SEL'SKOKHOZYAISTVENNAYA BIOLOGIYA
[AGRICULTURAL BIOLOGY], 2015, V. 50, № 2, pp. 225-236 ISSN 2313-4836 (Online)

Radiation biology, protection technologies

UDC 636.5:591.1:539.1.047

doi: 10.15389/agrobiology.2015.2.225rus
doi: 10.15389/agrobiology.2015.2.225eng

ABNORMALITIES IN THE OFFSPRING OF HENS SUBJECTED TO CHRONIC EFFECT OF ^{131}I

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Received November 17, 2014

Abstract

Modern radiobiology and medicine is faced with distant consequences of ^{131}I chronic effects on descendants of animals and people suffered from the Chernobyl' accident. Different disturbances in embryogenesis and congenital malformations are observed in the offspring of those whose thyroid gland was exposed to irradiation with ^{131}I. Nevertheless, a long term prediction of ^{131}I chronic effect in multiple generations is not still cleared up. So, we studied abnormalities of development in seven generations of offsprings which were obtained from Russian white hens subjected to chronic influence of ^{131}I during 30 day administration of the isotope at equal daily doses of 0.11, 1.1, 2.1 and 4.6 mBq/kg. A total of 2573 offspring chickens were obtained 3, 6 and 12 month after the isotope administration has been started, and then they were surveyed for 1.5 to 2.5 years and compared to 2782 control chickens of the same age whose parents were not subjected to irradiation. The offspring of ^{131}I radiation-exposed poultry was characterized by higher postnatal death rates, accelerated sexual maturation which was expressed in an earlier molding and 1st egg laying time, as well as with some decrease in body weight of mature birds for the majority of the generations. The offspring of the first three poultry generations from the group exposed to ^{131}I at a dose of 1.1 mBq/kg had tumors on the jaws, neck and/or conjunctiva. Among the chicken generations 4 and 5, the degenerates appeared at a frequency of 0.75% with abnormalities mostly manifested as head defects (e.g., absence or deformation of eyes or X-shaped jaw decussations). The first four offspring generations older than 6 months being under test exhibited tumors similar to lymphoreticulosarcoma in soft tissues at a frequency from 0.8 to 4.7%. Chickens death in the experimental groups resulted mainly from disorders of gastrointestinal tract, liver, heart and kidney. An increased mortality seemed to be related with changes occurred in the course of embryogenesis leading to improper regulation and adaptation in early postnatal period.

Keywords: incorporated ^{131}I, hens, offspring, developmental abnormalities.

Research into the remote effects of the chronic exposure of animals and people to the radiation of iodine radionuclides on the health of their offspring is one of the topical and complex problems of radiobiology and medicine [1-4]. The importance of such research is explained by the increased interest in the problem after the Chernobyl accident [5-7].

Experimental and epidemiological investigations showed that the parents' thyroid gland exposed to radioactive iodine can cause various disorders in the process of embryogenesis and congenital malformations in the offspring (8–10). However, since data on teratogenic impact of incorporated radionuclides are incomplete, this problem should be further investigated [11-15]. For example, long term predictions of the effects of a chronic exposure to radioactive iodine isotopes on multiple generations are still not clear [16].

The purpose of this work was to investigate congenital abnormalities in the offspring of chickens after a chronic exposure to ^{131}I.

Technique. The subjects of the research performed under vivarium conditions were Russian white chickens and seven generations of their offspring. The parent stock was split into 4 experimental (42-57 birds in each, 183 chickens in total) and 4 control (30-47 birds in each, 150 chickens in total) groups.

All hens were kept with roosters in the ratio of 1 rooster per 10 hens. For 30 days, the birds in groups I, II, III and IV (experimental) received ^{131}I in equal portions and daily doses of 0.11, 1.1, 2.1, and 4.6 MBq/kg correspondingly. The control chickens of the same age received usual nonradioactive water in the same amounts. The birds received the isotope in February and March in the morning before feeding.

A total of 7 generations of offspring were obtained from the hens of the parent stock 3, 6, and 12 months after the beginning of isotope administration (6 generations were studied). For 1.5-2.5 years, the general condition and survival rate of each generation were monitored. The body weight was taken at the age of 1, 10, 20, 30, 40, 50, 60 days and 3, 6, 9, 12, 18, 24 months. Only 10 chickens in each group were weighed before they were 30 days old, with all the birds weighed on all the other dates.

The juvenile molt was estimated by the change of the primary feathers in 3, 4, 5-month-old chickens [17]. The intensity of the molt in all mature birds was studied during September, October and November by the change of the primary feathers.

Postmortem investigations of the internal organs were performed on the killed and died offspring [18]. In each group, 3-5 chickens were killed at the age of 1 day, 1, 3, 6 and 12 months by bleeding. After a macroscopic examination, the internal organs were weighed. The tissue samples taken from liver, spleen, kidneys, heart, thyroid gland, adrenals, and testes were fixed in 10 % solution of neutral formalin and embedded in paraffin. The histological sections were stained with hematoxylin and eosine and using Van Gieson staining protocol.

The experimental data were processed using a standard analysis of variance [19].

Results. In total, 2,573 experimental and 2,782 control offspring chickens were obtained from the hens (Fig. 1).

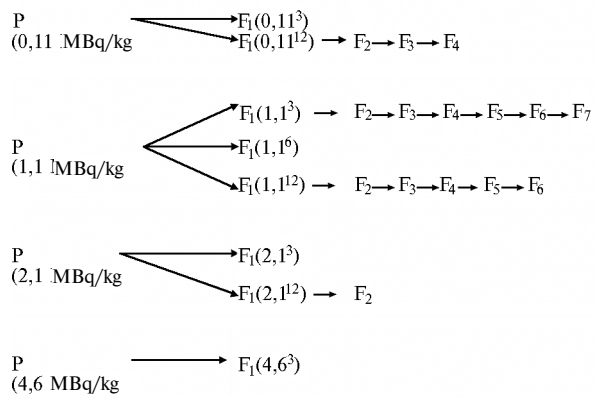

Fig. 1. Obtaining offspring from the Russian white chickens damaged by ^{131}I: P — parents, F_1-F_7 — offspring of generations 1-7 (the experiment was carried out under vivarium conditions). The daily dose of ^{131}I (MBq/kg) received by the parents is given in parentheses. The superscript indicates the number of months after the beginning of ^{131}I administration when F_1 chickens appeared

There were no differences in the appearance between 1-day-old experimental chickens in the 1st, 2nd, 3rd, 6th and 7th generations and the chickens of the same age in the corresponding control groups. Malformations, mainly in the head area, were detected in 0.75 % of 4th and 5th generations of chickens, which received 1.1 MBq/kg of ^{131}I. An experimental chicken from the $F_4(1.1^3)$ group had no right eye. The jaws were deformed in an X-shape fashion (Fig. 2, A). The digits on the legs were bent, and the neck was crooked. It could hardly move and died 2 days after hatching.

A $F_4(1.1^{12})$ chicken had a 1.5×0.5 cm fissure on the right-hand-side of the abdominal wall covered by a transparent skin with a few feathers and light exudate under it. The abdominal organs could be clearly seen. Later on, by day 6, the fissure was replaced by a 1.5×0.5 cm cicatrix. The chicken died

7 days after hatching.

Fig. 2. Pathologies in the development of offspring of Russian white chickens damaged by ^{131}I: A — a missing eye and deformation of jaws in a $F_4(1.1^3)$ chicken, B — a tumor on the neck of a $F_2(1.1^{12})$ chicken at the age of 11 months (the experiment was carried out under vivarium conditions). The daily dose of ^{131}I (MBq/kg) received by the parents is given in parentheses. The superscript indicates the number of months after the beginning of ^{131}I administration when F_1 chickens appeared.

In the same $F_4(1.1^{12})$ group, one chicken hatched with its eyes closed. Opened by force, the eyes could reveal a pupil through a narrow gap. The chicken died 1 day after hatching.

The left eye of a $F_5(1.1^3)$ chicken, which lived for 18 days, was located lower than the right one and represented a swollen round lump the size of a hazelnut with dough-like consistency. The pupil could be hardly seen through a narrow gap.

No deviations were noted in the body structure of other hatched experimental and control chickens.

The changes in the appearance of the experimental birds were observed at the age of 7-21 months. They only appeared in the first three offspring generations of chickens damaged by ^{131}I at the dose of 1.1 MBq/kg and were represented by neoplasms on jaws, neck and conjunctiva. The neoplasms were registered in relatively young (7-12 months) F_1 and F_2 birds and in older (20-21 months) F_3 birds (see Fig. 2, B). No similar abnormalities were noted in other experimental and control offspring groups.

The body weight of 1-day-old experimental chickens increased in $F_1(0.11^{12})$, $F_1(1.1^6)$, $F_1(2.1^{12})$, $F_3(0.11^{12})$, $F_5(1.1^{12})$ groups and decreased in $F_1(1.1^3)$, $F_2(1.1^3)$, $F_2(1.1^{12})$, $F_2(2.1^{12})$, $F_3(1.1^3)$, $F_4(1.1^3)$ groups compared with the control chickens. For the next 18-30 months of life, starting from puberty period, the offspring of the radiation-exposed chickens (both hens and roosters) demonstrated reliably lower weight gains than the offspring from the healthy parents.

The juvenile molt in the experimental and control chickens in all the experimental generations did not start until they were 30 days old.

The 3-month-old experimental and control F_1, F_2 and F_3 hens from group 0.11^{12} did not show essential differences in the amount of replaced primary feathers. For F_2 roosters from the same group the replacement of feathers was reliably more intense than in the control roosters. In group 1.1^3, the amount of replaced feathers increased in F_5 experimental chickens compared with the control. The amount of replaced flight feathers was reliably lower in $F_2(1.1^{12})$ experimental hens and higher in $F_1(1.1^{12})$ roosters. Group 2.1^{12} was characterized by the accelerated replacement of primary feathers in both F_2 hens and roosters ($P < 0.05$).

According to the obtained data, the offspring of the chickens exposed to various doses of ^{131}I molted more intensely than the control chickens. Deviations in the offspring molt depended on the time passed from the beginning of the radiation exposure of the parents: the molt was accelerated in the first generation of birds, and delayed in the subsequent generations.

The control and experimental hens started laying eggs at the age of 5.5 and 6.0 months. Three generations of experimental hens from group 0.11^{12} reached reproductive age at the same time as the control groups. $F_1(1.1^3)$ offspring reached it 14 days earlier than the control hens, and it was 3 days earlier for $F_2(1.1^{12})$ offspring, 32 days earlier for $F_3(1.1^{12})$, 6 days earlier for $F_4(1.1^3)$, and 16 days earlier for $F_4(1.1^{12})$. F_1 offspring of the chickens which received ^{131}I at the dose of 2.1 MBq/kg started laying eggs 20 days later. The molt in $F_1(1.2^3)$ chickens was more intense, and in $F_2(1.2^3)$ and $F_3(1.2^3)$ groups it was more prolonged than in the control groups. Slower molt was also noted in the experimental offspring from group 1.2^{12}.

Both experimental and control offspring showed similar dynamics of age-related changes in the internal organs. Up to the age of 6 months, no deviations in the morphology of the internal organs were noted in the killed birds. Pathological changes, which were not observed in the control groups, were found in died and killed experimental chickens of older age.

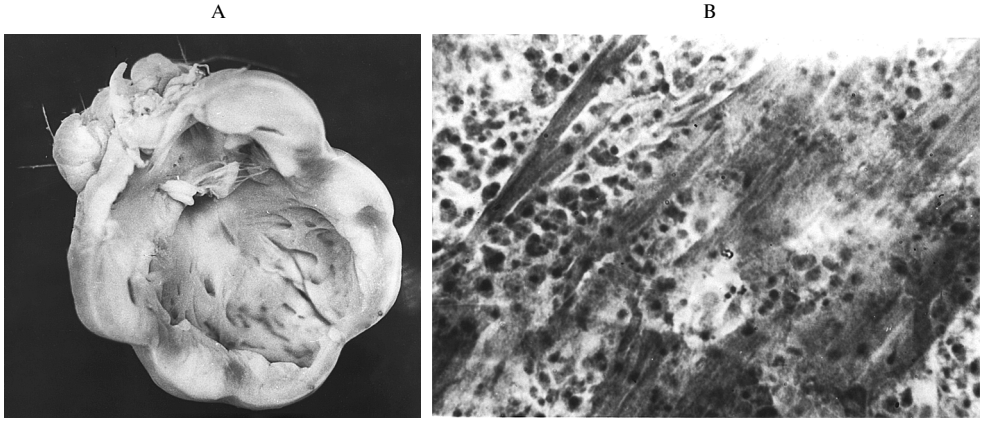

Fig. 3. Heart pathology in a 7-month-old $F_2(2.1^3)$ rooster of the Russian white chicken damaged by ^{131}I: A — a general view of a torose enlarged heart, B — a microphotograph of heart tissues (proliferation of lymphoid and reticular cells, atrophy of muscle fibers can be seen; the experiment was carried out under vivarium conditions). Hematoxylin and eosine stain, magnification ×400. The daily dose of ^{131}I received by the parents was 2.1 MBq/kg; F_1 chickens appeared 3 months after the beginning of the exposure to ^{131}I.

The abnormalities in the heart structure manifested themselves as a growth of a yellow-white tissue on the epicardium, endocardium and myocardium. In case of minor lesions, there were a few nodules in the heart; in case of severe lesions, the heart became torose, its weight increased and sometimes reached 26 g (Fig. 3, A).

A histologic examination revealed infiltration of lymphoid and reticular cells between the muscle fibers of the heart. In the areas of large aggregations of these cells, only a few bundles of thinned muscle fibers with poorly defined cross striations remained (see Fig. 3, B). Cardiac pathologies in the experimental birds suggestive of leukemic damage based on their macro- and microscopic structure were registered many times: one case in each of the $F_1(0.11^{12})$, $F_1(2.1^{12})$ and $F_2(1.1^{12})$ groups and five cases in each of the $F_2(1.1^3)$ and $F_2(2.1^{12})$ groups.

Similar areas of a whitish neoplasm tissue were found in the liver on the experimental chickens. The size of the nodules in the parenchyma varied

from a millet grain to a hazelnut, the organ was enlarged.

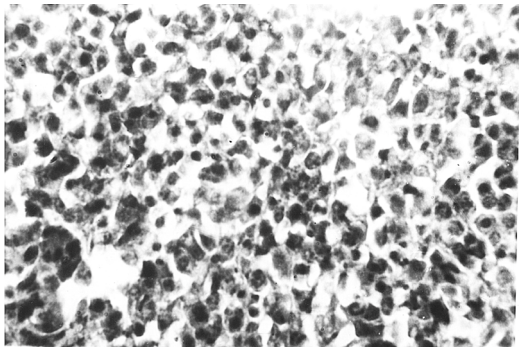

Fig. 4. A microphotograph of reticulosarcoma in the liver of a 9-month-old chicken from $F_2(1.1^3)$ group, one of the offspring of the Russian white chicken damaged by ^{131}I (the experiment was carried out under vivarium conditions). Hematoxylin and eosine stain, magnification ×400. The daily dose of ^{131}I received by the parents was 1.1 MBq/kg; F_1 chickens appeared 3 months after the beginning of the exposure to ^{131}I

Depending on the degree of the damage, a microscopic examination of the liver revealed more or less pronounced proliferation of poorly differentiated reticular and lymphoid cellular elements along the blood vessels. Bigger agglomerations of these cells looked like reticulosarcoma, with the liver tissue being fragmented, its cells in the condition of fatty degeneration and necrobiosis (Fig. 4). Such changes in the liver were identified in five cases, two of them were in $F_1(1.1^3)$ and three were in $F_2(1.1^3)$.

The most frequent disorders in the offspring of the chickens damaged by ^{131}I were kidney morphology disorders. Atrophy of the left kidney and hypertrophy of the right kidney were observed in an experimental $F_2(1.1^{12})$ hen and an experimental $F_1(1.1^3)$ rooster. In 12 cases, morphological changes in the kidneys of the experimental chickens manifested themselves as nodules of a white tissue in the parenchyma of the organ, and as light color roundish lumps emerging on the surface, with dense consistency, the size of a hazelnut or a walnut, frequently stratified if viewed in cross section (Fig. 5). The weight of the kidneys was increased and sometimes reached 25 g.

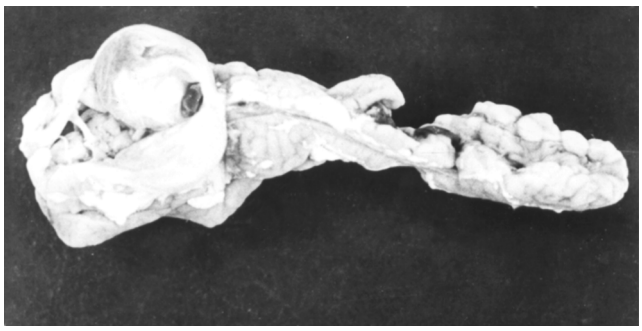

Fig. 5. A neoplasm on a kidney of a 7-month-old $F_4(1.1^3)$ rooster, one of the offspring of the Russian white chicken damaged by ^{131}I (the experiment was carried out under vivarium conditions). Hematoxylin and eosine stain, magnification ×400. The daily dose of ^{131}I received by the parents was 1.1 MBq/kg; F_1 chickens appeared 3 months after the beginning of the exposure to ^{131}I.

Heavy plethora, cellular dissociation and epithalaxia of canaliculi, clusters of young reticular cells between the canaliculi were found during a histological investigation. In case of severe kidney lesions, these cells almost completely substituted the renal parenchyma, which indicated reticulosis. Such macro- and microscopic changes in kidneys were observed once in $F_1(1.1^3)$, two times in $F_1(1.1^{12})$, once in $F_1(2.1^{12})$, two times in $F_2(1.1^{12})$, once in F_2 (1.1^{12}), three times in $F_3(1.1^3)$, once in $F_3(1.1^{12})$, and once in $F_4(1.1^3)$.

Morphological changes in the thyroid gland were noted in two experimental chickens from $F_1(2.1^{12})$ and $F_2(1.1^3)$ groups. The thyroid glands were enlarged, torose, and anisochromatic. A histological investigation revealed a reduced number of follicles and the parenchyma of the gland substituted with lymphoid and reticular cells.

The changes in the morphology of the ovaries were noted once in each of the $F_1(1.1^3)$, $F_1(2,1^{12})$, and $F_2(1.1^{12})$ groups and twice in $F_2(1.1^3)$ group. The

ovaries of these chickens had the appearance of a cauliflower, with yolk spherules colored from white to black, often with hemorrhages. A histological investigation revealed a curved structure of the organ, with aggregations of lymphoid and reticular cellular elements in the parenchyma.

Numerous yellowish nodules having elastic consistency, pea- to walnut-size, were found on the mesentery and intestine of two experimental chickens in $F_1(1.1^3)$ and $F_2(1.1^3)$ groups. A microscopic examination of the tumor-like growths revealed aggregations of reticular cells.

During an autopsy, a yellowish lump having elastic consistency, lard-like if viewed in cross section, hazelnut-size, was found on a lung of a 7-month-old $F_2(1.1^3)$ hen. A histological investigation revealed that the cellular composition of the neoplasm was inhomogeneous. Among the lymphoid and reticular cells, the lymphoid cells prevailed, with stromal overgrowth around the organ; the cytolysis was marked. The stated features indicated that it was lympho-reticulosarcoma.

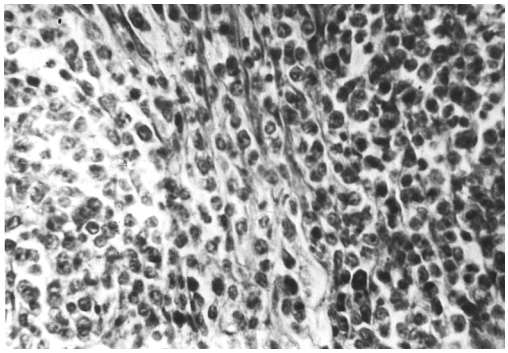

Fig. 6. A microphotograph of a tumor on the back of a 7-month-old experimental $F_2(1.1^3)$ rooster, a one of the offspring of the Russian white chickens damaged by ^{131}I (proliferation of reticular cells is observed; the experiment was carried out under vivarium conditions). Hematoxylin and eosine staining, magnification ×400. The daily dose of ^{131}I received by the parents was 1.1 MBq/kg; F_1 chickens appeared 3 months after the beginning of the exposure to ^{131}I

The tumors in the thoracic cavity, the cellular composition of which was suggestive of reticulosarcoma, were found in two experimental offspring chickens. A $F_2(1.1^{12})$ hen had a tumor the size of a chicken egg, of firm consistency, stratified if viewed in cross section, which was located next to the right lung and had grown into the intercostal muscles. Most of the thoracic cavity of a $F_3(1.1^{12})$ hen was occupied by a tumor-like growth.

In one case, a rounded tumor the size of a chicken egg having elastic consistency, lard-like if viewed in cross section, was found on the back of a 7-month-old experimental rooster from $F_2(1.1^3)$ group under the skin between the wings. A microscopic examination revealed that its tissue was distinctly vascularized, a marked stroma surrounded aggregations of immature reticular cells. The structure of the tumor tissue was similar to reticulosarcoma (Fig. 6).

The offspring survival rate of ^{131}I radiation-exposed chickens was decreased (Table 1). The highest mortality rate among the experimental chickens was noted during the 1st month of their life. By the end of the 1st month, in groups 0.11^{12}; 1.1^3; 1.1^6; 1.1^{12}; 2.1^{12}, and 4.6^3 of generation F_1 the number of died chickens was 10.2 %, 7.0 %, 7.8 %, 7.7 %, 41.5 %, and 45.5 % higher than in the control groups.

Decreased survival trend among the experimental F_1 chickens continued into the older ages; however, much fewer chickens in the groups 0.11^3; 1.1^3; 1.1^{12}; 2.1^2 died than in the 1st month of their life. By the age of 18 months, the differences in the survival rates among the experimental and control birds in these groups increased by < 3 % as compared with the corresponding indicators for 1-month-old chickens. In group 4.6^3, the mortality rate among the experimental chickens increased progressively with their age and all of them died by the end of the 2nd month of their life.

1. Age-related dynamics of the survival rate (%) in the offspring generations of the Russian white chickens subjected to experimental exposure to ^{131}I in different doses (experiment under vivarium conditions)

Age	Group	F_1	F_2	F_3	F_4	F_5	F_6
1 month	0.11^{12}	69.8	78.0	76.5	–	–	–
	Control	78.0	81.1	78.0	–	–	–
	1.1^{3}	79.0	78.8	76.8	52.3	71.4	72.3
	Control	86.0	84.7	87.2	71.2	81.7	80.3
	1.1^{6}	72.2	–	–	–	–	–
	Control	80.0	–	–	–	–	–
	1.1^{12}	83.4	76.9	57.7	81.7	69.8	–
	Control	91.1	93.7	74.3	85.3	78.0	–
	2.1^{12}	36.5	59.6	–	–	–	–
	Control	78.0	81.1	–	–	–	–
	4.6^{3}	32.5	–	–	–	–	–
	Control	78.0	–	–	–	–	–
2 months	0.11^{12}	65.6	69.2	74.3	–	–	–
	Control	75.0	79.2	73.8	–	–	–
	1.1^{3}	77.2	75.9	71.6	51.2	66.9	47.9
	Control	82.0	83.6	83.9	69.4	81.2	67.9
	1.1^{12}	81.4	73.5	50.8	75.5	60.3	–
	Control	90.0	90.6	67.3	83.2	73.8	–
	2.1^{12}	32.4	59.6	–	–	–	–
	Control	75.0	79.2	–	–	–	–
3 months	0.11^{12}	65.6	69.2	72.0	–	–	–
	Control	75.0	79.2	67.7	–	–	–
	1.1^{3}	77.2	74.6	70.5	50.0	66.9	47.9
	Control	81.3	82.6	82.6	69.4	80.3	67.9
	1.1^{12}	80.7	73.5	49.2	75.5	60.3	–
	Control	90.0	89.1	65.3	82.7	67.7	–
	2.1^{12}	32.4	59.6	–	–	–	–
	Control	75.0	79.2	–	–	–	–
6 months	0.11^{12}	63.4	69.2	68.2	–	–	–
	Control	73.5	78.3	67.1	–	–	–
	1.1^{3}	74.6	67.0	69.5	46.5	66.9	46.9
	Control	81.3	80.5	81.7	67.6	79.8	67.4
	1.1^{12}	75.8	72.7	48.5	74.7	60.7	–
	Control	88.9	87.5	65.3	82.3	67.1	–
	2.1^{12}	32.4	59.6	–	–	–	–
	Control	73.5	78.3	–	–	–	–
12 months	0.11^{12}	63.4	68.9	68.2	–	–	–
	Control	72.0	78.3	67.1	–	–	–
	1.1^{3}	71.9	60.3	64.2	42.0	66.9	46.9
	Control	78.6	77.8	79.0	64.7	79.4	65.8
	1.1^{12}	71.8	64.9	43.8	72.2	60.3	–
	Control	84.4	86.7	65.3	81.2	67.1	–
	2.1^{12}	29.6	–	–	–	–	–
	Control	72.0	–	–	–	–	–
18 months	0.11^{12}	61.2	68.9	68.2	–	–	–
	Control	70.5	78.3	67.1	–	–	–
	1.1^{3}	67.6	55.5	59.9	33.6	65.7	46.9
	Control	77.3	76.7	78.5	62.2	78.9	46.8
	1.1^{12}	68.9	57.3	41.5	72.2	60.3	–
	Control	84.4	83.6	63.4	80.7	67.1	–
	2.1^{12}	28.2	–	–	–	–	–
	Control	70.5	–	–	–	–	–
24 months	1.1^{3}	66.7	51.7	57.4	–	–	–
	Control	75.9	75.1	78.5	–	–	–
	1.1^{12}	64.0	52.3	–	–	–	–
	Control	84.4	82.3	–	–	–	–
30 months	1.1^{3}	66.7	50.0	–	–	–	–
	Control	75.9	75.1	–	–	–	–
	1.1^{12}	62.6	–	–	–	–	–
	Control	82.2	–	–	–	–	–

N o t e : Column «Group» shows a daily dose of ^{131}I (MBq/kg), received by the parents. The superscript indicates the number of months after the beginning of ^{131}I administration when F_1 chickens appeared. The dashes mean that the investigation was not performed.

The next generations of the experimental birds demonstrated decreased survival rates too. By the age of 6 months, the survival rate of F_2 chickens in groups 0.11^{12}; 1.1^{3}; 1.1^{12} and 2.1^{12} was lower than in the control groups by 9.1 %, 13.5 %, 14.8 %, and 17.7 % correspondingly. A similar survival dynamics in the experi-

mental groups was observed in generations 3-6 too.

Weak newborn chickens, which mainly died in the 1st month of their life, represented the largest share in the total number of deaths among the control and experimental offspring.

2. Frequency of lethal outcomes depending on their causes in the generations of the Russian white chicken offspring subjected to experimental exposure to ^{131}I in different doses (experiment under vivarium conditions)

Generation	Group	Total died	Born weak	With disorders		
				of gastrointestinal tract, liver	heart	kidneys
F_1	0.11^{12}	36	29/80.6	5/13.9	2/5.5	–
	Control	20	17/85.0	3/15.0	–	–
	1.1^{3}	39	24/61.5	14/36.0	1/2.5	–
	Control	36	21/58.3	14/39.0	1/2.7	–
	1.1^{12}	54	24/44.5	25/46.3	3/5.5	2/3.7
	Control	16	8/50.0	7/43.7	1/6.6	–
	2.1^{12}	51	45/88.4	4/7.8	2/3.8	–
	Control	20	17/85.0	3/15.0	–	–
	4.6^{3}	28	28/100	–	–	–
	Control	20	17/85.0	3/15.0	–	–
F_2	0.11^{12}	23	17/74.0	6/26.0	–	–
	Control	23	20/87.0	3/13.0	–	–
	1.1^{3}	118	50/42.3	58/49.0	2/1.8	–
	Control	47	29/62.0	16/34.0	–	–
	1.1^{12}	50	27/54.0	20/40.0	2/4.0	–
	Control	23	8/34.8	15/65.2	–	–
	2.1^{12}	23	22/95.5	–	1/4.5	–
	Control	23	20/87.0	3/13.0	–	–
F_3	0.11^{12}	42	31/74.0	10/23.7	1/2.3	–
	Control	66	38/57.5	27/41.0	1/1.5	–
	1.1^{3}	81	44/54.5	33/40.7	2/2.4	2/2.4
	Control	40	24/60.0	15/37.5	1/2.5	–
	1.1^{12}	76	55/72.4	20/26.3	1/1.3	–
	Control	37	26/70.2	11/29.8	–	–
F_4	1.1^{3}	57	41/72.0	15/26.3	1/1.7	–
	Control	42	32/76.2	10/23.8	–	–
	1.1^{12}	76	50/65.8	24/31.6	2/2.6	–
	Control	38	29/76.4	8/21.0	1/2.6	–
F_5	1.1^{3}	46	39/84.8	6/13.0	1/2.2	–
	Control	46	40/87.0	6/13.0	–	–
	1.1^{12}	25	16/64.0	8/32.0	1/4.0	–
	Control	54	37/68.6	15/27.8	2/3.6	–
F_6	1.1^{3}	50	26/52.0	22/44.0	1/2.0	1/2.0
	Control	66	38/57.5	27/41.0	1/1.5	–

N o t e : Column «Group» shows a daily dose of ^{131}I (MBq/kg), received by the parents. The superscript indicates the number of months after the beginning of ^{131}I administration when F_1 chickens appeared. Before the slash—an absolute value, after the slash a percentage of the total number of died birds is indicated. The dashes mean no registered cases of bird death.

The percentage of died young chicken in F_4 and F_5 in relation to F_1 increased both in the experimental and control groups.

The main reason of the lethal outcomes in the offspring of ^{131}I radiation-exposed chickens was the fact that they were born weak; the next one was disorders of gastrointestinal tract, liver, heart and kidneys. After the 6th month, tumor-like damages developed in the soft tissues of the bodies of the experimental offspring. With respect to the total number of chickens, the frequency of such tumors was 1.1 % in $F_1(0.11^{12})$, 3.5 % in $F_1(1.1^{3})$, 3.4 % in $F_1(1.1^{12})$, 3.8 % in $F_1(2.1^{12})$, 4.7 % in $F_2(1.1^{3})$, 3.4 % in $F_2(1.1^{12})$, 1.4 % in $F_2(2.1^{12})$, 1.0 % in $F_3(1.1^{3})$, 0.7 % in $F_3(1.1^{12})$, and 0.8 % in $F_4(1.1^{12})$. On average, the percentage of neoplasms of internal organs in the 1st generation offspring, which hatched 3 months after the beginning of the parents' exposure to ^{131}I in the dose of 1.1 MBq/kg, was 3.3 %, for the 2nd generation, which hatched 12 months after the exposure, it was 2.9 %. The tumors were located in skeletal muscles, thoracic and abdominal cavity organs. In their structure, the tumors were similar to lymphoreticulosarcoma or reticulosarcoma. Carcinogenic processes seemed to

become one of the factors reducing survival rates of the experimental birds.

The mortality rate among the young birds depended on the dose of ^{131}I received by the parents, i.e. higher doses led to more deaths among the chickens than the lower ones. No dose-related distribution of frequency of gastrointestinal tract, liver, heart and kidney disorders was noted. No renal lesions or neoplasms were encountered in the control groups.

Our results correlate with experimental data obtained on rats exposed to ^{131}I during pregnancy [20]. Physical growth and development disorders, increase in the frequency of tumors in various locations, damage to the genetic material in the germ cells were found to be possible in the offspring born from the carriers of ^{131}I. The scientists accept that the main cause of these phenomena is ovocyte, embryo and fetus damage induced by the direct effect of radiation. The obtained data also correlate with the results of the investigation involving the offspring of people who were exposed in the Eastern-Ural and Chernobyl traces [21-26]. The analysis of the consequences of accidental and professional exposure shows that prolonged radiation exposure may result in reproductive dysfunctions, i.e. increase in the frequency of adverse pregnancy outcomes, delays in and disorders of physical development of born offspring, high mortality rate and cancer morbidity [27-33].

In the XXI century, there are much more adverse effects of a range of human activities, including ionizing radiation, on the human body and reproductive health [34, 35]. One of the challenges the scientific community is facing is a comprehensive unbiased assessment of this serious threat, scientific justification and implementation of the most efficient measures aimed at prevention and maximum mitigation of the adverse effects of radiation, which could be passed down to the next generations [36, 37].

Thus, a number of development abnormalities was found in the offspring of the chickens subjected to a chronic exposure to ^{131}I in the doses from 0.11 to 4.6 MBq/kg. These disorders manifested themselves as decreased postnatal survival rates, reduction of body weight, congenital abnormalities, and more frequent neoplasms. An increased mortality among the offspring of ^{131}I radiation-exposed chickens seems to be connected with the changes taking place during embryogenesis and resulting in improper regulation and adaptation in the early postnatal period. The main causes of death among the offspring of the ^{131}I radiation-exposed chickens were disorders of gastrointestinal tract, liver, heart and kidneys. Tumors in the soft parts of the body were found in F_1-F_4 offspring aged 6 months or older. In their structure, the tumors were similar to lymphoreticulosarcoma or reticulosarcoma.

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