prises the autosome to which the X is translocated. Replication of only the short arm, the actual X, was taken into account.

In both the lots of continuously labelled cells ($3^1/_2$ and $2^1/_2$ h ^{3}H-TdR), one X was hot in most of the plates. A total of 483 metaphases with one late replicating X were scored. Of these, the long-necked X (X^l) was late replicating in 57.8% (279 cells) and the short-necked X (X^s) displayed the same property in the rest of the 42.2% (204 cells) metaphases (figures 2a, b). Assuming that the X-chromosome inactivation takes place at random, the percentage value of each type of the late replicating X deviates considerably from the expected value of 50%. A simple χ^2 test reveals that this difference between the 2 X's is statistically very significant ($\chi^2 = 11.64$; $p > 0.001$).

The prerequisite for the cytological evaluation of randomness of X-inactivation in mammalian females[4] is the occurrence of 2 morphologically distinguishable but normal X-chromosomes in a female. Therefore the two female systems mostly used for this purpose are mules as well as mice heterozygous for Cattanach's X-autosome translocation. In both the instances, however, contradictory results have been obtained[6–10]. Recently, in Cattanach mice, Takagi and Sasaki[11] as well as Wake et al.[12] have shown that whereas in advanced stages of embryonic development the frequency of late replicating X was random, in early embryonic development paternal X was preferentially late replicating. These results are in accordance with earlier findings of Chandley[9], who observed selective inactivation of paternal X in Cattanach mice. The present report on the Muntjac X's also illustrates preferential late replication of the X^l chromosome. We do not know the pattern of inheritance of these 2 X's and hence cannot derive their parentage. Moreover, although the results are clear-cut, it may be premature to draw decisive evidence in favour of non-randomness of X-inactivation. The present data could as well be an exaggerated expression of what might have been a chance deflection at the time of differentiation of the X-inactivation in the primordial cells. Alternatively preferential distribution (or selection) of one type of X in a highly differentiated tissue cannot be ruled out as a possibility.

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No association between Gilbert's syndrome, the AB0 blood groups and the haptoglobin phenotypes[1,2]

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Summary. The different AB0 blood groups and haptoglobin phenotypes are as frequent in a well-defined group of subjects with Gilbert's syndrome as in appropriate samples of the general population.

Gilbert's syndrome is generally characterized by mild unconjugated hyperbilirubinemia in the absence of evidence for hepatic or hemolytic disease. Although a familial incidence with autosomal dominant transmission and variable expressivity has been noted[3–8], the mode of inheritance has not been satisfactorily established. Since genetic markers may be useful in such situations, the recently reported association between slow acetylator phenotypes and Gilbert's syndrome appears noteworthy[9]. In a search for further genetic markers, the association between Gilbert's syndrome, the AB0 blood group system and the haptoglobin phenotypes was investigated in the subjects previously published[9].

Material and methods. The records of the 27 patients with Gilbert's syndrome investigated for the acetylator phenotype[9] were examined for blood group determinations. In 22 cases serum (stored at -20 °C) was available for determinations of serum haptoglobin phenotypes. Determinations were done by starch gel electrophoresis. The diagnosis of Gilbert's syndrome was based on a) long-

AB0 blood groups and haptoglobin phenotypes in patients with Gilbert's syndrome

Analyzed proportions	Gilbert's syndrome	Controls	χ^{2*}
0 : A	13:11	1067:1274	0.703
0 : (B+AB)	13: 3	1067: 366	0.410
A : (B+AB)	11: 3	1274: 366	0.006
A : (0+B+AB)	11:16	1274:1433	0.432
Hp 2-1 : Hp 2-2	12: 7	1376: 970	0.160
Hp 2-1 : Hp 1-1	12: 3	1376: 431	0.127
Hp 2-2 : Hp 1-1	7: 3	970: 431	0.163
Hp 2-1 : (Hp 1-1+Hp 2-2)	12:10	1376:1401	0.218

*χ^2 corresponding to $p < 0.05$ is 3.9.

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standing or intermittent unconjugated hyperbilirubinemia, b) exclusion of hepatic disease by clinical examination and by normal findings of the following laboratory tests: fasting total serum bile acids, GOT, alkaline phosphatase, serum protein electrophoresis, hepatitis B_s-antigen, BSP-tests[10] and galactose elimination capacity[11]. No patient had overt hemolysis.

The distribution of A, B, 0 and AB blood groups and Hp 1-1, Hp 2-1 and Hp 2-2 haptoglobin phenotypes in this sample was compared with a series investigated for forensic purposes[12]. The available number of cases allowed analysis of groups 0, A and B + AB combined as well as of Hp 2-1, Hp 2-2, and Hp 1-1 + Hp 2-2 combined. Differences in the proportion of blood groups and haptoglobin phenotypes in Gilbert's syndrome and in the control series were tested for statistical significance according to Woolf[13].

Results. The incidence of the different genetic traits is detailed in the table. No differences could be detected between the patients with Gilbert's syndrome and the control population.

Discussion. Associations between diseases and some genetic polymorphisms, although rare, are well known phenomena[14]. They are usually interpreted as suggestive evidence for genetic factors contributing to the pathogenesis of the diseases in question. The present study is similar to the results obtained by testing the HL-A histocompatibility antigens in subjects with Gilbert's syndrome, by showing that the AB0 blood group system and the haptoglobin phenotypes cannot be used as genetic markers in this conditions. The association between acetylator phenotype and Gilbert's syndrome observed in the same sample of patients becomes the more relevant.

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Chromosomes of the black abalone (Haliotis cracherodii)[1]

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Summary. The chromosomes of the black abalone Haliotis cracherodii are described for the first time. The diploid number is 36, with no apparent sexual dimorphism.

Several gastropod karyotypes are described in the literature appearing from 1905[3] to the recent past[4–6]. These are mainly of snails and nudibrachs. Cytogenetic studies on abalone are lacking. 4-year-old 500 g specimens, 1 male and 1 female, were obtained from the intertidal zone near Diablo Canyon, California, and used for this study. 5 ml of a 0.1% Colcemid solution (in sea water) were injected into the center of the foot. 4 h later, the animal was dissected[2] and tissue fragments were obtained from the gill, mantle, digestive gland, gonad and blood. The tissues were placed in Hanks balanced salt solution containing 0.3% trypsin for subsequent mincing. After centrifugation the cell pellet was suspended in 5 ml 0.075 M KCl and allowed to sit for 20 min at room temperature. Following 3 fixations in absolute methanol: glacial acetic acid (3:1) the cells were dropped onto slides, air dried, and stained in Giemsa (10% in Gurr's Buffer).

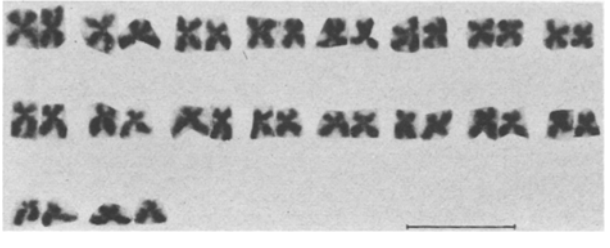

Fig. A. Karyotype of a male Haliotis cracherodii.

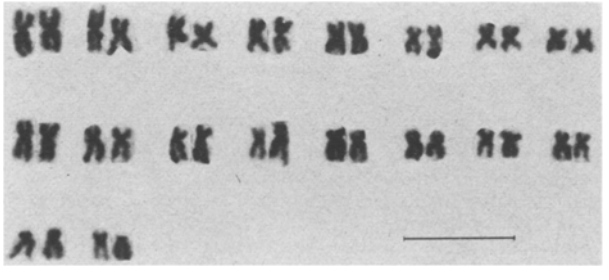

Fig. B. Karyotype of a female Haliotis cracherodii. Bar equals 10 μm.

The digestive gland was the only tissue to yield mitoses. Of 21 total countable metaphases, 15 had 36 chromosomes, 3 had 35, 2 had 34 and 1 had 18. 4 male and 3 female karyotypes were prepared. Examples are shown in figures A and B. These show 16 metacentric, 16 submetacentric, and 4 acrocentric chromosomes. The chromosomes are small, the largest being approximately 4 μm long. No sexual dimorphism is observable in these karyotypes. Sex determination may be at the intrachromosomal level. It is also possible that these karyotypes could represent tetraploids, especially since these cells came from the liver-like digestive gland. Further work will be necessary to clarify these ambiguities.

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