A portion of the observed variation in SCE frequencies among primates might be the result of inherent differences in BrdUrd incorporation. Stetka and Carrano[12] found that the BrdUrd/base pair ratio correlated well with SCE frequency. The incorporation of BrdUrd is not linearly proportional to the molarity of BrdUrd in the culture medium, while the BrdUrd induced SCE frequencies are linearly proportional to the percentage substitution of BrdUrd for dThd[13]. It appears that BrdUrd competes with dThd for incorporation into DNA and that this competition determines the frequency of BrdUrd induced SCE. That mutagenicity of BrdUrd is not determined by the amount of the analog incorporated into DNA[14] might not be a valid factor for explaining interspecies differences in BrdUrd induced SCE since increased SCE frequency is not necessarily equivalent to increased mutagenicity[15,16].

1 I wish to thank S. Noble, A. Kirkilionis and D. Lang for their assistance, Dr S.L. Cleland of Connaught Laboratories, Toronto, who provided most of the monkey blood samples and the Metro Toronto Zoo for providing the gorilla blood samples.
2 H. Kato, Nature, Lond. *251,* 70 (1974).
3 S.A. Latt and L.A. Juergens, in: Population Cytogenetics Studies in Humans, p.217. Eds E.B. Hook and I.H. Porter. Academic Press, New York 1975.
4 B. Lambert, K. Hansson, J. Lindsten, M. Sten and B. Werelius, Hereditas *83,* 163 (1976).
5 W.F. Morgan and P.E. Crossen, Mutation Res. *42,* 305 (1977).
6 S. Pathak, O.G. Ward and T.C. Hsu, Experientia *33,* 875 (1977).
7 A.F. McFee and M.N. Sherrill, Mutation Res. *62,* 131 (1979).
8 P. Perry and S. Wolff, Nature, Lond. *251,* 156 (1974).
9 H. Kato, Int. Rev. Cytol. *49,* 55 (1977).
10 E. Giulotto, A. Mottura, R. Giorgi, L. de Carli and F. Nuzzo, Mutation Res. *70,* 343 (1980).
11 B. Beek and G. Obe, Hum. Genet. *49,* 51 (1979).
12 D.G. Stetka and A.V. Carrano, Chromosoma *63,* 21 (1977).
13 J.A. Mazrimas and D.G. Stetka, Exp. Cell Res. *117,* 23 (1978).
14 E.R. Kaufman and R.L. Davidson, Proc. natl Acad. Sci. USA *75,* 4982 (1978).
15 A. Kinsella and M. Radman, Proc. natl Acad. Sci. USA *75,* 6149 (1978).
16 M.O. Bradley, I.C. Hsu and C.C. Harris, Nature, Lond. *282,* 318 (1979).

## Red blood cell glutathione peroxidase in simple trisomy 21 and translocation 21/22[1]

J. Kędziora, R. Łukaszewicz, M. Koter[2], G. Bartosz[2], B. Pawłowska[3] and D. Aitkin[3]

*Department of Physiology, WAM Medical Academy, Pl. 9 Maja No. 1, PL-90-647 Łódź (Poland), 3 August 1981*

*Summary.* Red blood cell glutathione peroxidase activity was increased by about 50% in all Down's syndrome patients studied. It was slightly lower in translocation as compared with simple trisomy 21 but much higher with respect to controls.

Since the connection between trisomy 21 and Down's syndrome phenotype was postulated[4], numerous studies investigating enzyme activities in trisomy 21 have been carried out[5-9]. The most important recent discoveries in this field include the gene dosage effect[10], and localization of genes responsible for the phenotype of Down's syndrome and SOD-1 production[11,12]. However, activities of enzymes, being conditioned by many factors, seldom confirm theoretical predictions directly[13,14].

Inter alia, reports concerning the glutathione peroxidase (GSH-Px) activity are not consistent, some authors reporting its increased activity in Down's syndrome[15], and others finding no statistically significant increase[16]. In this report we give evidence for a statistically significant elevation of the GSH-Px level in erythrocytes of Down's syndrome patients.

*Material and methods.* 21 patients with Down's syndrome (19 with trisomy 21 and 2 with unbalanced translocation 21/22) were studied. Their karyotypes, determined according to Moorhead et al.[17], are shown in table 2. GSH-Px activity was estimated by the method of Beutler[18] in hemolysates prepared from heparinized blood. All absorbance measurements were performed using a Unicam SP 800 spectrophotometer (accuracy of 0.001).

*Results and discussion.* Data presented in tables 1 and 2 show an increase of about 50% in GSH-Px activity in patients with trisomy 21 as compared to controls. The difference is statistically significant ($p < 0.01$ using Student's t-test). Since in our previous work[19] we found a different behavior of SOD-1 activity in erythrocytes of patients with trisomy 21 and with unbalanced translocations 21/14 and 21/22, it seemed worthwhile to compare the GSH-Px activity in Down's syndrome due to translocation 21/22 with the activity in standard trisomy 21. Activity of the enzyme in erythrocytes of translocation patients was also increased but was slightly lower than in trisomy 21 (table 2). The low number of cases with translocation precludes detailed statistical analysis but the data obtained suggest some position effect.

Table 1. GSH-Px activity in erythrocytes of control subjects (units per g of hemoglobin)

| No. | Sex | Activity |
|---|---|---|
| 1 | M | 33.85 |
| 2 | M | 32.46 |
| 3 | M | 33.15 |
| 4 | M | 31.90 |
| 5 | M | 35.03 |
| 6 | M | 33.64 |
| 7 | M | 31.89 |
| 8 | F | 35.40 |
| 9 | F | 34.23 |
| 10 | F | 33.80 |
| 11 | F | 34.23 |
| 12 | F | 33.45 |
| 13 | F | 32.75 |
| 14 | F | 33.84 |
| 15 | F | 32.50 |
| | Mean | 33.45 |
| | SD | 0.39 |

The observed increase of GSH-Px activity in erythrocytes of Down's syndrome patients could be due to various causes. The simplest explanation would be to ascribe it to a gene dosage effect due to the linkage of the locus for GSH-Px to chromosome 21. However, enzymes known not to be linked to this chromosome were also reported to be elevated in the erythrocytes of Down's syndrome patients[5-9,15,20], suggesting a more complex basis for this phenomenon. In any case, the elevated activity of GSH-Px, a protective, $H_2O_2$-removing enzyme, may be of importance for the physiology of the Down's syndrome erythrocyte, which is subjected to increased generation of $H_2O_2$ due to the increased activity of superoxide dismutase[19].

Table 2. GSH-Px activity in erythrocytes of patients with Down's syndrome (units per g hemoglobin)

| No. | Sex | Karyotype | Activity |
|---|---|---|---|
| 1 | M | 47, XY, +G | 53.45 |
| 2 | M | 47, XY, +G | 51.15 |
| 3 | M | 47, XY, +G | 49.06 |
| 4 | M | 47, XY, +G | 57.30 |
| 5 | M | 47, XY, +G | 50.90 |
| 6 | M | 47, XY, +G | 55.12 |
| 7 | M | 47, XY, +G | 53.45 |
| 8 | M | 47, XY, +G | 48.95 |
| 9 | M | 47, XY, +G | 53.27 |
| 10 | M | 47, XY, +G | 50.38 |
| | | Mean | 52.30 |
| | | SD | 0.85 |
| 11 | F | 47, XX, +G | 52.06 |
| 12 | F | 47, XX, +G | 56.20 |
| 13 | F | 47, XX, +G | 51.36 |
| 14 | F | 47, XX, +G | 52.80 |
| 15 | F | 47, XX, +G | 47.38 |
| 16 | F | 47, XX, +G | 53.26 |
| 17 | F | 47, XX, +G | 53.27 |
| 18 | F | 47, XX, +G | 52.93 |
| 19 | F | 47, XX, +G | 56.90 |
| | | Mean | 52.88 |
| | | SD | 0.91 |
| 20 | F | 46,XX, −22, +t/21q22q/mat | 49.20 |
| 21 | M | 46,XY, −22, +t/21q22q/mat | 46.33 |


1 We are indebted to Prof. M. Ferguson-Smith from the Department of Medical Genetics, Royal Hospital for Sick Children, Glasgow, Scotland, for helpful discussions regarding our results.
2 Department of Biophysics, University of Łódź, Łódź (Poland).
3 Department of Medical Genetics, Royal Hospital for Sick Children, York Hill, Glasgow (Scotland).
4 J. Lejeune, N. Gautier and R. Turpin, C.r. Acad. Sci. Paris *248*, 1721 (1959).
5 A.C. Baikie, B.P. Loder, G.C. de Grouchy and D.B. Pitt, Lancet *1*, 412 (1965).
6 J. Lejeune, Ann. Genet. *22*, 67 61979).
7 R. Cox, Ann. N.Y. Acad. Sci. *166*, 406 (1976).
8 J. Kedziora, D. Rożynkowa, M. Kopff and J. Jeske, Hum. Genet. *34*, 9 (1976).
9 K.H. Gustavson, L. Wetterberg, M. Bäckström and S.B. Rose, Clin. Genet. *4*, 279 (1973).
10 P.M. Sinet, D. Allard, J. Lejeune and H. Jerôme, C.r. hebd. Séanc. Paris *278*, 3267 (1974).
11 P.M. Sinet, J. Couturier and D. Dutrillaux, Exp. Cell Res. *97*, 47 (1976).
12 Y. Nishida, I. Akaoka, T. Nishizawa, M. Maruki and K. Maruki, Ann. rheum. Dis. *36*, 261 (1977).
13 R. Herring, J. Phillips, H. Goodman and J. King, Lancet *1*, 1157 (1967).
14 L.-Y. Shih, P. Wong, T. Inoue, M. Makler and D. Hsia, Lancet *2*, 747 (1965).
15 P.M. Sinet, A.M. Michelson, A. Bazin and J. Lejeune and H. Jerome, Biochem. biophys. Res. Commun. *67*, 910 (1976).
16 A.F. Zakharov, Theoretical aspects of medical genetics, p.30. Meditsina, Moscow 1979.
17 P.S. Moorhead, P.C. Nowell, W.J. Mellman, D.M. Battist and D.A. Hungerford, Exp. Cell Res. *20*, 613 (1960).
18 E. Beutler, in: Red cell metabolism, 2nd edn, p.71. Grune & Stratton, New York, San Francisco and London 1973.
19 J. Kędziora, J. Jeske, H. Witas, G. Bartosz and W. Leyko, Acta biol. med. germ. *36*, 779 (1977).
20 F. Rosner, B.H. Ong, R.S. Paine and M. Preshanand, J. Am. med. Ass. *198*, 326 (1966).


## Induction of triploids in *Rhodeus ocellatus ocellatus* by cold shock treatment of fertilized eggs

K. Ueno and B. Arimoto[1]

*Department of Fisheries, Faculty of Agriculture, Kinki University, Higashi-Osaka, 577 (Japan), 22 September 1981*

*Summary.* Triploids were induced by cold shock in fertilized eggs of *Rhodeus ocellatus ocellatus*. The maximum percentage (95%) of triploidy was obtained from eggs treated at 5 min after fertilization. The triploids grew normally to adult size, and they were all sterile males.

Few studies have been reported on artificial polyploidy in fish, so the existing knowledge of the characteristics and techniques of production concerning polyploidy are not very useful in the practical breeding of fish. However, the morphological and physiological changes induced in fish by gene duplication could be useful for the improvement of fish breeding; such changes have recently proved to be helpful in plants. Treatment by low or high temperatures[2-5], X-rays[6] and chemicals[7] has been used to induce polyploidy in fish. The present paper deals with the necessary conditions of low temperature to induce triploid fish, their fertility and sex ratio.

For the subject fish, the rose bitterling, *Rhodeus ocellatus ocellatus* was chosen for this experiment because this acheilognathine fish has no chromosomal polymorphisms within the same species or among the different tissues in the body of one fish[8]. Also, it grows into an adult within 1 year under the climatic conditions prevailing in Japan. The parent fishes for the spawning of eggs were obtained from the lower stream of the Yodo River (Akagawa-cho, Osaka Prefecture), and raised in laboratory aquaria until the eggs matured. The eggs were obtained from several fully mature females using the stripping method. A sufficient number of eggs were immediately inseminated by sperm which was obtained from several males.

For the cold shock treatment, the eggs were exposed directly to cold water which was maintained at a temperature of 0-0.2 °C for 1 h beginning within 1 min after