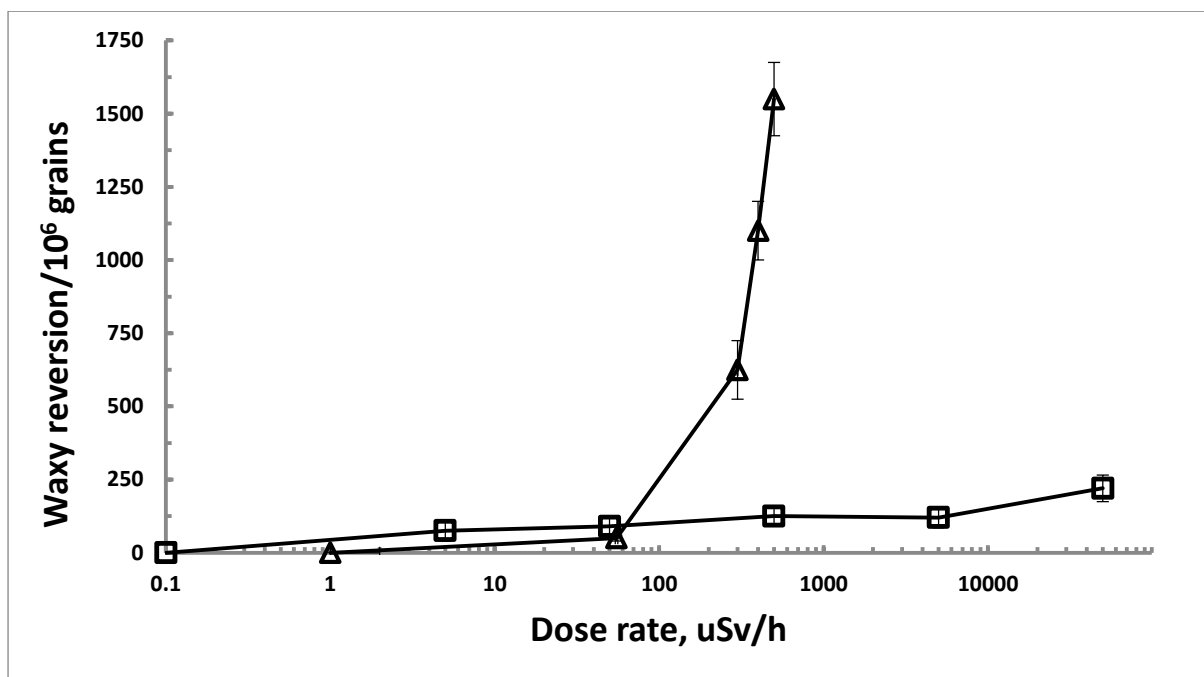
original pollution, the main parameters of genetic variability of chronically irradiated Scots pine populations were considerably higher than values from the reference site. Changes in the genetic makeup of Scots pine populations were observed at dose rates greater than 10.4 µGy h^{-1} [91]. Dose rates at which genetic changes were detected in Scots pine were much lower than those proposed as the benchmark level for a detectable effect [92, 93]. Of course, one should remember that the Chernobyl pine population studied was originally (in 1986) highly irradiated from short lived radionuclides that have now decayed. It was shown that plants can transmit stress responses to their unaffected progeny [94], which may, in part, explain higher than expected genetic changes in radio-contaminated plant populations. The molecular mechanism of such plant responses is not yet well studied and will be discussed in the next chapter.

In addition to mutation rates in somatic cells after irradiation it is very important also to follow mutation rates in generative cells, as these are responsible for the transfer of genetic information to future generations. Such cells are haploid and do not possess the advantages of a second copy of the genetic material and, thus, they are very radiosensitive [3]. The genotoxic effects of Chernobyl contamination have been studied in the *waxy* strain of barley. Increased levels of radionuclide pollution during ontogenesis resulted in a higher incidence of meiotic disturbances and abnormalities during formation of male gametophytes [95]. This barley strain was also used to score waxy reversion in mature pollen grains grown at different levels/composition of Chernobyl contamination [3]

The waxy test on barley pollen proved to be sufficiently sensitive to evaluate the increase in waxy-reversion frequencies in pollen of plants exposed to as little as 3 mSv over 55 days. A dose of 50–200 mSv from external irradiation was needed to double the mutation rate in barley pollen. Moreover,

the smaller doses (dose rates) were more harmful than the higher doses if measured as the increase in mutation frequency per unit of irradiation. But the most striking result was exponential increase in the waxy mutation rate in the case of Chernobyl radionuclide contamination, which included not only gamma and beta-emitting radionuclides but also alpha particles (Fig1).

It is clear that we face a higher genotoxicity from combined irradiation due to mixed radionuclide contamination than from similar doses from external chronic irradiation. It is obvious that mutagenic effects in plant cells, in the case of radionuclide contamination of the plant populations, could not be extrapolated only on the basis of measured/estimated dose, but separate investigations should be carried out to establish the genotoxicity of specific radionuclide mixtures and possible toxic chemical synergisms that can affect

Figure.1 Frequencies of radiation-induced *waxy* reversions in barley pollen collected at the gamma-field (squares) and Chernobyl pollution in 1987 (triangles). Adapted from [3]

the level of cytogenetic damage in somatic and germ cells, reproductive capacity, adaptive differentiation and the general fate of the plant populations [3, 91]
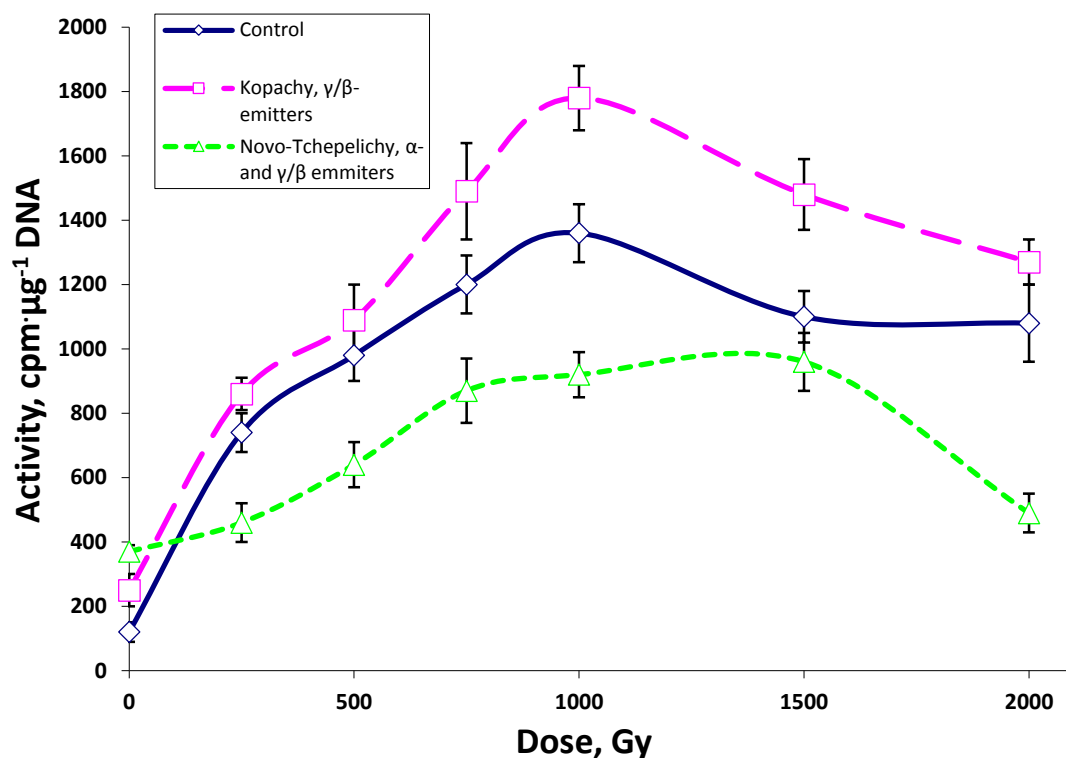
IV.   Adaptation strategy of plant populations to the chronic irradiation conditions of Chernobyl

The plants that have remained in the contaminated Chernobyl areas since 1986 encapsulate all the effects of radiation. Such plants are exposed to α-, β- and γ-emitting radionuclides from external sources as well as to radionuclides that are absorbed from the soil and/or accumulated internally from the plant surface [4]. This combined irradiation leads to an accumulation of DNA damage within each cell and would be expected to trigger defensive responses and/or adaptation to irradiation stress. An adaptive response could be considered as a nonspecific phenomenon in which exposure to a minimal stress could result in increased resistance to higher levels of the same or other types of stress later on [96]. Adaptation is a complex process by which populations of organisms respond to long-term environmental stresses through permanent genetic change [97]. Among the probable underlying mechanisms of adaptation is the activation of multiple signaling pathways that trigger cell defenses, including activation of DNA repair systems, antioxidant production and up-regulation of free radical scavengers. Nevertheless our knowledge of the adaptation of plants to chronic irradiation is still limited. Ecological surveys in the Chernobyl area have revealed specific plant communities that have become adapted for survival in the radio-contaminated soils, despite the presence of genetic damage in chronically irradiated plants [98, 99], but the mechanism of such adaptation is unclear.

One of the obvious mechanisms of plant adaptation is up-regulation of DNA repair functions, and we have evidence that both SSB and DSB repair play a

role in adaptive responses in plants [96, 100]. Thus, in many original studies on plants exposed to radionuclide contamination of Chernobyl, scientists looked at the efficiency of different repair mechanisms under irradiation stress. In early experiments with birch pollen from Chernobyl sites, no evidence of radiation stimulation of DNA repair activity or of any alteration in the expression of DNA repair enzymes was found [101], even though birch pollen is known to adapt its repair function as a reaction to UV stress [102]. Moreover, it was shown that DNA repair synthesis was strongly inhibited in the first generation of pollen formed on birch trees after the Chernobyl accident, in 1987 [103]. In the years that followed (1988–1990) subsequent generations of pollen have regained much of their capacity to perform DNA excision repair after radiation stress, except in pollen from the highest levels of contamination from combined α- and γ/β-irradiation [104]. It was only much later, in the 1998 pollen collection, that the first evidence appeared that pollen formed at intermediate levels of contamination, with only γ/β-emitters present, was showing an enhanced DNA repair capability and an increased tolerance to irradiation stress (Fig.2).

It is no surprise that, in birch, adaptation to irradiation was found in pollen many years after first the exposure to radio-contamination; little evidence exists for the adaptation of long-lived plants (e.g. woody species) in contrast to species with short life cycles, where intense selection can result in rapid genetic changes in the population [90]. Nevertheless a similar adaptation pattern was demonstrated for another woody specious – Scots pine, for which seed from the 1997 Chernobyl collection showed a higher resistance to acute gamma irradiation, despite an increased frequency of aberrant cells in the progeny [105]. Obviously, natural selection of favourable mutations in the "classical sense" cannot play a role in the adaptation of long-lived species, so one of the possible explanations of adaption in the case of pine trees is a selection of cells

Figure 2. Dose dependency of DNA repair synthesis in birch pollen from sites with different radionuclide contamination. Adapted from[106]

on the basis of their radiosensitivity – e.g. elimination of radiosensitive cells and accumulation of a radioresistant pool of cells [105]. An alternative explanation is the induction of heritable epigenetic changes that can modulate expression of stress response genes. This hypothesis was tested on pine trees and significant dose-dependent hyper-methylation of genomic DNA was confirmed in Chernobyl trees grown at different contamination levels[107].

Another well-known adaptive responses to any stress – an increase in variation – can also be caused by exposure to radiation. An increased variety of morphogenetic changes has been documented in 96 plant species belonging to 28 families that are widespread around the Chernobyl reactor site [108]. A higher cytogenetic variation, compared to the control values, indicates active adaptation of the plants[6]

The most comprehensive study on the molecular aspects of plant adaptation to life in the ChE zone, was carried out during 1986 –1992 using native *Arabidopsis* plants [109]. It uncovered the role of homologous recombination in the adaptation mechanisms, where adapted (resistant to mutagens) populations of *Arabidopsis* show a more than 10-fold lower frequency of extrachromosomal homologous recombination (HR) events, which may prevent significant genome rearrangements. This is despite that fact that homologous recombination is expected to deal with an increased incidence of DSBs in such plants and the level of HR events usually show an increase after chronic irradiation in plants [75, 110]. Also, it is quite possible that, under prolonged accumulation of DSBs in irradiated genomes, plants shift their strand break repair mechanism toward NHEJ – a more efficient, but more error-prone mechanism of repair. Another feature of radio-adapted *Arabidopsis* populations is a significant difference in the expression of radical scavenging (*CAT1* and *FSD3*) and DNA-repair (*RAD1* and *RAD51-like*) genes upon exposure to mutagens (including X-rays), as well as a higher level of global genome methylation.

It is clear that adaptation to ionising radiation is a complex process involving epigenetic regulation of gene expression and genome stabilisation, and, as a rule, adaptation improves a plants' general stress resistance, including resistance to environmental mutagens[109, 111]. Such examples of "non-specific" adaptation to environmental stress factors in the ChE zone was shown in *Oenothera biennis* plants first collected after 12 years of radio-contamination. When the seeds were subjected to stress in the form of accelerated ageing (which leads to enhanced DNA fragmentation and premature death of seed embryos [112]) the germination parameters were higher in seeds from radionuclide contaminated regions compared to the controls. Moreover, these

more radio-resistant seeds were also better able to withstand osmotic stress[106].

Evolutionary adaptation potentially helps plant species counter stress conditions in their ecological surroundings. However, this rule does not always apply and some radiosensitive species may lack the capacity to adapt in a particular radio-ecological situation [90]. For example, no obvious adaption was evident in *Linum usitatissimum* plants grown in the radio-contaminated ChE zone [113], despite the same research group showing adaptation of *Glycine max* plants grown at similar conditions of radio-contamination, including adaptation to stress from the heavy metals [114]. The same species may show different levels of adaptation (e.g. seeds *Pinus silvestris*) if collected at different times or in different radio-contaminated areas [89].

In the absence of adaptation to chronic irradiation conditions in some plant populations, the question arises – under what conditions should we expect radio-adaptation? As the response of a population depends both on the biological features of the plants (type of organism, method of reproduction, repair efficiency, radio-resistance etc.) and the type of irradiation (e.g. relative biological effectiveness of radiation, dose rates, linear energy transfer) [90] all these parameters should be taken into the account when radio-adaptation is investigated. The role of different types of irradiation on adaptation of *Oenothera biennis* was shown when plants growing under combined α- and γ/β-irradiation failed to adapt, in contrast to plants exposed to radio-contamination with γ/β-emitters only[106]. Moreover, in this case, even the potential mechanism of failure to adapt was proposed to be based on the inability of cells to upregulate repair of SSBs in the presence of α-emitters in the environment [101, 115].

The adaptation of plants in radio-contaminated environments may also include changes in their metabolism, carbon consumption etc. [116] and all this will have further effects on the resistance of plants to biotic stress factors, as discussed below.

V. Chernobyl contamination and resistance of plants to fungi and viruses

In addition to the accumulation of hidden genetic damage, plants in radio-contaminated areas of Chernobyl can also face a significant risk from biotic stress factors. The combined action of radiation and biotic stress on plants is a potential danger to plant populations, for at least two reasons. First, low-dose chronic irradiation can reduce the phytoimmunity potential of plants and, consequently, their ability to resist infection. Second, it can act as a mutagenic factor and enhance race formation, leading to the emergence of new clones in the populations of pathogenic microorganisms. After the Chernobyl disaster, a substantial increase in the rate of radiation mutagenesis could be expected, especially in those populations of plant pathogens which are characterised by high rates of reproduction [117]. Among these pathogens, pathogenic fungi cause the most diversified and harmful diseases of cultivated plants.

It was found that that low-dose chronic radiation decreased plant immunity potential. Analysis of the incidence of wheat powdery mildew in three cultivars revealed that the extent of this disease was 2-fold higher in plants grown from seeds collected in the ChE zone than that in plants grown from uncontaminated control seeds [118]. The data were confirmed in a set of experiments involving artificial inoculation of wheat plants in a greenhouse. Seedlings of three wheat cultivars grown from contaminated seeds were infected at the second leaf stage by brown rust spores. It turned out that the incidence and development of disease in the seedlings was 2.6-fold higher than in seedlings grown from control seeds.

To discover the biochemical mechanisms underlying the decrease in plant resistance under radionuclide contamination, an analysis of protease inhibitors, which could form stable complexes with the proteolytic enzymes of the pathogens, thus restricting disease development, was carried out on chronically irradiated wheat, rye and corn cultivars. The absorbed dose during the vegetation period on experimental plots in the 10-km ChE zone was between 7–8 cGy for cereals and 3 cGy for corn. It was shown that the activity of proteinase inhibitors (trypsin, chymotrypsin, subtilisin) in plants grown on radionuclide-contaminated plots was significantly decreased. In wheat and rye grains the activity was decreased by as much as15–60 % compared to the control [119]. It is not clear, however, whether other plant defence responses (phytoalexin synthesis and/or accumulation of PR-proteins) could also be affected by low-dose chronic radiation. However, the decrease in activity of proteinase inhibitors appears to diminish plant disease resistance.

Further evidence on the link between chronic irradiation, levels of proteinase inhibitors and plant disease resistance was obtained using the lysine opaque mutation of corn (W64A o2/o2) which has decreased levels of proteinase inhibitors both in grains and leaves. In the mutant corn grains the proteinase inhibitor activity was 3–4-fold less than in unirradiated plants and 2-fold less than in irradiated plants of the original corn line (Table 1). Clearly the corn *opaque* mutant with decreased levels of proteinase inhibitors is highly susceptible to ionising radiation and could be a useful tool in understanding low-dose effects and changes in plant resistance.

Changes in virulence and in aggressivity of plant pathogenic fungi could occur simultaneously with a decrease in plant resistance under chronic irradiation stress in the ChE zone. Wheat leaf rust and oat crown rust caused countrywide losses of up to 10% in Ukraine in recent years even without the effects of irradiation. Genes for resistance to wheat and rye rust diseases may be very

effective against some races, but fail completely to protect against others [120].Thus epidemics can occur when new virulent races of rust appear and are able to increase to a destructive levels before new cultivars could be developed with resistance to them.

Table 1. Specific activity of proteinase inhibitors in corn grains

N	Sample	Trypsin inhibitor		Chymotrypsyn inhibitor		Subtilisin inhibitor	
		$mg{\cdot}g^{-1}$ protein	%, control	$mg{\cdot}g^{-1}$ protein	%, control	$mg{\cdot}g^{-1}$ protein	%, control
1	W64+/+ Control	106	-	36	-	118	-
2	W64+/+ ChE Zone	66	62	23	64	90	76
3	W64o2/o2 Control	129	-	20	-	130	-
4	W64o2/o2 ChE Zone	34	26	6	30	30	23

Because the sexual stage population of the most common causal agent of rust *P. graminis* had been studied in Ukraine long before the Chernobyl accident (e.g from 1966 in "Manevoe" region near Kanev [121]), a distinctive "zero point" was available, enabling analysis of the changes in stem rust population structure in the ChE zone. Analysis of wheat, rye, barley, oats, and grasses on experimental plots in the 10-km ChE zone revealed stem rust development on 12 varieties of cereals. The incidence of disease was about 50–85 % in practically 100 %-damaged crops. All three main forms of *P. graminis* were capable of developing

on many cereal grass species, which serve as a reservoir of infection accumulation between vegetation periods [122]. 642 monopustule clones of stem rust were isolated. Among them, eight physiological races of the pathogen were revealed (11, 21, 34, 40, 100, 189, 3к), as well as a race whose characteristics were absent in the European Cadastre of Races, which was named as race "X" [122]. All races demonstrated high virulence based on their reactions in a set of 12 wheat lines with different genes for rust resistance.

Analysis of genotypes of *P. graminis* on monogenic lines showed that more virulent clones were present with higher frequency in the "Chernobyl" population. It was suggested that active form- and race-producing processes occurred under chronic radiation due to an excess of infectious material in the ChE zone. As a result, the population structure of *P. graminis* was changing with the appearance of a "new" population and high frequency of more virulent clones.

Knowledge about chronic radiation-induced pathogen population changes has been growing during the 30 years since the Chernobyl catastrophe. Certain changes in the population of harmful organisms which had occurred in the ChE zone are now confirmed. Among confirmed cases are five new physiological races of the causal agent of wheat powdery mildew on cv. Kiyanka grown under exposure to a dose of 180 mR·h^{-1}[123]. Two of them possess a high virulence, and the danger from the new virulent race X, new for Europe, is still unknown. On another wheat cv. Polesskaya three new races of powdery mildew were identified that had not yet been described in the European Cadastre of Races. Many of the newly recognised effects of chronic irradiation are similar to systemic stress or immune responses, in that there is no simple relationship between exposure and effect and the outcome is not obviously dependent on the dose which makes prediction of the danger from irradiation very difficult [124].
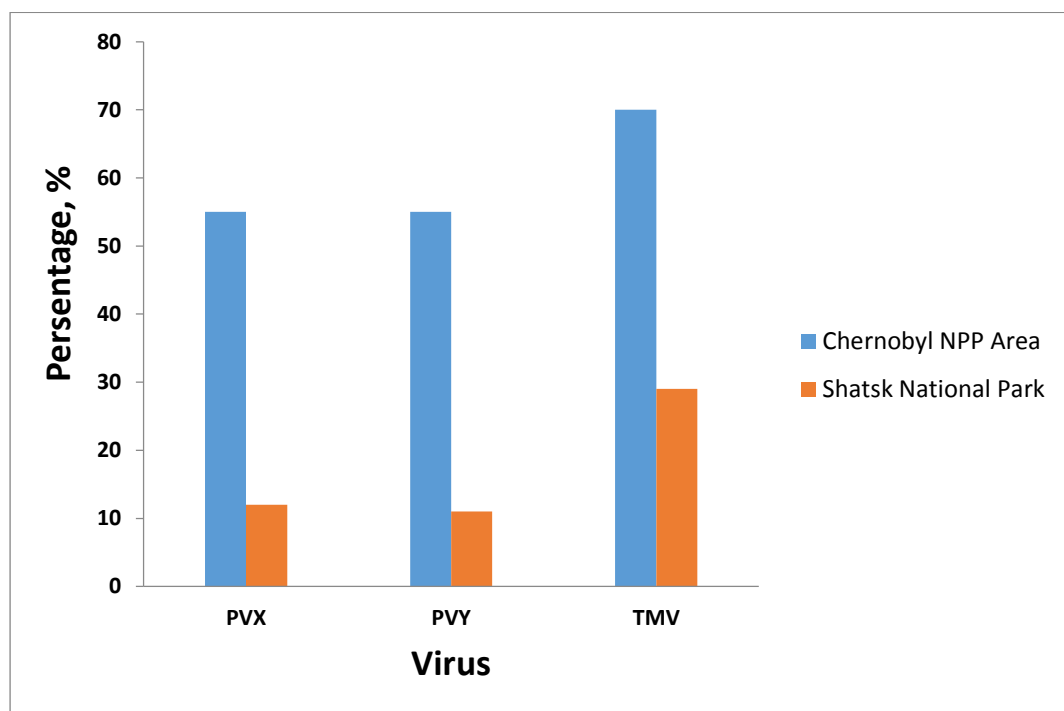
It is too early to draw definitive conclusions about the decrease in plant disease resistance and the increase in plant pathogen virulence under low-dose chronic radiation [125]. But there it no doubt that a special type of monitoring of microevolution processes in plants and fungal pathogens under chronic radiation should provide a better understanding of how serious these threats to agriculture are.

Plant viruses are less numerous than fungal pathogens but they also could be harmful if the transformed environment were to facilitate virus infections. It was shown that ionising radiation and heavy metal contamination of soil may lead to significant changes in symptoms induced by virus infection, elevation of virus content in plants, and possibly mutations in plant virus genomes [119, 124, 126]. Chemical stress factors including heavy metals and radionuclides are known to induce various biologically significant phenomena in the course of viral disease. The development of viral infection under the chronic effect of radioactivity may be accompanied by atypical visual symptoms, aggravated cell pathologies and, supposedly, genetic modifications of both the virus and its host plant [126, 127].

Special concern lies at the population level of virus infections as it has been suggested that there might be an increase in the prevalence and diversity of viruses – raising questions about biosafety. The occurrence of 10 plant viruses (TMV, PVY, PVX, PVM, PVS, PVF, PLRV, BYMV, AMV, CMV) was serologically analysed in wild-growing plants from the ChE zone. It was demonstrated that virus antigens were more frequently detected in plants growing in the radioactively polluted region compared to ecologically 'safe' territories (e.g. Shatsk National Park) (Fig.3) [128]. This finding may be linked to the elevated virulence of the pathogens, decrease in plant resistance against

the viruses, lack of plant fitness to the environment or by a combination of these factors [129, 130].

The host range of a virus is one of the hallmarks of its biological properties. Biological characteristics of isolates of *Tobacco mosaic virus* (TMV) collected from the ChE zone and Shatsk National Park (designated TMVch and TMVsh, correspondingly) were investigated and compared with those of the reference U1 strain of TMV. TMVch was shown to differ in its host range and symptoms, inducing local necroses on *N. tabacum* cv. Samsun plants (which typically reacted systemically to TMV infection) and it is not possible to infect *N. glutinosa* and *N. sylvestris* test plants [131, 132]. Genetic modifications of the TMVch genome have been suggested as a root cause.

Figure 3. Comparison of encountering frequency for PVX, PVY, and TMV in plant samples from regions differing in the level of radioactivity pollution: (PVX – *Potato virus X*, PVY – *Potato virus Y*, TMV – *Tobacco mosaic virus*)

ELISA assays, using both homologous and heterologous specific antisera, showed that purified coat proteins of TMVch, TMVsh and TMV U1 were
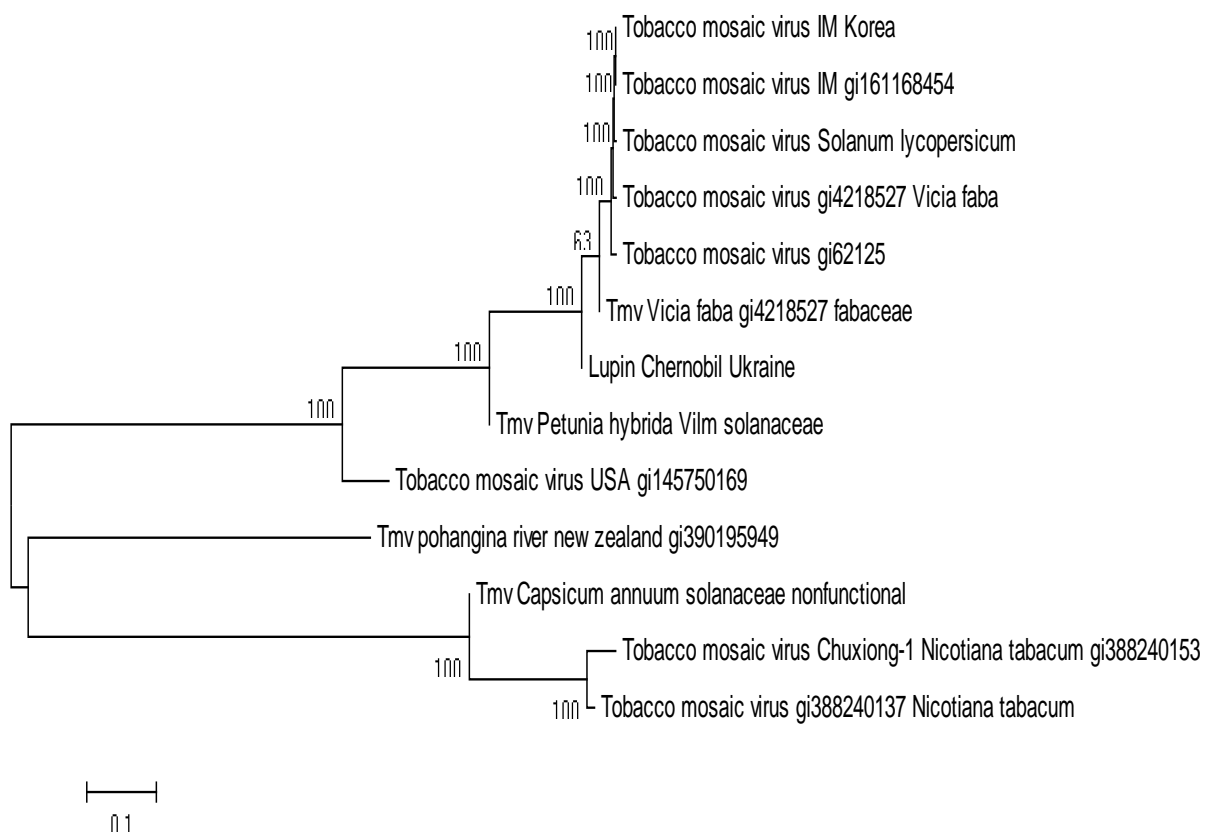
serologically identical,. HPLC-based peptide mapping of tryptic fragments of coat proteins of TMVch and TMVsh isolates did not indicate significant differences in elution profiles. However, such tryptic fragments were shown to be serologically different, suggesting amino acid changes in antigenic determinants of virus isolates. In particular, several fractions of the coat protein of the Chernobyl isolate (TMVch) had a stronger affinity to the antisera compared to those of both TMVsh and TMV U1, also indicating possible change(s) in amino acid composition and/or the 3-dimensional structure of the coat protein molecule of TMVch, leading to the appearance/display of a new/modified epitope(s) [133].

Genetic analysis of virus isolates recovered a novel strain of TMV from lupin plants (′) in the 30 kilometre ChE zone. As lupin was not a known host for TMV, it was suggested that alteration in the properties of 'classical' TMV could be attributed to mutational effects of chronic radioactivity. Polymerase, coat and movement protein gene sequences of this Chernobyl isolate have been compared to sequences available in the Genbank database (TMV isolates sampled from different geographical regions and hosts). Phylogenetic comparison of all three sequenced genes of the lupin isolate of TMV demonstrated that it was homologous to those recovered from 'atypical' host plants belonging to *Balsaminaceae* and *Oleaceae* families. The lupin isolate of TMV was most closely related to the IM strain of TMV isolated from *Impatiens* and to the SXFQ strain (Fig.4), suggesting the possible divergence of these isolates from a single common parent form of the virus or convergence resulting from analogous mutations in different phylogenetic lines [134].

It was found that the tentative timescale of divergence of these three isolates/strains of TMV (lupin, IM, and SXFQ) from a common parent form was between 50–100 years. These data may invalidate the hypothesis that the chronic irradiation (which developed after the Chernobyl disaster only 30 years

ago) was the driver of virus divergence. However, the statistical confidence of results (from the BEAST 2 software used) lies within a range of 50 years, suggesting that full-genome analysis of the lupin isolate of TMV is essential for making solid conclusions about the role of radio-contamination in virus microevolution.

Figure 4. Phylogenetic analysis of the lupin isolate of TMV based on partial RdRP sequence (ME method, 1000 bootstrap replications)

VI Chernobyl and Microevolution

The response of cells and multi-cell systems to the action of ionising radiation is characterised by the development of two parallel processes. The first is associated with damage to cell ultra-structures; the second reflects the cell's response to the perception of radiation as an alarm signal.

The first type of responses is associated with damage to molecular targets, the most important of which are the chromatin of the cell nucleus, mitochondrial DNA, cell membrane systems and molecular assemblies performing the functions of molecular machines. This form of radiation damage has been very thoroughly investigated and the effects that result from damage to these molecular targets is known generally as target effects. The second form of response to irradiation is the complex active response of cells and systems to the perception of radiation as a signal of cytogenetic threat. This has been investigated less completely. These responses of cells and multicellular organisms to irradiation are summarised in Fig. 5.

Irradiation

Perception of radiation as the alarm   Radiation damage to targets

Reception of signal   Cell nucleus

Mitochondrial DNA

Mobilization of gene regulation

Membrane systems

"Molecular machines*

Nontarget effects   *Target effects*

Figure 5. Two pathways of response to radiation

Targeted effects are revealed in the appearance of chromosome aberrations, point mutations, impaired cell membranes, inhibiting the synthesis of proteins and other substances, distortion of bioenergetics processes. These forms of damage can be mitigated, to a certain extent by means of repair processes. In
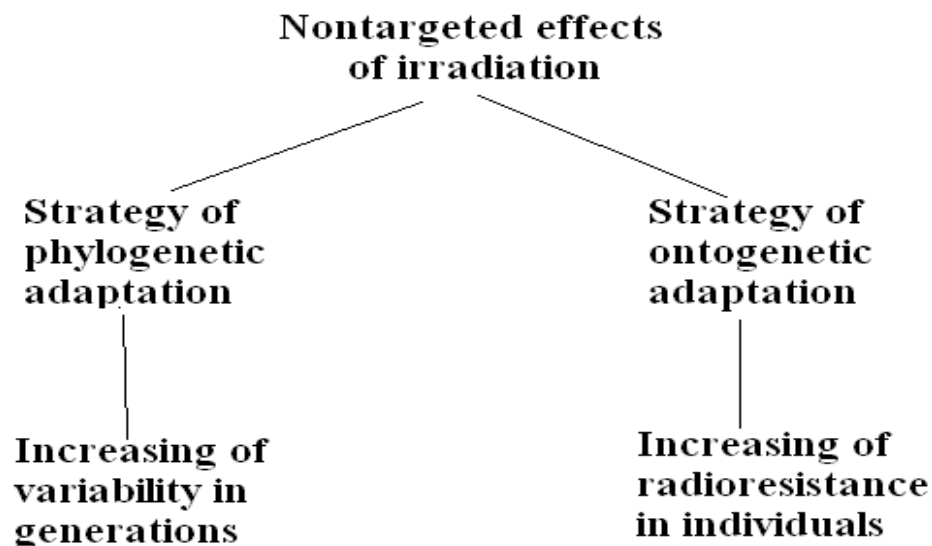
particular, single-strand and double-strand DNA breaks and other types of damage to DNA can be repaired. Repair of membranes occurs by accelerating the membrane flows and increasing the intensity of lipid synthesis. Repair of "molecular machines" is provided by the self-assembly processes using *de novo* synthesised macromolecules. Obviously, differences in the radio-resistance of different species are due both to the targets affected and to the ability to repair the different types of damage to them.

Non-targeted effects are of an entirely different nature compared with the responses of the target effects in cells and multicellular organisms. The appearance of damaged molecules in irradiated cells is perceived by cells as a signal of cytogenetic threat arising in the environment. The cells actively respond to this signal, and a complex process, whose aim is to reduce the risk to the species from the negative impact of radiation, begins in cells and multicellular systems.

The active response of cells and multicellular systems to irradiation appears mainly in the formation of two strategies to adapt to high levels of radiation as an alien and harmful stress factor. One strategy is to improve the radiation stability of the individual. This is the so-called ontogenetic adaptation. In particular, it is shown in radio-adaptation. The essential point of radio-adaptation is to improve radiation stability through a variety of processes including synthesis of DNA repair enzymes, increasing the concentration of antioxidants and other substances that together form a natural background radio-resistance. In addition to these biochemical mechanisms of radio-resistance, radio-adaptation can include increasing cellular processes that extend the presynthetic phase in the cell cycle, thus improving DNA repair. These increase the chance of repopulation recovery and the intensification of cell selection as a way to reduce the genetic load in the cells of the organism.

A fundamentally different type of adaptation, called phylogenetic adaptation, plays an important role. Its aim is to increase the genetic and

epigenetic diversity, within species, that is needed to create a wealth of material for natural selection to increased radioresistance. A diagram illustrating the two adaptation strategies in populations is shown in Fig. 6.

Nontargeted effects
of irradiation

Strategy of
phylogenetic
adaptation

Strategy of
ontogenetic
adaptation

Increasing of
variability in
generations

Increasing of
radioresistance
in individuals

Figure 6. Two strategies of adaptation to chronic irradiation in radionuclide anomalies.

The ratio between these two pathways of the organism's reaction to radiation essentially depends on the dose and the method of irradiation. The first pathway is related to the response to the radiation injuries of molecular targets within cells and it is characteristic for high doses and acute exposures. The second pathway predominates at low doses of ionising radiation and chronic exposure.

Many radiobiological reactions are associated with mechanisms that are involved in the regulation of genetic and epigenetic variability. For example, the induction of genome instability and apparent bystander effects are associated with these mechanisms that constitute the organism's response to radiation.

The fact that the individual variation in a population exposed to radiation at small doses greatly increases can be seen in various experiments on the value

of the variance (standard deviation) of quantitative traits. The increase in biodiversity is observed at different levels of organisation of the organism. In plants it can be seen in the size of the whole plant or its individual organs and on the size of the cells in a particular tissue.

It should be noted that the increase in the size variability of initials in the apical meristem is not dependent on the radiation dose. That can be attributed to the regulatory nature of this phenomenon. This is another example of the increase in volatility during chronic exposure. The coefficient of variation in St. John's wort (*Hypericum perforatum)* growing in the exclusion zone is higher than in the population of this species in the uncontaminated area [135].

Levels of radionuclide contamination

	0 (control)	5	50	600
Coefficient of variation (%)	28	36	37	43

It has also been shown that, in the zone of radioactive contamination, variability of growth and reproductive processes in Scots pine increases sharply [136].

There are various mechanisms to improve genetic and epigenetic diversity. Among them the following events play a greater role in plants:

- augmentation of the frequency of meiotic recombination (this process is accelerated under the influence of low-dose radiation);
- increase in the activity of transposons (the effect of irradiation is confirmed [137]);
- impairment of DNA repair (shown in pollen of birch growing in territory contaminated with radionuclides [106]);

- reduced role of parthenocarpic reproduction (the effect of irradiation in small doses on the increase in the frequency of sexual reproduction has been shown in plants [138]);
- radiation induction of genomic instability [139].

As can be seen, all mechanisms leading to increased genetic diversity in populations are activated significantly by the action of radiation. It should be noted that the impact of radiation on the mechanisms to ensure an increase of heterozygosis is very noticeable. This can be seen from Table 2 which shows the relationship between the number of plants reproducing sexually and the territory contaminated with radionuclides [135].

Table 2. The frequency of St. John's wort (*Hypericum perforatum*), resulting from sexual (2n) and apomictic (4n) breeding in the Chernobyl exclusion zone.

Place of records	Number of plants (in percentage)	
	sexual origin	apomicts
Control 10–14 $\mu R \cdot h^{-1}$	41.1	58.9
Chernobyl (low level of pollution – 60–120 $\mu R \cdot h^{-1}$)	81.2	18.8
Chistogalovka (high level of pollution – 590 – 2600 $\mu R \cdot h^{-1}$)	95.0	5.0

Plants capable of apomixis increase the frequency of sexual reproduction with increasing contamination levels and, therefore, decrease the contribution of apomictic seed formation [135]. A similar phenomenon is observed in some polychaetes, in which an increase in the role of sexual reproduction and weakening of the role of vegetative propagation take place with increasing dose under chronic exposure. It is interesting to note that the coefficients of variation

of the quantitative values of morphological features are much higher in plant sexual rather than vegetative propagation. This is seen from the data in Table 3.

Table 3. The coefficients of variation of morphological features of plants of different origin at different levels of radionuclide contamination of soil

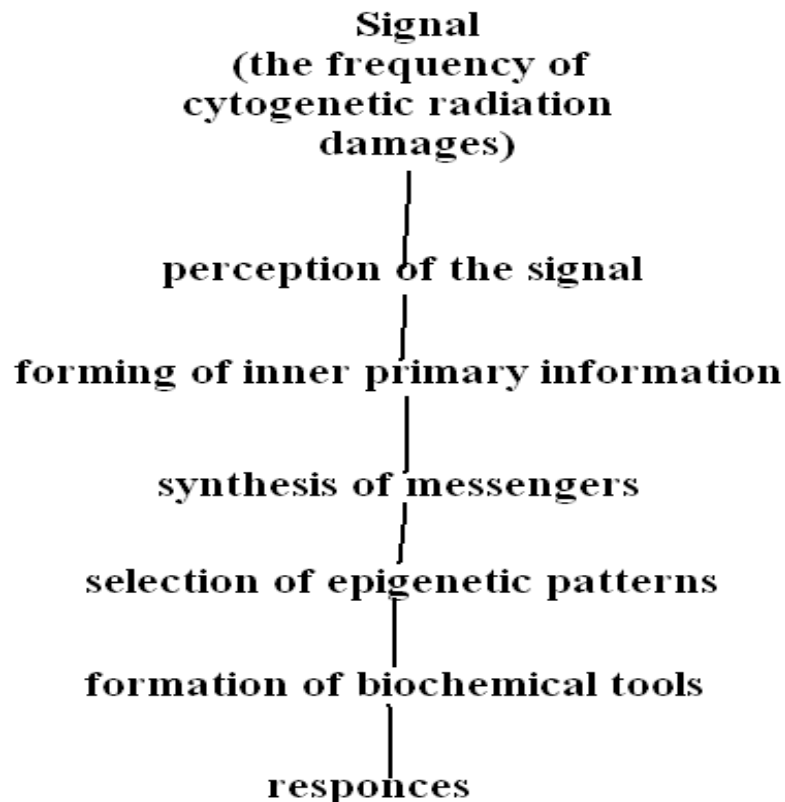| Mode of reproduction (%)	Level of radionuclide contamination (kBq m^{-2})			
	$1.85.10^1$	$1.85.10^2$	$1.85.10^3$	$2,22.10^4$
Sexual	34	38	37	42
Apomicts	7	8	10	8

The ratio of hermaphrodite and unisexual flowers under the influence of chronic exposure is changed so that heterozygosis in seed generation would be increased. The effects of chronic irradiation embrace various genetic events, from genome rearrangement to point mutations.

Plants 'try' to use all possible ways leading to increased heterozygosis. An active response to the alarm signal is carried out through the participation of epigenetic phenomena. This is the result found in experiments in which there is a significant change in the cytosine methylation profiles in the DNA of the plants when they are irradiated with gamma rays or ultraviolet radiation. Fig.7 shows a general signaling diagram illustrating a process of forming a biological response to irradiation.

Accordingly chronic irradiation at low dose-rate is perceived by the organism as a stress effect. Ontogenetic and phylogenetic adaptations should be elicited in response to this stress. The first path of adaptation is associated with the activation of genes controlling DNA repair, membrane currents, and the synthesis of antioxidants as well as a repopulation in meristem tissues. The

second path of adaptation is manifested in the processes that leads to an increase in phenotypic diversity in populations, thus increasing the efficiency of natural selection for enhanced radioresistance in a species. Phenotypic polymorphism in a population also increases due to the epigenetic mechanisms that lead to the appearance of pseudo-mutations.

Signal
(the frequency of
cytogenetic radiation
damages)

perception of the signal

forming of inner primary information

synthesis of messengers

selection of epigenetic patterns

formation of biochemical tools

responces

Figure 7. Circuit response to irradiation by signal transduction pathway

The above data convincingly demonstrate that under the conditions of chronic exposure to low dose rate a wide range of processes leading to the emergence of a population with genetically modified forms and epigenetic pseudo-mutations are involved.

Accelerating the pace of adaptation in hot spots can be a very dangerous phenomenon for the biota. In fact, a spreading of mutant forms can occur that may break the biological balance in ecosystems and lead to the impoverishment of biodiversity. Since interpopulation homeostasis in biocommunities is

associated with the coevolution of many species, in particular in "parasite-host" systems, the altered rate of evolution can disrupt the equilibrium of the biota in ecosystems, not only in hotspots, but also in adjacent areas to which mutated individuals can migrate.

The events constituting microevolution cause accelerated evolutionary changes on the intra-specific level. Because populations of some species are composed of a relatively small number of individuals in ecosystem hotspots, the probability of genetic drift is increased due to the declining fertility of some species which are replenished by migration from areas adjacent to the anthropogenic radionuclide anomaly [140, 141]. This process is accompanied by an increase in the intensity of gene flow. Naturally, the high rate of microevolution processes can be revealed only in those species that are characterised by short periods in the life cycle and where they have a sufficiently large number of generations under conditions of chronic exposure. The examples of stem rust of cereals, the fungus *Puccinia graminis* Pers. and mildew *Erysiphe sp.*,demonstrat an increase in the development of these pathogens due to the emergence of new highly virulent races in the Chernobyl exclusion zone [119]. Results obtained, both in the greenhouse and in field trials, in the Chernobyl Exclusion Zone demonstrate a decrease in disease resistance of wheat, rye and corn cultivars under low doses of chronic irradiation. Seeds of plants grown under conditions of chronic exposure are damaged greatly by fungal infection. The hotspots are gradually becoming the nidus of new virulent forms of pathogens.

Risks to the biota due to an increase in micro-evolutionary processes in the anthropogenic radionuclide anomalies can result in the appearance of rare mutations, an increase in the genetic and epigenetic polymorphism of populations and loss of the balance of species in ecosystems. Clearly, it is necessary to institute monitoring of microevolution processes in hot spots as a helpful security measure. In natural radionuclide anomalies associated with uranium deposits the

range of deviation in genetic variation is not as significant as in the conditions of anthropogenic anomalies. It seems that mechanisms for stabilising variability in populations evolved over a long period of time.

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