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Chromosomes Aberations and Enviromental Factors

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SUMMARY

Explanation the topic: Changes in genetic material can lead to aberrant cell in the direction of disorders of cellular regulation, malignant transformation, cell death, or if the adjustment was made at the level of the reproductive cells, to genetic changes in some of the consequent offspring.

The topic position in scientific/professional public: Breaking of chromosomes can occur spontaneously or can be induced. Chromatid/chromosome breakings can be induced by different environmental factors: chemicals, biological clastogenic agents, accidentally or intentionally.

Conclusions: The authors suggest:

- making conditions for strong respect of environmental regulations;
- to use higher plants for the early detection of environmental mutagens;
- create and orderly update National radionuclide database.

Keywords: environment, chromosomal damage, mutagenic

EXPLANATION THE TOPIC

Analysis of the human karyotype to metaphase chromosomes allows a clear insight into the presence of structural and numerical chromosome aberrations, which represent the change in structure and number of chromosomes with respect to a normal human chromosome complement [1,2].

Some aberrant cells (a cell with the aberrations of chromosomes) are able to survive up to 10 cell divisions due to the presence

of unstable structural chromosomal aberrations, while the other may carry the stable chromosomal aberrations (inversions, translocations) and by dividing survive a large number of offspring [3].

Changes in genetic material can lead to aberrant cell in the direction of disorders of cellular regulation, malignant transformation, cell death, or if the adjustment was made at the level of the reproductive cells, to genetic changes in some of the consequent offspring [1,2,3].

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The influence of radiation on DNA in the G0 or G1 phase of the cell cycle, may cause discontinuation in the DNA, which are duplicated in the S phase of the cell cycle. If they are not removed by enzymes of reparative system, these radiolesions can be detected as structural chromosomal aberrations. The single-stranded, chromatid discontinuations are repaired [4,5]. Unstable structural chromosome aberrations such as dicentric type, ring chromosomes or acentric fragments are used as biomarkers of ionizing radiation activity. These structural chromosome aberrations make highest percentage of unstable aberrations in in vitro irradiation of the cell population of lymphocytes. They are indicators of a recent irradiation and exposure [6,7].

THE TOPIC POSITION IN SCIENTIFIC/PROFESSIONAL PUBLIC

Breaking of chromosomes can occur spontaneously or can be induced. Spontaneous chromatid/chromosome discontinuations and resulting structural chromosomal rearrangementsdo not occur rarely. There are indications that the frequency of their occurrence is in a positive correlation with age [4,8]. Chromatid/ chromosome breakings can also be induced by the action of various clastogenic agents (fracturing chromosomes).

Chemicals: exposing to complex mixtures of chemicals such as heavy metals, diesel emission particles and dust many of which are known as environmental chromosomal aberrations inducers [9]. Chemical mutagenesis and chromosomal changes have been reported in human population groups with occupational exposure to specific chemicals, such as lead, vinyl chloride, benzene (an industrial solvent and precursor in the production of drugs, plastics, synthetic rubber and dyes) pesticides, and styrene. In the exterior surrounding, the best known mutagens include the following metals: arsenic, cadmium, chromium, nickel [10,11].

Some study strongly suggests that exposure to pentachlorophenol, lindane, transfluthrin, cyfluthrin, and natural pyrethrum has a genotoxic effect on the epithelial cells of human nasal mucosa [12]. Fungicide, insecticides, pesticides, herbicides are the most frequently etiologic factor for chromosomal break [12]. It is found the enriched uranium caused breaks in the chromosomes that make up the DNA. Called clastogenic damage, the effects were related to the amount of radiation the enriched uranium released [9,13,14]. Aromatic amines and amides have been associated with carcinogenesis since 1895 when German physician Ludwig Rehn observed high incidence of bladder cancer among workers in German synthetic aromatic amine dye industry [15].

Environmental Radiation or "ubiquitous background radiation" is emitted from both natural and human-made radioactive chemicals (radionuclides) [16,17]. The radionuclides that enter the body are: terrestrial (uranium, thorium and their decay products, as well as potassium-40) and cosmogenic. The radionuclides enter the body through the food we eat, water we drink, and air we breathe [18, 19]. According to the period of taking effect and the impact on the human body environmental radiation could be:

a) Short-Term Health Effects of Radiation Exposure and Contamination: Acute Radiation Syndrome (Radiation sickness, known as acute radiation syndrome (ARS) and Cutaneous Radiation Injury

b) Long-Term Health Effects of Radiation Exposure and Contamination: Cancer, Prenatal Radiation Exposure, Mental Health.

In addition to protect habitants living on harmful environmental conditions it would be useful to have radionuclide database. as some high developed countries have [19]. Lymphocyte cells has high sensitivity to evaluate radiation damage. Radioexposion biomarker is presence of binuclear lymphocytes. The fundamental frequency of micronuclei in binuclear cells is quite variable. Value ranging from 2-36 micronuclei per 1000 binuclear cells [20,21]. Moreover, dependence on age and gender is noted 1 [4]. Also, the frequency of micronuclei is variable in relation to the diet [22], and an increased incidence of micronucleus can be seen in correlation with the operation of its various physical, chemical and biological agents (clastogenic agents) [21,23].

Environmental factors can induce chromosomal aberrations (CAs) usually divided into chromosome-type (CSAs) and chromatid-type aberrations (CTAs), whereas CSAs are good biomarkers for the prediction of cancer development [24]. A high and stable yield of CSAs persisted at least 1 year after external irradiation. The nature of the volume irradiated containing large blood vessels was the major determinant of the observed biologic dose [24,25].

War situation can permanently damage environment, and thus lead to potential health endangerment for the next hundred years or more. In this way, we cannot count the generations that carry chromosomal abnormalities to their offspring. An example is a war activity such as NATO air strikes with anti tank ammunition containing depleted uranium [2]. We suggest further investigation to evaluate the levels of radioactivity in indoor environments, which will reflect more closely the risks of the local population.

In research of experimental treatment of animals with non-genotoxic compounds, could form the mechanistic basis for what is called "spontaneous" tumor incidence when experimental animals' endogenous genotoxicity is present. The presence of endogenous DNA damage implies that exogenous DNAcarcinogen adducts give rise to an incremental damage which is expected to be proportional to the carcinogen dose at the lowest levels. An increased tumor risk due to exposure to exogenous genotoxic carcinogens could therefore be assessed in terms of the background DNA damage, for instance in multiples of the mean level or of the inter individual variability in a population. With endogenous DNA damage being present as a background, the additional risk from exogenous increments could be rated in relation to this unavoidable aspect of carcinogenesis [26].

Biological clastogenic agents: Chromosomal aberrations are described after EBV infection, polio virus, herpes virus, chickenpox, mumps virus, hepatitis viruses (B and C), influenza viruses, cytomegalovirus, as well as Mycobacterium tuberculosis [27]. Higher plants are recognized as excellent indicators of cytogenetic and mutagenic effects of environmental chemicals and are applicable for the detection of environmental mutagens both indoor and outdoor [28].

CONCLUSION

Further action needed for better environmental health in the future: Chemical substances are an essential part of modern life so the protection of them is in the proper handling and respectful procedures for chemical substances. To raise awareness, health professionals need to pay greater attention to the environmental condition. It is clear that as some civilization global developing and at the same time eco-destructive processes are not possible to terminate, it is necessary to increase the level of knowledge that would enable us to survive.

The authors suggest:

- making conditions for strong respect of environmental regulations;

- to use higher plants for the early detection of environmental mutagens;

- create and orderly update National radionuclide database.

REFERENCES

1. Katsantoni A, S Nakou, I Antoniadou-Koumatou, and G B Côté The effects of severe mixed environmental pollution on human chromosomes. J Med Genet. 1986 Oct; 23(5): 452-455.

2. Slavica I, Haverić S, Haverić A. Chromosome aberrations as bioindicators of environmental genotoxicity, Bosn J Basic Med Sci. 2007; 7 (4): 311-316.

3. Saou-Hsing Liou, Jia-Chyi Lung, Yeong-Hwang Chen, Tsann Yang, Ling-Ling Hsieh, Chien-Jen Chen, Trong-Neng Wu. Increased Chromosome-Type Chromosome Aberration Frequencies as Biomarkers of Cancer Risk in a Blackfoot Endemic Area. Cancer research, 1999; 59 (April 1): 1481-1484.

4. Fenech J, Morley AA (1986) Cytokinesis-block micronucleus method in human lymphocytes: Effect of in vivo ageing and low-dose X-irradiation. Mutat. Res. 193-198

5. Joksic G, Petrovic S (2004) Lack of adaptive response of human lymphocytes exposed in vivo to low doses of ionizing radiation. Journal of Environmental Pathology, Toxicology and Oncology. Vol 23: 195-206.

6. Jovicic D, Milacic S, Kovacevic R, Petrovic I (2004) Cytogenetic Analysis Chromosomal Status of Subjects in The Viciniti of Uranium-Contaminated Areas. 11th International Congress of the International Radiation Protection Association 22-28 May 2004, Madrid. Spain. Proceeding of the IRPA 11.

7. Rozgaj R, Kosuba V, Simic D (2002) The frequency of dicentrics and acentrics and the incidence of rogue cells in radiation workers. Mutagenesis. 170:135-139.

8. Therman E. Susman M. Causes of chromosome breakage, In: Therman E. Susman M, editors, Human chromosomes, New York Springer Werlag, Inc 1993. p. 107-117.

9. Darolles C,, Broggio D, Feugier A, Frelon S, Dublineau I, De Meo M, Petitot F. Different genotoxic profiles between depleted and enriched uranium, Toxicology Letters, 2010;19:2337-348.

10. Valko, M.; Morris, H.; Cronin, M. T. (2005). Metals, toxicity and oxidative stress. Current medicinal chemistry. 12 (10): 1161-1208.

11. Health Risk Assessment Guidance for Metals -Mutagenicity. Engineering Biology Research Consortium (EBRC); August 2007, https://www.ebrc.org

12. Tisch M, Faulde MK, Maier H. Genotoxic effects of pentachlorophenol, lindane, transfluthrin, cy-fluthrin, and natural pyrethrum on human mucosal cells of the inferior and middle nasal conchae. Am J Rhinol. 2005 Mar-Apr; 19(2):141-51.

13. Finley WH. Effect of drugs on chromosome structure. Am J Clin Nutr. 1975 May; 28(5):521-9.

14. d'Alesio V, Pacelli R, Durante M, Canale Cama G, Cella L, Gialanella G, Grossi G, Pugliese M, Punzo G, Sardi I, Scampoli P, Solla R, Salvatore M. Lymph nodes in the irradiated field influence the yield of radiation-induced chromosomal aberrations in lymphocytes from breast cancer patients. Int J Radiat Oncol Biol Phys. 2003 Nov 1; 57(3):732-8.

15. Vineis P, Aromatic amines and cancer, Cancer Causes and Control, 1997, 8, pp. 346-355.

16. Grasty RL, Lamarre JR. The annual effective dose from natural sources of ionizing radiation in Canada. Radiation Protection Dosimetry 108:215-226; 2004.

17. National Council on Radiation Protection and Measurements. Ionizing radiation exposure of the population of the United States. Bethesda, MD: National Council on Radiation Protection and Measurements; NCRP Report No. 160; 2009.

18. González AJ. Radiation safety standards and their application: international policies and current issues. Health Phys. 2004 Sep; 87(3):258-72.

19. LaBone ED, Farfán EB, Lee PL, Jannik GT, Donnelly EH, Foley TQ.Assessment of radionuclide databases in CAP88 mainframe version 1.0 and Windows-based version 3.0. Health Phys. 2009 Sep; 97(3):242-7.

20. Bauchinger M, Braselmann H (1989) "Use of micronuclei in biological dosimetry of apsorbed radiation dose", Chromosome Aberrations-Basic and Applied Aspects, Springer-Verlag Berlin. 202-211.

21. Jovicic D, Rajacic I, Milacic S, Kovacevic R, Tanaskovic I (2002) Frequency of Micronuclei of Human Population Occupationally Exposed to Radionuclides. European IRPA Congress. Proceedings. Editors F. D. Alberti and C. Osimani, European Commision Joint Research Centre

22. Fenech M, Aitken C, Rinaldi J (1998) Folate, vitamin B12, homocysteine status and DNA damage in young Australian adults. Carcinogenesis 19: 1163-1171

23. Rozgaj R, Kosuba V (2000) Chromosome aberra-

tions and micronucleus frequency in anesthesiology personnel. Arh. Hig. Rada Toksikol. 51:361-368.

24. Légal JD, De Crevoisier R, Lartigau E, Morsli K, Dossou J, Chavaudra N, Sanfilippo N, Bourhis J, Eschwège F, Parmentier C. Chromosomal aberrations induced by chemotherapy and radiotherapy in lymphocytes from patients with breast carcinoma. Int J Radiat Oncol Biol Phys. 2002 Apr 1; 52(5):1186-95.

25. Hagmar L, Strömberg U, Bonassi S, Hansteen IL, Knudsen LE, Lindholm C, Norppa H. Impact of types of lymphocyte chromosomal aberrations on human cancer risk: results from Nordic and Italian cohorts. Cancer Res. 2004 Mar 15; 64(6):2258-63.

26. Lutz WK. Endogenous genotoxic agents and processes as a basis of spontaneous carcinogenesis. Mutat Res. 1990 May; 238(3):287-95.

27. Paranjape SP. Virus induced chromosomal abnormalities in Chinese hamster lung cell line and human peripheral blood lymphocyte culture, Ind j of experimental biology, 2003 (41):112-117.

28. Grant WF. Higher plant assays for the detection of chromosomal aberrations and gene mutations—a brief historical background on their use for screening and monitoring environmental chemicals, Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis, 1999;426(2)

Hromozomske aberacije i životna sredina

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KRATAK SADRŽAJ

Tema: Promene u genetskom materijalu mogu dovesti do aberacija ćelija u pravcu poremecaja ćelijske regulacije, maligne transformacije, ćelijske smrti, a na nivou reproduktivnih ćelija do genetske promene u narednim generacijama.

Pozicioniranje teme u medicinskoj javnosti: Prekidi hromozoma nastaju spontano ili indukovano. Hromatidna/hromozomska oštećenja mogu biti indukovana različitim faktorima životne sredine: hemijskim, biološkim klastogenim agensima, slučajno ili namerno.

Zaključak: Autori sugerišu:

- postavljanje uslova za strogo poštovanje Regulative za očuvanje životne sredine;
- korišćenje viših biljaka za ranu detekciju mutagena životne sredine;
- kreiranje i uredno ažuriranje Nacionalne baze radionuklida.

Ključne reči: životna sredina, oštećenje hromozoma, mutageni

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