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EFFECTS OF RADIATION ON INTRAUTERINE ON DEVELOPMENT IN MAMMALS.

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Abstract

This study examines the effects of radiation on wild vertebrate populations living in polluted environments. "Radiation impacts on biota" is a European Commission Project EPIC database. The compilation of data, based on Russian publications, shows the effects of radiation in high-radionuclide regions. The data range from acute accident-caused irradiation to lifelong exposures with modest dose rates. Radiation affects death, reproduction, health, ecology, cytogenetics, and the ability to adapt to radiation, among other things. The unique radiation impacts of different radionuclides and the severity of their effects on different species are discussed.

Keywords: *Radiation , mammals*

Introduction

Ionizing radiation has had widespread use in the fields of medicine and crystallography ever since Rontgen's discovery of X-ray radiation in 1895. However, in the years after its discovery, a growing number of studies suggested that exposure to ionising radiation might be extremely detrimental to living things. We now have a fundamental comprehension of the manner in which ionising radiation influences the cells of living organisms and are aware that not all cells exhibit the same level of sensitivity to radiation.

Lesions in the DNA can be caused directly or indirectly by ionising radiation thanks to the generation of hydroxyl free radicals. Lesions that are induced in DNA include double strand breaks (DSBs), single strand breaks (SSBs), and clusters of DNA lesions. A cluster of DNA lesions is defined as two or more individual lesions occurring within one or two helical turns of the DNA. DSBs and SSBs are the types of lesions that are induced. Both double-strand breaks (DSBs) and clusters of DNA lesions are regarded as being extremely harmful for all cell types. Irradiation may cause DNA to break, which triggers a cascade of biological reactions, the first of which is the recognition of DNA damage and a pause in the cell cycle. It is generally agreed upon that the ineffective repair of DNA lesions is a crucial event in the chain of events that leads to mutagenesis, genomic instability, and cell death.

According to the rule of Bergonie and Tribondeau, the radiosensitivity of a tissue is directly proportional to the number of undifferentiated cells present in the tissue, as well as the mitotic activity of those cells and the amount of time such cells spend in the proliferative phase. This fundamental law of radiation biology applies, as well, to embryos while they are in the process of development. Radiation that is ionising may frequently have a devastating effect on embryos that are in their earliest stages. Even while embryos are, on average, quite susceptible to radiation, the degree to which they are sensitive varies across the many phases of embryonic development. Russell and Russell (1954) provided a condensed review of the effects of radiation on human and rodent embryos and foetuses. They demonstrated that there are three distinct phases of differential radiation sensitivity during the development of both mice and humans:

preimplantation, organogenesis, and foetus. The morphological changes that are linked with radiation exposure can be used to date each epoch. Embryos that are irradiated during the preimplantation period, also known as the period before implantation, are subject to the "all-or-none" rule. This means that while some irradiated embryos are able to develop normally if they make it through the gestation period, others either perish or are unable to implant. Irradiated embryos display the most striking phenotypes, which include aberrant head formation and temporarily tiny body size. This happens because organogenesis is the stage of embryonic development in which the primary organs grow. Some cases exhibit signs of mental impairment and microcephaly, however this might vary depending on the amount of radiation exposure. Exposure to ionising radiation during the foetal phase produces persistent growth retardation in mice and rats. The foetal period is characterised by growth, and exposure to ionising radiation during this period causes lasting growth retardation. Irradiation during pregnancy has been found to cause mental impairment in certain unborn human children.

Recent research has focused on analysing the specific cellular and molecular mechanisms that are responsible for the response to ionising radiation during the preimplantation period. This research may explain the differences in sensitivity and response to ionising radiation that occur between each developmental period. We will focus on the variation in sensitivity and response to radiation during the preimplantation (before gastrulation) period and discuss the underlying cellular and molecular mechanisms in this review. Given that there are detailed reviews about the mechanisms of the response to ionising radiation during the organogenesis and foetus periods, we will focus on the preimplantation (before gastrulation) period. Embryos that are in this stage have a quality known as totipotency, and the absence of cellular responses is directly tied to the presence of this quality. It is also essential to point out in this context that the rate at which certain biological systems respond to ionising radiation might vary over time depending on the species. In order to shed light on the essential processes of totipotency or pluripotency, it is necessary to conduct a review of the cellular mechanisms that were active throughout this phase of each model organism.

Mammals

Experiments carried out on mice have been the primary source of information on the effects of radiation exposure at the preimplantation stage. Radiation exposure during this time period often results in the death of animals. Only particular strains of mice, which will be discussed in more detail below, display the defects. Embryos are at their most resistant during the S phase of the first cell cycle, which occurs between 2 and 6 hours post-fertilization (hpf). The early pronuclear stage is when the level of sensitivity is at its peak. In the days that immediately follow this period, sensitivity experiences a precipitous decline. During this time frame, the LD50 value shifts from 1.5 Gy (immediately following sperm entrance) to 0.3 Gy (4–6 hpf), and then progressively increases to around 3.5 Gy by the 5th day after fertilisation (dpf). The sensitivity, on the other hand, appears to change quite a bit, most likely depending on the time of the irradiation in relation to the stage of the cell cycle. Both the 'Heiligenberger Stamm' (also known as HLG) and the CF1 mouse strain exhibit malformations as a result of exposure to ionising radiation. Irradiated embryos of these strains, although still in the preimplantation stage, develop malformations known as gastroschisis, exencephaly, and polydactyly.

Effects of space radiation on mammalian cells

As was noted in the introduction, the radiation that astronauts are exposed to in space is composed of GCR and SPE. Of these two types of radiation, heavy ions in particular are anticipated to have a significant detrimental effect on the astronauts' health. It's possible that erecting an effective barrier will appear to be the simplest and most apparent answer. However, given the technology that is available now, passive shielding is only effective for SPE, and its use for GCR is restricted because of the stringent mass limits that are present in spaceflight. Consequently, techniques based on genetics and biomedicine. When thinking about the biological impacts of ionising radiation, it is imperative not to mix the developing embryo with the adult that will eventually emerge from that embryo (RUGH 1962). Both in terms of intensity and nature, the responses of the two are completely distinct from one another. Regardless of the test that is performed, every stage of embryonic or foetal development has a greater radiosensitivity than the post-natal organism has. In the early stages, shortly after fertilisation, lethality is at its highest; nevertheless, congenital abnormalities are more likely to emerge after roentgen irradiation in the later stages (Rugh 1959, 1961, 1962; RUGH & GRUPP 1959). Still later, right before delivery, neither fatality nor congenital consequences are apparent; rather, the foetus responds by developing functional sequelae, which may not completely appear for several years after the exposure. Additionally, it is important to keep in mind that after approximately five months of human development, the gonads develop and contain primitive germ cells that are themselves very radiosensitive. These cells not only accumulate the harmful effects of radiations in the form of genic mutations, but they also pass them along unchanged to their offspring. We have a tendency to overlook that when dealing with ionising radiations, we are dealing with a physical entity when we consider the fact that the embryo and foetus are so radiosensitive.

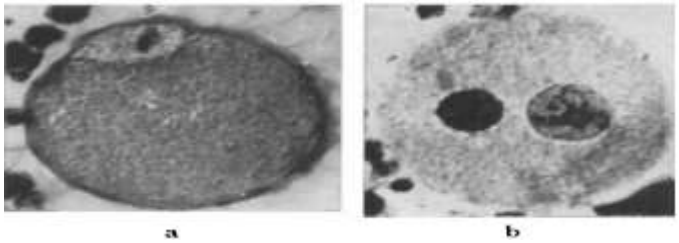

Fig. 1. a) Fertilizing mouse egg with spermatozoon. Roentgen irradiation is like exposing both gametes separately. This stage is very sensitive to roentgen irradiation, causing death. b) Effect of roentgen irradiation on zygote before pronuclear fusion. 15 r exposure hyperchromatic male pronucleus.

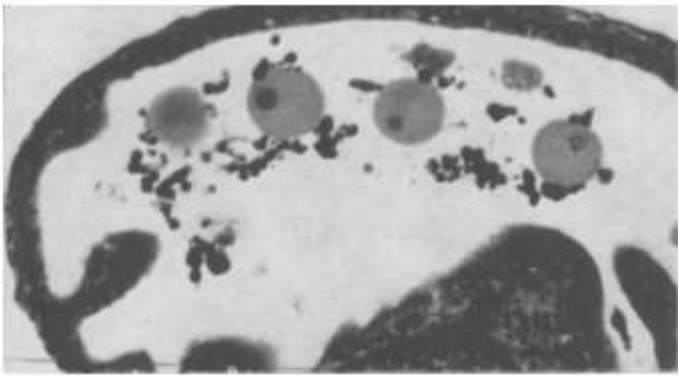

Fig. 2. Shortly after mating, a mouse's oviduct shows four fertilised eggs..

Stunning. 1 molecule in 10 million (1 000 r) will kill most adult animals, and 1 molecule in 1 billion (10 r) will induce lymphopenia in adults and chromosomal abnormalities and congenital malformations in embryos. Embryos and foetuses are more radiosensitive than adults to powerful ionising radiations. Roentgen irradiation of the embryo at the start of neurogenesis causes the most gross CNS abnormalities (HICKS et coll. 1952, 1953, 1954, 1959). This phase is 7.5 to 9.5 days for rodents and 18 to 25 days for humans. Any trauma would certainly produce these aberrations at this time, but ionising radiations are so penetrating and immediate that 48% of the population is affected.

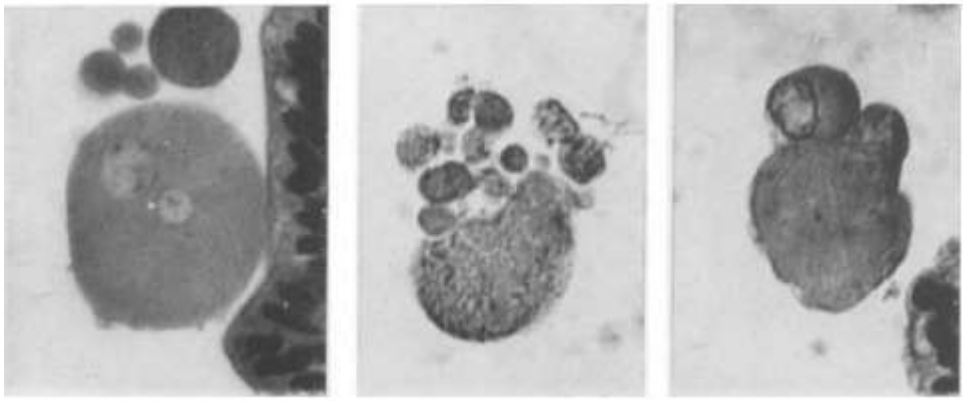

Fig. 3. a) Fig. 2 shows 15 r fragmentation of a fertilised egg. Pinched cytoplasm retains both pronuclei. 15 r exposure completely fragments zygote. c) Exposed zygote pinching off cytoplasm, but pronuclei remain. Without nuclei, the remaining cytoplasmic bulk died. These impacts of 15 r are statistically rare.

Exencephaly develops in 8.5-day-old mouse embryos (RUGH & GRUPP 1959, 1961, 1962). After organogenesis, ionising radiation cannot cause gross congenital abnormalities. Some abnormalities can be produced, but they damage the brain's cyt-architecture. Exposures far lower than 200 r are now considered to have functional consequences, not usually at birth. There is evidence that behaviour impacts, detectable by electroencephalography, and potentially other mental disorders may follow even low-level foetal exposure years later. Before neurogenesis began at 7.5 days in the mouse embryo, it was thought ionising radiations couldn't cause congenital sequelae (RUSSELL 1950, 1956, 1957 ; RUSSELL & RUSSELL 1956). High irradiation levels destroyed most early embryos. A dosage that kills off the majority of early embryos might lower the occurrence of congenital abnormalities. As little as 10 r to a 2-cell mouse embryo can induce cerebral hernia (exencephalia). True, it's rare, but the fact that it happens in a type of mice where it's never been observed shows that 10 r may not be the threshold dose for congenital consequences. (3 in 18 000 non-irradiated control embryos had this abnormality).

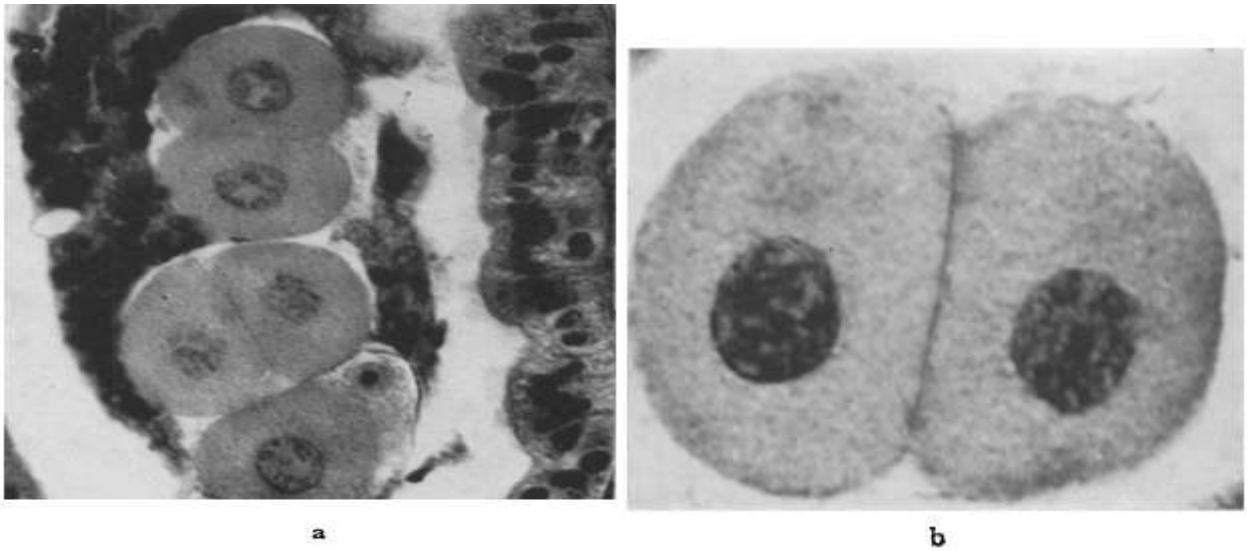

Fig. 4. a) One 2-cell mouse embryo has a single blastomere but a persisting polar body. It's also radiosensitive. b) 15 r exposure at (a) stage; hyperchromatic nucleus.

A developmental disorder known as exencephalia, also known as brain hernia, hinders the closure of the neural folds at the mid-brain region. As a result, the cranial roof does not form correctly, and the brain protrudes through the roof of the skull. This condition, along with the associated anencephaly, occurs in around one in five hundred cases of human births, but it is something that can be created consistently in rodent embryos by irradiating them with roentgen at appropriate stages. At this point in time, it is only possible to speculate as to whether or not protracted diagnostic irradiation or even background irradiation might be the root cause of this aberration in human births. However, it is necessary to posit that the radiosensitivity of the human embryo is probably quite comparable to that of the embryo of a rat. Even if we are unable to extrapolate from the rodent to the human, we should still consider the results obtained from rodents to be suggestive. These assertions have been made on the basis of the data obtained from the examination of over 60,000 mouse embryos and foetuses just before and after they were born. It just so happens that when a mouse gives birth to a child with a congenital abnormality, the mouse will immediately kill and consume the child. As a result, in order to collect accurate statistical data, it is necessary to examine the majority of these foetuses just prior to the time when the baby is expected to be born. Studies are now being conducted with the goal of gaining a deeper comprehension of the relationship between embryonic or foetal exposure and a wide array of congenital abnormalities. However, it appears very certain that the research will have to be directed to the levels of electron microscopy for structural effects, as well as to the electroencephalogram, the electroretinogram, and conventional behaviour tests, in order to figure out whether or not the impacts have been structural.

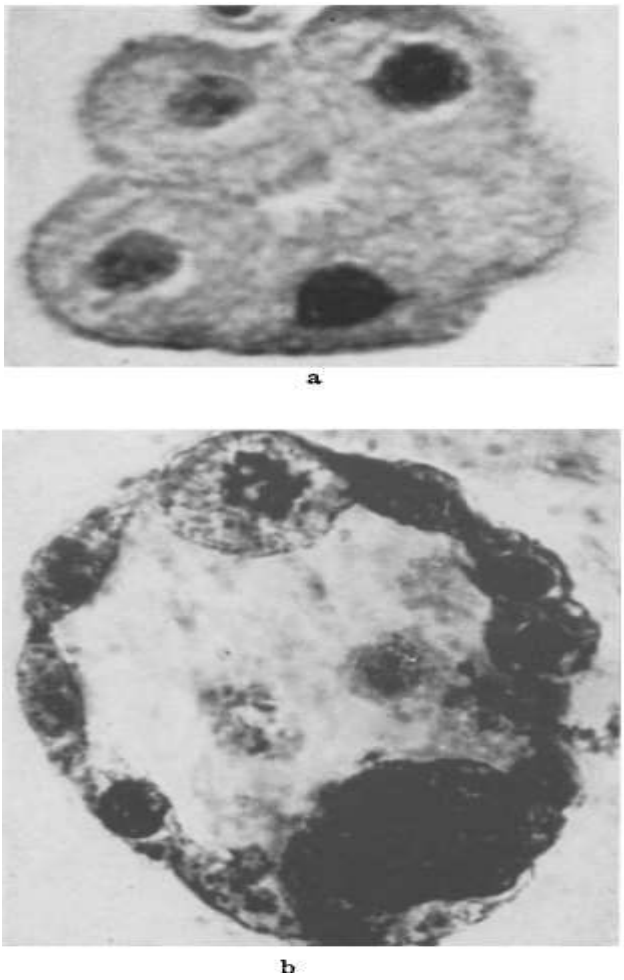

Fig. 5. Roentgen treated to 15 r at the two-cell stage, but showing cleavage with hyperchromatic nuclei. Four cell stage. This embryo would most likely pass away in the near future. b) This blastula was exposed at the 2-cell stage, and when it was examined after three days, it had a giant cell that had extra chromosomes. This, along with vacuolization, is a common sequelae to the early insult caused by roentgen irradiation.

impacts that were not picked up by the histopathology. In addition, we are compelled to reduce the degree of exposure, and we are now researching the effect of a single dose of 1, 5, and 10 r over the course of each and every 24 hour period from the time of fertilisation until 18.5 days into the pregnancy. The immediate and long-term consequences, including infertility, leukaemia, cataracts, ageing, and the occurrence of a variety of malignancies, of exposing embryos and foetuses in this way are currently the subject of research. Embryos and foetuses are being examined. Radiology for diagnostic purposes is on par with scalpels in terms of their importance to the practise of medicine. Ionizing radiations, when employed for non-threatening purposes, have advanced civilization more quickly than any other single physical help that is known to man, and they will continue to do so in the future. Irradiation with low doses of roentgen at therapeutic levels is responsible for the daily saving of the lives of thousands of people. In light of the fact that ionising radiations are capable of inflicting such severe damage on the developing embryo and foetus, it is impossible to find any basis for condemning them. Instead, the physician should be made aware of these results and strongly encouraged to take precautions to safeguard the foetus in the event that pelvic irradiation is necessary. Further, because the human embryo is probably most radiosensitive at a time when

a pregnancy is least suspected, specifically between 15 and 42 days after the onset of menstruation, it is rapidly becoming an accepted practise to limit extensive radiodiagnosis of the female pelvis to the first nine days following the onset of menstruation in order to avoid the earliest stages of an unsuspected pregnancy. This is done in order to protect the unborn child from being exposed to potentially harmful radiation. By proceeding in this manner, we stand a chance of gradually lowering the 5!h anticipated incidence of congenital abnormalities in human births.

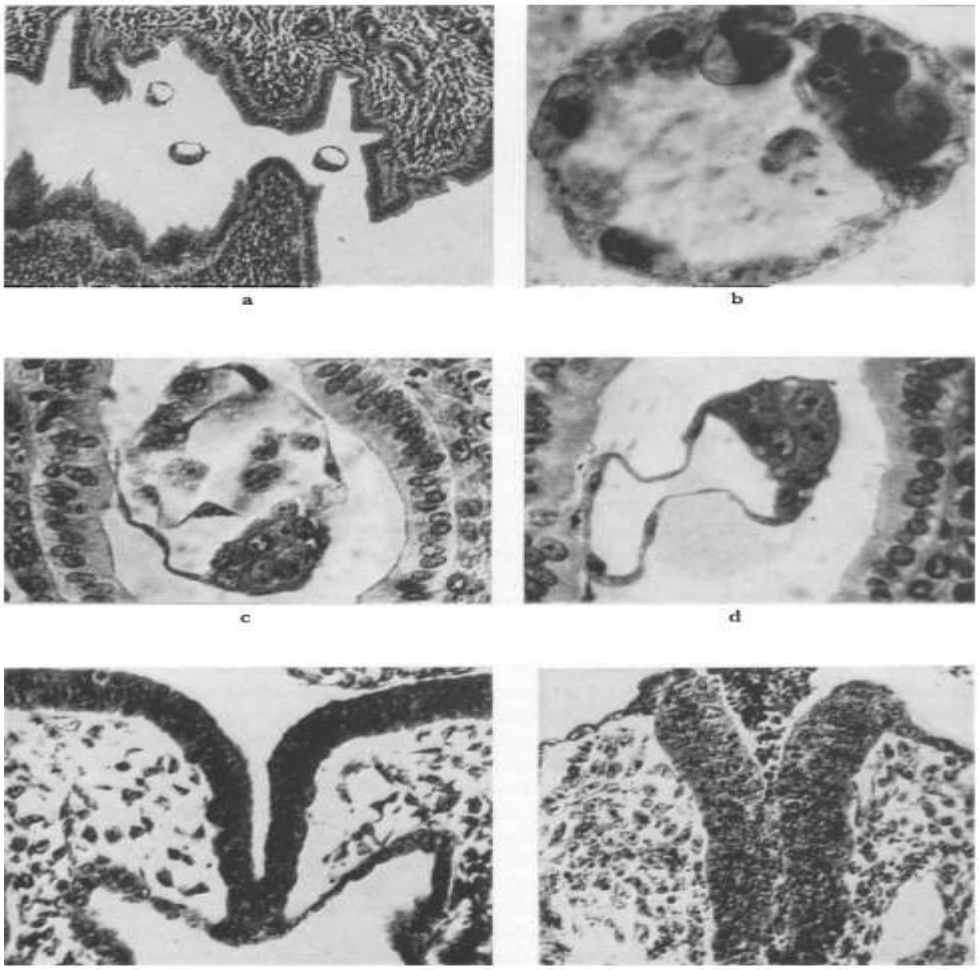

Fig. 6. For legend, see opposite page.

Fig. 7. When 3.5-day-old mouse embryos are exposed to 50 r and survive, they may develop severe cephalic anomalies. The damage is mainly to the CNS and anterior, but there are also kidney, heart, etc. anomalies. These people would die.

Fig. 8. At 18.5 days, 5 of 11 mice in a bicornuate mouse uterus had exencephalia, or brain hernia. Exposure to 200 r at 8.5 days of development, when CNS neuroblasts were abundant, caused this.

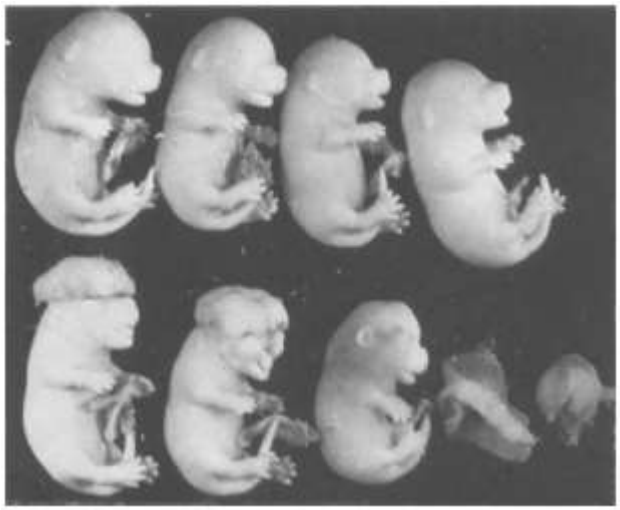

Fig. 9. Heterozygosity protects adult mice, but not embryos. This litter of hybrid mice (CFl x C57) exposed at 8.5 days to 200 r shows stunting, death, and resorption. Exencephaly, gross stunting are intermediate effects.

Fig. 10. Female mouse exposed to 100 r and bred to a normal male causes hereditary exencephalia. This rare, genetic anomaly appeared in three generations.

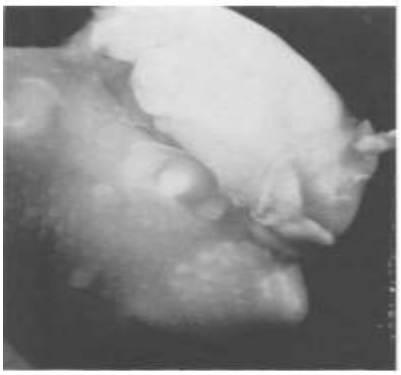

Fig. 11. Exencephalia brain hernia caused by interrupted brain and cranial roof development. The head and eye appear balanced.

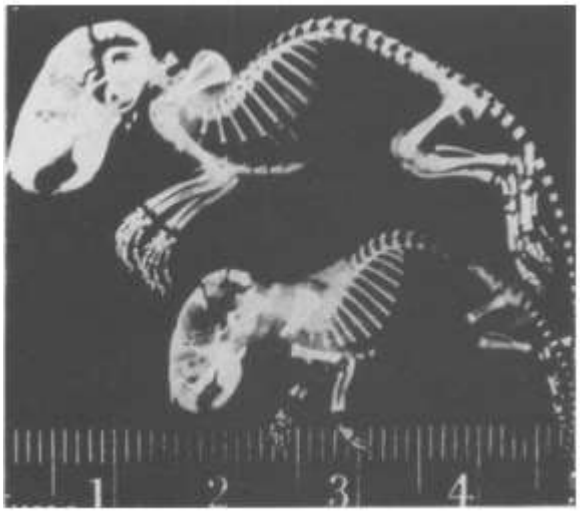

Fig. 12. Roentgen views of a normal (above) and fetally irradiated (below) mouse skeleton at birth show topographical normality but extreme stunting in the exposed mouse. Due to early cell loss, the embryo uses the remaining cells to develop a well-balanced, though smaller, individual.

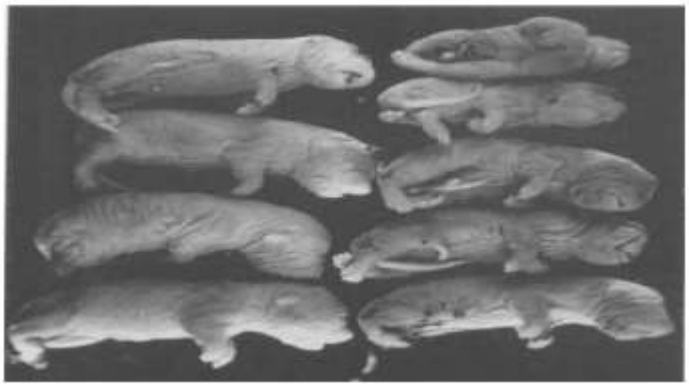

Fig. 13. At 9.5 days embryonic development, 100 r exposed an entire litter of rats. The number of anomalies in a single roentgen-irradiated litter is striking..

Fig. 14. Fig. 13 shows enlarged views of a few littermates with substantial CNS abnormalities.

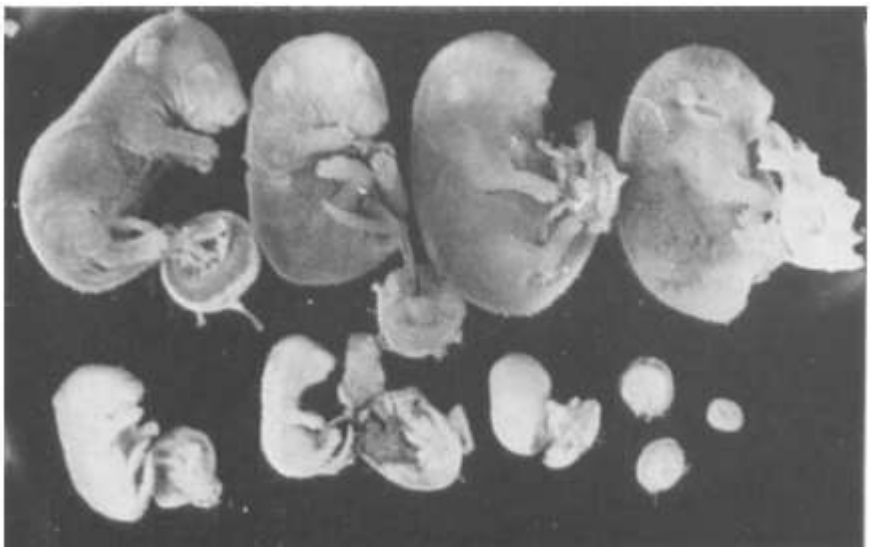

Fig. 15. Entire litter of rat embryos just before delivery, showing death and resorption of three dead foetuses and the remaining foetuses looking more like mouse embryos.

Fig. 16. Hemi-sected skulls of two rats at 2 weeks old show (above) normal brain and (below) collapsed brain of hydrocephalus individual.

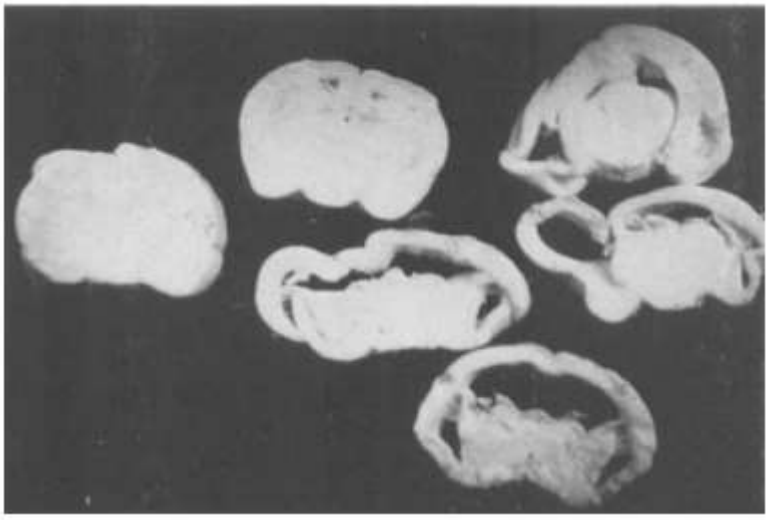

Fig. 17. Transverse sections of 2-month-old rat brains. The left control and five littermates with hydrocephalus are shown..

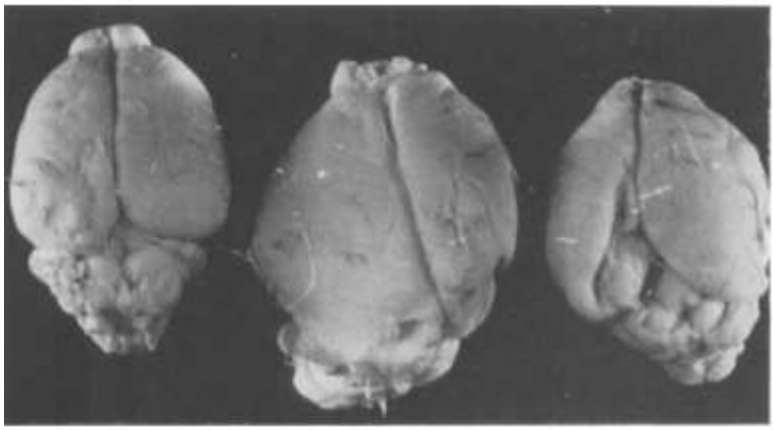

Fig. 18. Hydrocephalic rats treated with 100 r at 9.5 days gestation (left) and control (right). These irradiated brains are distorted.

Conclusion

It has been found that the mammalian embryo shows varying reactions to ionizing radiations such that it is always more radiosensitive than is the adult into which it develops, at some stages 100 times as sensitive. After neurogenesis is completed, congenital anomalies of that system are rarely- produced, but exencephaly (brain hernia) has been caused by ionizing radiations impinging upon the 2-cell stage of the mouse with an exposure of as little as 10 r.

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