GENETICA POLONICA
Vol. 29 1988 No. 2

THE EFFECT OF ORALLY APPLIED AQUEOUS SOLUTIONS OF LEAD AND ZINC ON CHROMOSOME ABERRATIONS AND INDUCTION OF SISTER CHROMATID EXCHANGES IN THE RAT (*RATTUS* sp.)

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Summary. The influence of orally applied aqueous solutions of lead and zinc as well as its combinations on the occurrence of chromosome aberrations in the rat has been studied. A cytogenetic analysis was performed on the bone marrow cells multiplied as a result of the initial culture. A routine test of chromosome aberrations and SCE test were used. The action of lead present in the solution on chromosomes was expressed by the induction of a strong chromatin erosion and by chromosome pulverization. The presence of zinc in aqueous solution first of all induced structural chromosome aberrations, particularly chromatid gaps. The frequency of fragments exchange between sister chromatids also significantly increased. The application of aqueous solution containing zinc after 6-week treatment of animals with solution with lead significantly reduced a genotoxic lead effect. Biological aspects of the obtained results are presented.

Lead is a protoplastic poison, which easily forms complexes with amine and carboxyl groups leading to enzyme inactivation and disturbance of structural protein functions in the cell (Dutkiewicz 1974). An effect of its action are disturbances in different systems, such as: central nervous system (Kittel 1983), alimentary tract system (Szewczykowski 1957), kidneys and cardiovascular system (Kittel 1983) and erythrocyte system, where special affinity to bone marrow was displayed by lead (Gawlicka 1980).

Workers of rafinery, electrotechnical, textile, gum industries, graphical plants, non-ferrous metalurgy, as well as drivers are occupationally exposed the most to a toxic action of lead.

Zinc is a trace element necessary for normal development of plants, animals and people. It enters into the composition of polymerase of DNA and RNA as well

[1] Received for publication: April 1987. The paper was presented at the Symposium IX of the Polish Genetics Society, Gdańsk 1988, September 1 - 3.

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as thymidine kinase (Byczkowska 1962). It has been found that zinc has a protective action in relation to toxic effects induced by lead in erythrocytes (Finelli et al. 1975, Cerklewski 1976, Papaioannou et al. 1978, Abdulla 1980, Flanagan et al. 1982, Cerklewski 1984, Chiba, Kikuchi 1984, Chiba, Kikuchi 1985). Lead inhibits a number of enzymes of heme biosynthesis, including dehydratase of delta-aminolevulinic acid (ALA-D), whereas treatment with zinc causes restoration of that enzyme under in vivo and in vitro conditions. Ruither et al. (1977) displayed that a toxic effect of lead chlorate on macrophages in mice in vitro is smaller in the presence of zinc ions.

The present paper contains results of the studies on the effect of lead and zinc on the occurrence of chromosome pathology in the rat in vivo.

MATERIAL AND METHODS

The studies were carried out on rat males and females of the race Wistar with the weight about 200 g.

The animals were randomly divided into 5 diagnostic groups, separately among males and among females.

Dosage:

Group 1 — Pb, 500 ppm for 6 weeks in the form of lead acetate in drinking water,

Group 2 — Zn, 240 ppm for 14 days in the form of zinc chlorate,

Group 3 — Pb+Zn, Pb 500 ppm for 6 weeks, then zinc 240 ppm for 14 days,

Group 4 — Pb+H_2O, Pb 500 ppm for 6 weeks, than water for 14 days,

Group 5 — water control — the animals had only water throughout the experiment.

Each group consisted of 4 animals — 2 males and 2 females. Twenty four hours after the last dose, the animals were anaesthetized by ethyl eter and material was taken for studies.

A cytogenetic analysis was performed on the basis of cultured bone marrow cells. In each case, there were 2 versions of culture — routine and with the addition of 5-bromodeoxyuridine of (BrdU) for the analysis of induction of sister chromatid exchanges (SCE). The cultures were conducted on the nutrient medium consisting of Eagle's fluid, bovine serum and antibiotics (all manufactured in Poland). It has been found that the optimal time of culture is 28 - 31 hours. It makes possible to obtain cells after 2 replication cycles, which is important for sister chromatid differentiation, as well as a sufficient accumulation of cells. Colcemid (Serva) was added 30 minutes before the planned end of culture. Colcemid in the amount of 0.2 ml was added at the concentration of 10 μg/ml to the culture having the volume of 10 ml.

Cytogenetic preparations were made according to the traditional method using 0.075 M KCl as hypotonic fluid as well as Carnoy's solution (methanol and ice-cold acetic acid 3 : 1) as a fixator.

The culture with BrdU was supplemented with 0.1 ml of that compound per 10 ml of the nutrient medium (0.2% was a working concentration of Brd U). The technique of Perry and Wolff (1974) was employed to obtain differentiation in the sister chromatid dyeing.

In the preparations from routine cultures 50 random metaphase plates in the field of vision were analysed in each case. In the case of SCE shiftings of chromosome fragments were counted in all encountered metaphases with differentiated sister chromatids. The number of SCE's was counted per 1 metaphase plate and per 1 chromosome for each studied group.

A statistic analysis of the results was performed by the *t*-Student's test (test for two means from small samples).

RESULTS

Group 1. (Pb 500 ppm). The most frequently encountered abnormality was the pattern of chromosome contour broadening with a simultaneous chromatin washing (that chromatin formed sheaths round the chromosomes and entire metaphases). The chromosomes showed weak stainability.

In many cells that effect began from the middle of a metaphase plate causing uneven intensive chromosome staining within it (Figs 1 and 2). The degree of erosion was different in individual metaphases. In cases, where contour broadening was smaller, aberrations of the chromatid and chromosome type with the presence of acentric fragments was observed.

Chromatin erosion occurred in 53.5% of cells. Besides that, 35% of cells displayed the state of a still stronger destruction consisting in chromosome pulverization. In some metaphases that was a complete degradation, whereas in others it was partial (Fig. 3 and 4). As compared to the control these results were statistically significant. Chromosome aberrations were few and in relation to the control were statistically insignificant.

Group 2. (Zn 240 ppm). The observed effects of zinc action are first of all structural aberrations of chromosomes (chromatid and chromosome gaps and breaks). A number of metaphases dipslayed simultaneously all the mentioned types of aberrations, and for that reason the number of these abnormalities was given per 1 metaphase plate. The aberration level multiply exceeded not only the control values, but also those detected in the remaining studied groups.

The most numerous were chromatid gaps. They were observed in 148 out of 200 analysed cells, which constitute 0.74 gaps per cell, while the control value was 0.5 gap/cell. Statistic calculations in that case showed the largest significance level among all the observed changes in individual groups.

Chromatid and chromosome breaks were 0.42 and 0.21 per cell, respectively, and were also statistically significant in relation to the control.

Treatment with zinc in the amount of 240 ppm did not cause chromosome pulverization, and the frequency of chromatin erosion was similar to that of the control and statistically nonsignificant.

Group 3. (Pb 500 ppm, Zn 240 ppm). Lead intake for 6 weeks followed by zinc intake for 2 weeks, cause erosion and chromosome pulverization in similar proportions — 44% and 38% of cells, respectively. These values were statistically significant.

It was found that in this group the degree of the chromosome contour broadening decreased, which markedly improved the quality of metaphase plates in comparison to group 1 (56 out of 88 cells with erosion displayed a lower degree of injuries).

The frequency of structural chromosome aberrations was statistically insignificant to the control.

Table 1. Chromosomal aberrations in rat bone marrow cells after the treatment with Pb and Zn

Treatment	No. of animals	No. of cells analysed	Changes observed									
			erosion		pulverisation		chromatid gaps		chromatid breaks		chromosome breaks	
			no.	%	no.	%	no.	per cell	no.	per cell	no.	per cell
Pb	4	200	107	53.5	70	35.0	6	0.03	6	0.03	4	0.02
Zn	4	200	(35)	17.5	—	—	148	0.74	85	0.42	42	0.21
Pb+Zn	4	200	88 (56)*	44.0 (28)	76	38.0	5	0.025	5	0.025	1	0.005
Pb+H_2O	4	200	178 (69)	89.0 (34.5)	17	8.5	1	0.005	—	—	2	0.01
H_2O	4	200	(38)	(19.0)	—	—	9	0.05	4	0.08	7	0.035

* The bracketed numbers show a frequency of cells with a little erosion

Table 2. Sister chromatid exchanges in the rat bone marrow cells after the treatment with Pb and Zn

Treatment	No. of animals	Total cell scored	Number of		SCE	
			chromosomes	SCE_s	per chromosome	per cell
Pb	4	34	1428	123	0.087	3.61
Zn	4	58	2436	285	0.116	4.91
Pb+Zn	4	85	3570	360	0.111	4.24
Pb+H_2O	4	40	1680	147	0.087	3.67
H_2O	4	100	4200	340	0.081	3.40

The number of chromosomes was obtained by multiplying the total cell scored by 42, i.e. the number of chromosomes in diploid rat cells.

Group 4. (Pb 500 ppm, H_2O). The percentage of cells with erosion in that group was extremely high and statistically significant (178 cells out of 200 analysed ones). It, should, however, be emphasized that the percentage of chromatin destruction in many cells was clearly smaller than that observed in group 1 (in 69 out of 178 cells with erosion the chromosomes preserved their shape with a slight chromatin washing making possible a cytogenetic analysis). Pulverization and fragmentation was revealed only in 8.5% of the cells, but that result was statistically significant.

An analysis of induction of the sister chromatid exchanges in the studied material showed a statistically significant difference in the SCE_s number/cell between all the experimental groups and the control group. The largest significance was revealed

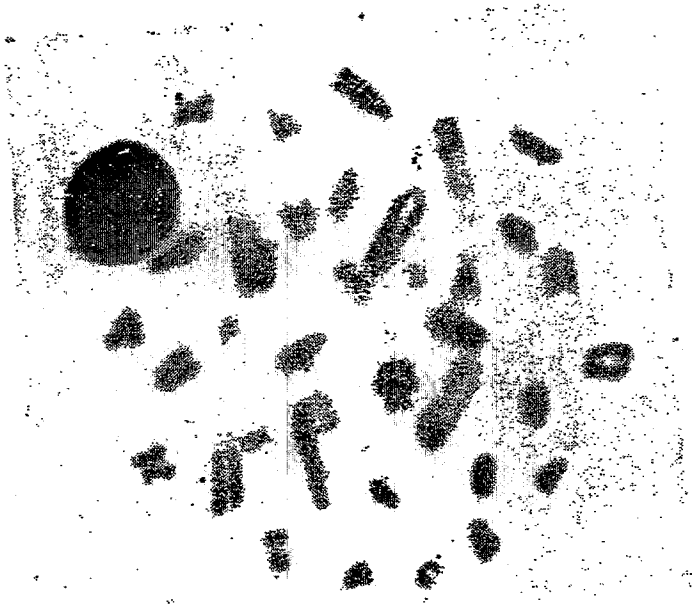

Fig. 1. A metaphase plate with visible washing of chromosome contours in the central part (Pb 500 ppm, 6 weeks)

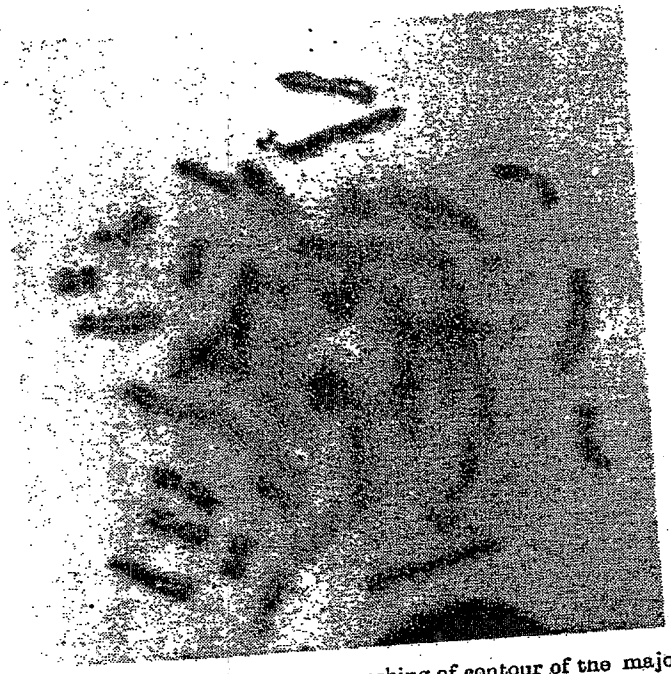

Fig. 2. A metaphase plate presenting a strong washing of contour of the majority of chromosomes (Pb 500 ppm, 6 weeks)

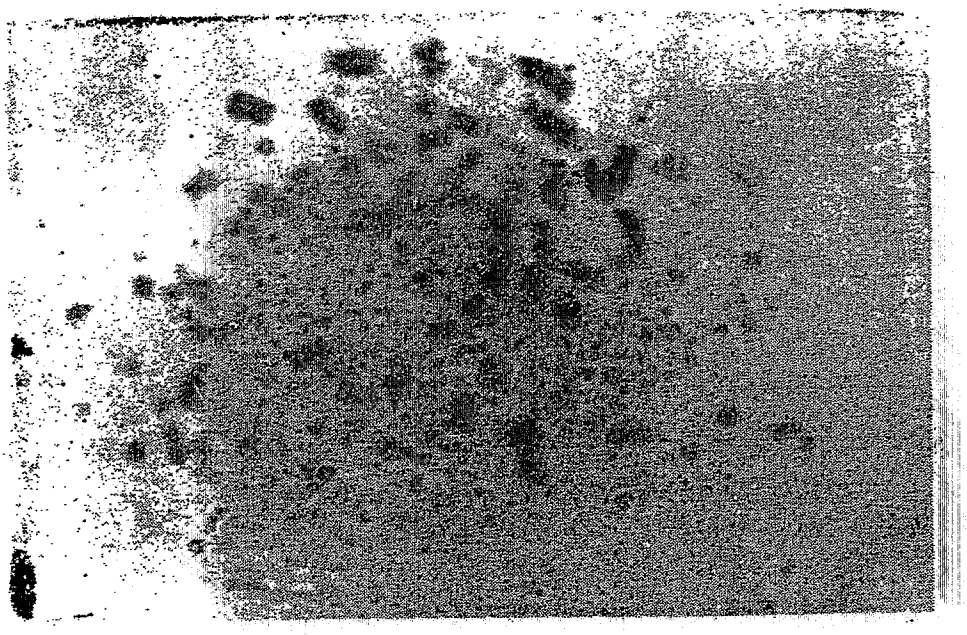

Fig. 3. A residue of a metaphase plate, in which the largest portion of chromosomes underwent pulverization (Pb 500 ppm, 6 weeks)

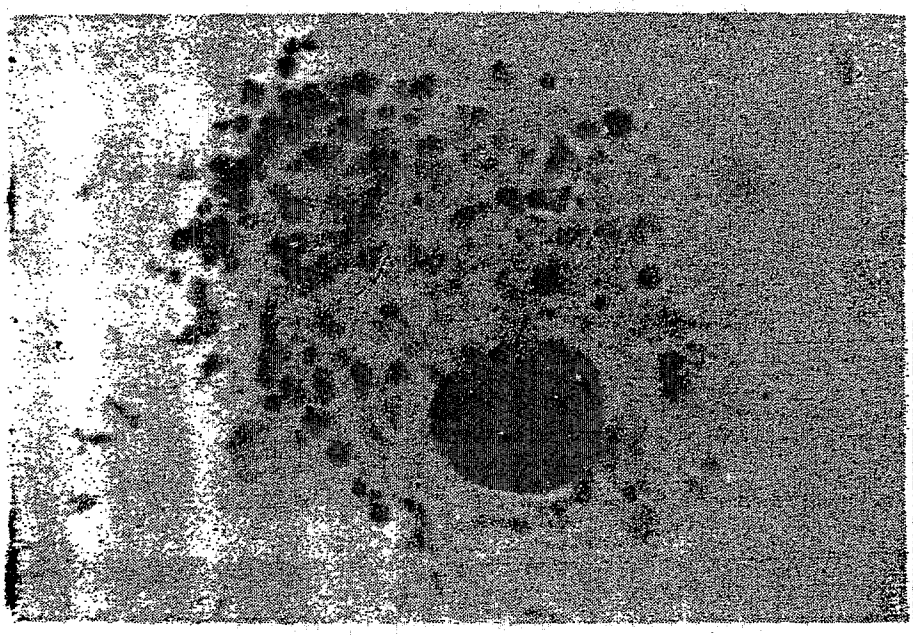

Fig. 4. A metaphase plate completely pulverized (Pb 500 ppm, 6 weeks)

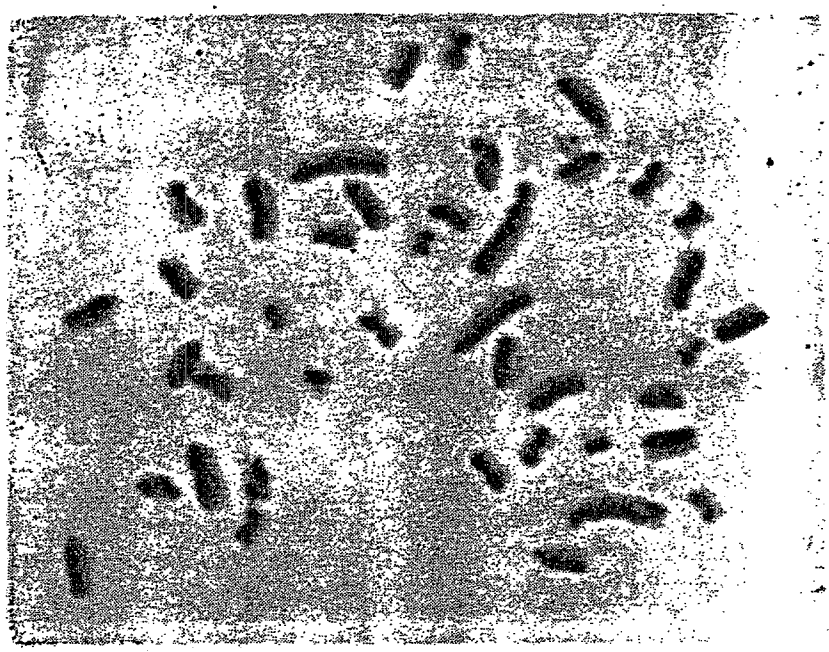

Fig. 5. A metaphase plate with sister chromatid differentiation (SCE test) in the case of lead and zinc action (Pb 500 ppm, 6 weeks and Zn 240 ppm, 14 days)

in the groups with zinc (groups 2 and 3). The number of SCE_s/cell in these groups was 4.91 (Zn) and 4.24 (Pb+Zn), while the control frequency was 3.40 SCE_s/cell (Fig. 5) (Tables 1, 2). Results obtained in male and female rats were similar.

DISCUSSION

An analysis of results of the studies on lead and zinc effect upon rat chromosomes has revealed certain abnormalities. An undoubtedly characteristic effect of lead action at the rate of 500 ppm for 6 weeks is washing of chromosome chromatin and in part of the cells — a more advanced destruction in the form of chromosome pulverization.

The cytogenetic analysis in the present paper was carried out on the bone marrow cells. As it is well-known, lead displayes the largest affinity to marrow (Gawlicka 1980). The lead level in the marrow exceeds 50-fold the content of this metal in blood. If the lead concentration in blood is 70 - 130 μg/100 ml that in bone marrow attains 4200 - 9200 μg/100 g of tissue (Dutkiewicz 1974, Gawlicka 1980). This undoubtedly affects the processes taking place in marrow, including also the state of genetic information and the rate and regularity of cell divisions.

Reports of the literature are focused mainly on cytogenetic studies among people occupationally exposed to the action of increased lead doses (Schwanitz et al. 1970, Forni, Secchi 1972, Schwanitz et al. 1975, Forni et al. 1976, Forni 1980, Forni et al. 1980). The material for these studies consisted of peripheral blood, i.e. of uncomparable tissue with regard to affinity to lead cumulation. The authors of these studies have revealed an evident increase in the frequency of structural aberrations of the chromatid type. Schwanitz et al. (1970) additionally observed spiralization defects and chromosome pulverization. Other authors did not confirm the occurrence of chromosome aberrations with regard to lead in industrial exposition (Bauchinger et al. 1972, O'Riordan, Evans 1974).

The present studies have shown that lead does not cause significantly increased frequencies of structural chromosome aberrations. An explanation of that may be the fact that the metaphases had strongly broadened contours and the chromosomes were little stainable, due to which they were insufficiently "readable" for a cytogenetic analysis. As a matter of fact, more breaks were observed in the cases, where erosion was weak, which permitted to estimate the chromosome structure. Therefore, the ability of lead to induce chromatid and chromosome breaks in the bone marrow cells cannot be completely negated. Improvement of the metaphase plates quality after putting away lead and application of 14-day water break (Pb+H_2O) indicates that changes induced by lead do not inhibit cell ability to regeneration and may be reversed to a certain extent.

A significant increase in the frequency of structural chromosome aberrations in blood lymphocytes under the effect of lead was revealed by Deknudt et al. (1977)

in a long-lasting experiment on monkeys in vivo, whereas a slightly increased level of chromosome fragments was detected in human lymphocytes cultured in vitro in the presence of subtoxic doses of salts of different heavy metals including also lead acetate (Deknudt, Deminatti 1978).

The first experimental work, which paid attention to a possibility of mutagenic action of lead in vivo was a publication by Muro and Goyer (1969). Mice orally got 1% of lead acetate in food. In the marrow cells there occurred a significant increase of chromosome gaps and breaks. Of interest is the interpretation of chromosome aberration induction by lead contained in the work of the mentioned authors. They suggest that lead may act on repair mechanisms of cell by exerting a negative effect on ATP and protein synthesis or may activate a number of enzymes, particularly DNA-ase, which causes damage to the genetic material.

In our studies a special ability to induce chromosome aberrations was displayed by zinc. These were the only significant effects of that metal action. A similar conclusion follows from the work by Eichhorn et al. (1973), who found that zinc forms stable combinations with various DNA groups, causing structural changes. This is important for normal functioning of nucleic acids.

Zinc fed to absorb lead caused a marked weakening of a toxic effect of that metal. The number of regular metaphases increased, and most of the metaphases had a week erosion. This corresponds to reports from the literature. It is considered that the most sensitive index of lead poisoning is a decrease in dehydrogenase activity of delta-aminolevulinic acid (ALA-D) in erythrocytes (Chiba, Kikuchi 1985). In the experiments on rats it was shown that lead inhibits 80 - 90% of that enzyme activity (Millar et al. 1970). An increase of zinc dose (above the required level) causes a marked reduction in lead absorption, which affects the lead level decrease in tissues and weakening of biochemical effects (Cerklewski, Forbes 1976). Zinc feeding after a 3-week treatment of rats with lead intensified lead discharge from erythrocytes. The speed of discharge increases proportionally to the zinc rate increase (Cerklewski 1984). The activity of ALA-D in blood, strongly decreased by lead action (200 ppm for 2 weeks), comes back to the control level more rapidly in animals feeding larger zinc doses. A similar action of zinc under in vitro conditions was displayed by Finelli et al. 1975. The same conclusion was made by Chiba and Kikuchi (1984) for zinc and manganese action in vitro in relation to toxic lead and tin effects in human, mouse and rabbit blood.

Our studies show agreement with such suggestions and extend hypotheses about protective action of zinc with reference to a harmful action of lead on the state of genetic information. That may be related with the suggested participation of zinc in functioning of DNA and RNA polymerases (Byczkowska 1962) and in consequence — with intensification of repair processes in cells.

The analysis of SCE induction was very difficult, especially in group 1, where a large percentage of injuries by erosion metaphases was noted. For that reason the number of analysed cells in that group is small. Troubles of a technical nature appeared also in other experimental groups due to the occurrence of chromatin erosion,

and in view of that all the metaphases covered by the analysis had well-differentiated sister chromatids.

Differences in the SCE number/cell as compared to the control were the largest in the case of action of zinc and zinc with lead. The presence of zinc, therefore, increased SCE induction. In the remaining studied groups deviations from the control levels were smaller, but all of them were statistically significant. A negative result of the SCE test was obtained by Beek and Obe (1975) in human lymphocytes after the action of lead acetate 10^{-5}M.

Unfortunately, the authors found the lack of mutagenic activity of lead salt, but stressed its highly harmful effect on developing fetus. Injuries may concern all the stages of organogenesis (Leonard et al. 1984).

Summing up the results in the present paper, it should be inferred that lead and zinc are metals with a high genotoxic activity in vivo, but interaction of zinc and lead weakens effects of lead action. Such a relation has not been found in the level of sister chromatid exchange induction.

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WPŁYW PODAWANEGO DOUSTNIE OŁOWIU I CYNKU W ROZTWORZE WODNYM NA POWSTAWANIE ABERRACJI CHROMOSOMOWYCH ORAZ INDUKCJĘ WYMIANY CHROMATYD SIOSTRZANYCH U SZCZURA (*RATTUS* sp.)

Streszczenie

Badano wpływ podawanego doustnie ołowiu, cynku oraz kombinacji tych metali w roztworze wodnym na powstawanie aberracji chromosomowych u szczura. Analizę cytogenetyczną przeprowadzono na komórkach szpiku kostnego namnożonych w wyniku hodowli pierwotnej. Zastosowano rutynowy test analizy aberracji chromosomowych oraz test SCE.

Działanie ołowiu w roztworze przejawiało się wywoływaniem silnej erozji chromatyny oraz pulweryzacji chromosomów. Obecność cynku w roztworze wywoływała przede wszystkim aberracje strukturalne chromosomów, szczególnie przerwy chromatydowe. Znamiennie zwiększała się też częstość wymiany odcinków między chromatydami siostrzanymi. Podawanie roztworu zawierającego cynk po uprzednim podawaniu zwierzętom roztworu z ołowiem wyraźnie zmniejszało genotoksyczny efekt ołowiu. Przedstawiono aspekty biologiczne uzyskanych wyników.

ВЛИЯНИЕ ПЕРОРАЛЬНО ПОДАВАЕМЫХ ВОДНЫХ РАСТВОРОВ СВИНЦА И ЦИНКА НА ОБРАЗОВАНИЕ ХРОМОСОМНЫХ АБЕРРАЦИЙ И ИНДУКЦИЮ СЕСТРИНСКОГО ХРОМАТИДНОГО ОБМЕНА У КРЫС (*RATTUS* sp.)

Резюме

Исследовалось влияние перорально подаваемых водных растворов свинца, цинка, а также комбинаций свинца с цинком на образование хромосомных аберраций у крыс. Цитогенетический анализ проводился на клетках костного мозга, размноженных в результате первичного разведения. Применён рутинный тест анализа хромосомных аберраций, а также тест SCE (сестринский хроматидный обмен).

Действие свинца, находящегося в водном растворе, проявлялось в сильной эрозии хроматина и в пульверизации хромосом. Цинк, находящийся в растворе, вызывал прежде всего структуральные аберации хромосом, особенно хроматидные разрывы, а также значительно повышал частоту обмена фрагментов между сестринскими хроматидами. Подача водного раствора цинка после предварительного 6-недельного перорального введения животным свинца, также в водном растворе, существенно уменьшила генотоксический эффект свинца.

Представлены биологические аспекты полученных результатов.