[CD44 gene expression in cancerous thyroid cells]

Abstract: The peculiarities of alternative CD44 mRNA splicing in thyroid cancer tissue of children from radiocontaminated areas was investigated. CD44 gene expression in thyroid cancer tissues of children exposed to radiation resembled that in spontaneously emerged cancers. It was concluded that CD44 gene expression is not the primary target of radioactive irradiation. Probably, the CD44 mRNA splicing deregulation is the consequence of cancer.
Differential diagnosis of hyperthyroidism syndrome using a highly sensitive method of determining TSH level in blood: clinical review and recommendations on use in Russia and the USA.

https://arctichealth.org/en/permalink/ahliterature217430

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<th>Author</th>
<th>J. Figge</th>
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<td>PubMed ID</td>
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Genetic aberrations in Chernobyl-related thyroid cancers: implications for possible future nuclear accidents or nuclear attacks.

https://arctichealth.org/en/permalink/ahliterature17932

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Adolescent
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Child
Chromosome Aberrations - radiation effects
Comparative Study
Humans
Mutation - radiation effects
Neoplasms, Radiation-Induced - diagnosis - epidemiology - genetics
Nuclear Warfare
Radioactive fallout
Thyroid Neoplasms - diagnosis - epidemiology - etiology - genetics
Ukraine

Abstract: Cases of thyroid cancer among children in Belarus represent a unique model system in which the cause of the cancer is known—radiation. Although other sources of radiation-induced cancers are diminishing (survivors of Hiroshima and Nagasaki, and individuals exposed to diagnostic or therapeutic radiation) fears of radiation exposure from accidents and terrorism are increasing. Our analysis of current data reveals that Chernobyl-related cancer cases might have a specific pattern of genetic aberrations. These data strongly confirm the hypothesis that radiation-induced cancers might arise as a result of specific gene aberrations that are distinct from those in sporadic cancers, suggesting that methods of prevention and treatment of radiation-induced cancers might require a different approach. Understanding of the molecular mechanisms of Chernobyl-related papillary thyroid carcinomas will help to identify mechanisms by which radiation causes aberrations and oncogenic cell transformation. Thus, in turn, it will be important in the development of new treatments or technologies to minimize the effects of radiation damage from nuclear accidents or nuclear attacks.

PubMed ID: 14768999 View in PubMed
Low prevalence of the ret/PTC3r1 rearrangement in a series of papillary thyroid carcinomas presenting in Belarus ten years post-Chernobyl.

https://arctichealth.org/en/permalink/ahliterature21341

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Keywords: Accidents, Radiation
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Byelarus
Carcinoma, Papillary - genetics
Drosophila Proteins
Female
Gene Frequency
Gene Rearrangement - genetics
Humans
Male
Molecular Sequence Data
Power Plants
Proto-Oncogene Proteins - genetics
Proto-Oncogene Proteins c-ret
Receptor Protein-Tyrosine Kinases - genetics
Thyroid Neoplasms - genetics
Ukraine

Abstract: After the Chernobyl accident in 1986, there was a significant increase in the incidence of papillary thyroid cancer in fallout-exposed children from Belarus. Radiation-induced rearrangements of chromosome 10 involving the c-ret proto-oncogene have been implicated in the pathogenesis of these cancers. The ret/PTC3r1 rearrangement was the most prevalent molecular lesion identified in post-Chernobyl papillary thyroid cancers arising in 1991 and 1992. We identified the ret/PTC1 rearrangement in 29% of 31 papillary thyroid cancers presenting in Belarus in 1996. In the present report, we examined 14 cases from this series (plus 1 additional case) and found a ret/PTC3r1 rearrangement in only 1 (7%). The prevalence of ret/PTC3r1 in this series is significantly lower than previously reported (p = 0.0006, Fisher exact test). This result suggests a switch in the ratio of ret/PTC3 to ret/PTC1 rearrangements in late (1996) versus early (1991-1992) post-Chernobyl papillary thyroid cancers.

PubMed ID: 9848713 View in PubMed
Molecular alterations involving p53 codons 167 and 183 in papillary thyroid carcinomas from chernobyl-contaminated regions of belarus.

https://arctichealth.org/en/permalink/ahliterature20588

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**Source:** Thyroid. 2000 Jan;10(1):25-30

**Date:** Jan-2000

**Language:** English

**Publication Type:** Article

**Keywords:**
- Accidents, Radiation
- Adolescent
- Adult
- Base Sequence - genetics
- Byelarus
- Carcinoma, Papillary - genetics
- Child
- Codon - genetics
- DNA, Neoplasm - genetics
- Female
- Humans
- Male
- Molecular Sequence Data
- Mutation - genetics
- Nuclear Reactors
- Thyroid Neoplasms - genetics
- Tumor Suppressor Protein p53 - genetics
- Ukraine

**Abstract:** After the Chernobyl accident in 1986, there was a significant increase in the incidence of papillary thyroid carcinoma in fallout-exposed children from Belarus. We studied the p53 gene from 24 papillary thyroid carcinoma cases presenting in 1996. All subjects lived in contaminated regions of Belarus at the time of the accident and were under age 20 when exposed to fallout. Exons 5 through 9 of p53 were amplified from genomic tumor DNA using the polymerase chain reaction (PCR). The PCR products were analyzed by direct DNA sequencing using an automated sequencer. Five cases each exhibited two molecular alterations within exon 5. Alterations were confirmed by sequencing in both directions. One alteration, involving codon 167 (CAG-->CAT) in all five cases, resulted in the substitution of HIS for GLN. The second alteration, involving codon 183 (TCA-->TGA) in all five cases, resulted in a premature termination codon. Leukocyte DNA from each of the positive cases was analyzed and found to contain only wild-type p53 sequence. These results suggest that mutations involving codons 167 and 183 in the p53 locus are important in the pathogenesis of a subset (21%) of radiation-induced papillary thyroid carcinomas from Belarus.

**PubMed ID:** 10691310 View in PubMed
After the accident at the Chernobyl nuclear power plant, a considerable increase in the incidence of thyroid cancer among children in Belarus was observed. In the present study, the frequency of the c-ret protooncogene rearrangements in samples of thyroid carcinomas resected and diagnosed in 1998 from individuals in Belarus was investigated. The ret/PTC1 oncogene was detected in 19% of the samples, and the ret/PTC3r1 oncogene, in 14%. The number of ret/PTC1 rearrangements observed in tumor cells from the patients whose age at the time of the accident was from 1 to 10 years, was greater compared to those whose age at the time was from 10 to 20 years, irrespective of the year of surgery (1996 or 1998). The majority of the patients with ret/PTC3r1 rearrangements lived in Gomel oblast, which was contaminated by the Chernobyl meltdown.
An increase in the incidence of papillary thyroid cancer has been documented in individuals exposed to Chernobyl fallout in 1986. Experiments using cultured human cells have suggested that radiation can induce the ret/PTC1 rearrangement involving the ret proto-oncogene. To test the hypothesis that the ret/PTC1 rearrangement is involved in the pathogenesis of Chernobyl-associated papillary thyroid carcinomas, we studied a panel of 31 cases from Belarus. All individuals lived in fallout-contaminated oblasts (regions) of Belarus at the time of the accident: Gomel (n = 13), Brest (n = 12), Minsk (n = 4), and Grodno (n = 2). All were under age 20 at the time of the accident; 20 were born between 1982 and 1986. Individual thyroid radiation doses were estimated at 1.1 to 110 rem. Patients underwent surgery in Minsk in 1996. Fifteen patients had locally advanced disease (stage T4). The majority had regional lymph node involvement (stage N1, n = 27). There were no distant metastases. Surgical specimens were frozen at -80 degrees C, RNA was extracted and cDNA prepared. The polymerase chain reaction (PCR) was performed with specific primers for ret/PTC1, and c-ret and GAPDH as controls. Controls were positive in all 31 cases. Nine cases yielded a positive PCR product for the ret/PTC1 rearrangement (29%). Thus, the ret/PTC1 rearrangement is a feature of some Chernobyl-associated papillary thyroid cancers, and is one possible mechanism involved in the pathogenesis of these cancers.