compared them with data of other autosomal inversions [Inv(1)371d and Inv(4)251d (Sonta et al. 1991)]. Males and females with inversion X chromosome were fertile, and the mean number in the litter was the same as that in karyotypically normal animals. By counting the marker chromosomes with unequal-length chromatids during the second meiotic metaphases (MII), we could detect the frequency of crossing-over and inverted chromosome segments in InvX+ females. Frequency of crossing-over on inverted segments of the X was very low as compared with the expected frequency, on the assumption that the crossing-over occurs in proportion to the length of chromosome segments. Individuals with a structurally abnormal X, which originated from these derivatives, were frequently born alive. Meiotic chromosome analysis also revealed that no increase in non-disjunction or normal segregation of autosomes was seen in InvX+ females and InvXY males. These findings suggest that there is no interchromosomal effect from the inversion of X during meiosis.

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A-34

TATEWAKI, R., H. OTANI and K. FURUSE, Department of Biology and Department of Anatomy, Shimane Medical University, Izumo, Shimane. Chromosomal anomalies of cells cultured under the diabetic condition.

We investigated morphologically and cytogenetically the causal mechanisms of developmental anomalies in diabetic pregnancies using non-obese diabetic (NOD) and ICR mice and have reported the results in meetings (1987–1994). In chromosome analyses, the post-implantation stage embryos of diabetic NOD (NOD-MI) had a high incidence of chromosomal anomalies and NOR associations (1992). Diabetic ICR mice induced by streptozotocin (STZ) also gave the same result (1994), thus influence of glucose and ketone body on the chromosomal anomalies was suggested.

In this study, we investigated the influence of glucose and ketone body on chromosomal anomalies using a cell culture system. Late gastrulation stage embryos (day 8, vaginal plug day 0) were used for primary cell culture. They were cultured in Eagle’s MEM (with 10% fetal calf serum) added with 300 mg/dl glucose, or 32 mM DL-β-hydroxybutyric acid (DL-β-OHB), or 300 mg/dl glucose plus 32 mM DL-β-OHB, based on Sadler’s report (Sadler et al., 1989) for 1 week. Incidence of numerical anomalies of chromosomes was 9.5% (p < 0.05, x²-test) in the addition of glucose, 8.1% in DL-β-OHB, 14.2% (p < 0.01) in glucose plus DL-β-OHB, whereas 5.0% in control. Aneuploidies in numerical anomalies was 5.9% in glucose, 5.6% in DL-β-OHB, 10.8% in glucose plus DL-β-OHB, whereas 4.0% in control, there was no significance difference (p < 0.05) between the addition of glucose and that of glucose plus DL-β-OHB. Incidence (4.7%, p > 0.05) of NOR associations in the addition of glucose plus DL-β-OHB increased in comparison with 2.9% in control, which is compatible with the increase in aneuploidy upon addition of glucose plus DL-β-OHB. Incidence of structural anomalies was 14.8% (p < 0.001) in glucose, 20.8% (p < 0.001) in DL-β-OHB, 15.1% (p < 0.001) in glucose plus DL-β-OHB, whereas 2.0% in control. These results suggested that chromosomal anomalies were induced by the addition of glucose and/or ketone body, that glucose plus ketone body increased numerical anomalies significantly, and that ketone body induced a high incidence of structural anomalies.

A-35

ISHIKAWA, H. and A. ENDO, Department of Hygiene and Preventive Medicine, Yamagata University School of Medicine, Yamagata. Analysis of colchicine-induced aneuploidies in embryos from XO mice.

During cytogenetic screening for breeding in our XO mouse colony, a high incidence of sex chromosome anomalies was observed in offspring of the colony. However, it was unknown why the incidence of chromosomal anomalies increased in the XO mouse colony. We speculate that oocytes from XO mice may be more susceptible to certain chromosomal mutations which produce an increase in the frequency of chromosomal abnormalities.

All mice used in this study were obtained from Jcl:ICR derived XO mouse colony, which has been maintained for 11–12 generations.Virgin XO and XX mice (3–5 months old) were allowed to mate with males of the same colony for 1 h. Both the pregnant XO and XX dams were subdivided into a colchicine-exposed group and a control group. The dams in the colchicine-exposed groups were intraperitoneally injected with 0.2 mg/kg colchicine dissolved in distilled water 6 h post-1-h mating. The mice were killed by cervical dislocation from 190 to 194 h (dg 8) after mating and embryos were prepared cytogenetically according to the method of Evans. We compared the incidence of numerical chromosomal abnormalities between XO dams and XX dams.

The results showed that the incidence of chromosomal anomalies induced by colchicine exposure did not differ distinctly between XX dams and XO dams. It was again found that triploidies and tetraploidies increased in XO embryos, either treated or untreated.

B-01

Not received.

B-02

MINAMIMOTO, T., Y. OHKUBO, H.H. IGAWA, T. SUGIHARA and N. SHINOHARA, Department of Plastic and Reconstructive Surgery, Hokkaido University School of Medicine, Sapporo, Hokkaido. A case of cleft lip, alveolus and palate associated with hypothyroidism and West syndrome.

We presented a very rare case of a congenital anomalous girl who had cleft lip, alveolus and palate associated with hypothyroidism and West syndrome. At 40 weeks and 1 day of gestation, she was born with a birth weight of 2,830 g. No other anomalies were found externally. In a Sapporo newborn screening test, she had no abnormalities in her endocrine system. The mother was 25 years old and the father was 33. They were both healthy and non-consanguineous. Two of the mother’s cousins and another of her relatives one mother’s kith and kin were cleft lip, alveolus palate patients.

At 2 months, she suffered from West syndrome and from 2 months she have had ACTH therapy by pediatricians. She had no brain shape abnormalities on CT and MRI but had hypsarhythmia on her electroencephalogram. We performed cheiroplasty at 10 months and velopectasty at 2 years and 11 months. At 5 years and 9 months, she suffered from hypothyroidism and had thyroid hormone therapy.

She had three abnormalities; cleft lip, alveolus and palate, West syndrome and hypothyroidism. The association of these three abnormalities may be explained as a spectrum of mid-face and brain anomalies. Mid-face anomaly was the cleft lip, alveolus and palate. Brain anomalies-West syndrome and hypothyroidism-were not visible radiographically.