
Comments on Patients with Sex Chromosome Aneuploidy: Dermatoglyphs, Parental Ages, Xg\(^a\) Blood Group

DIGAMBER S. BORGAONKAR and EMILIE MULES

From the Division of Medical Genetics, Department of Medicine, The Johns Hopkins University School of Medicine, Baltimore, Maryland 21205, U.S.A.

It is known (Holt, 1968) that certain quantitative dermatoglyphic traits are genetically controlled and that chromosomal aberrations, both autosomal and gonosomal, have a modifying effect on traits such as total finger ridge count. Several suggestions have been made about the effect of sex chromosome aneuploidy on total finger ridge count. Low total finger ridge count is considered by Alter (1965) to be associated with hyperploidy of the sex chromosomes, probably owing to the presence of arches rather than alteration in over-all pattern. Penrose (1967) states that the 'Y-chromosome has less influence than the X—only about one third as much.' He further observed that the effects of subtracting sex chromosomes were cumulative but not quite additive. Later, Penrose (1968) stated, 'the presence of an X chromosome has approximately twice the effect on pattern size reduction as the Y chromosome,' thus revising his earlier observation. Hunter (1968) concluded that any effect of the Y chromosome on the mean total finger ridge count is more difficult to evaluate, and concluded that if the Y chromosome does affect the total finger ridge count, it does so only slightly. Uchida, Miller, and Soltan (1964) concluded that males with XXY chromosomal complement have characteristic dermatoglyphic patterns involving the presence of an ulnar triradius in the hypothenar area and arches and small patterns on the fingers. Alter et al. (1966) consider the hypothenar pattern fairly characteristic of the XXY complement. Data on dermatoglyphs of patients with sex chromosome aneuploidy have been collected with the prospect of developing diagnostic criteria based thereon, such as has been done for patients with trisomy 21 or Down's syndrome (Walker, 1958; Borgaonkar et al., 1968a; Polani and Polani, 1969).

We have collected data on parental ages at birth of sex chromosome aneuploid individuals, and have compared the findings with those of previous workers. In the case of the 47,XXY males one needs to be concerned with paternal age, as nondisjunction of the Y chromosome at the second meiotic division during paternal spermatogenesis results in an extra Y chromosome in the zygote. We present data collected through correspondence, from the literature, and from our own studies on 72 patients with 47,XXY chromosomes. In addition, data on parental ages at the time of birth of males with 48,XXYY chromosomes have been compiled and are presented.

With the availability of Xg\(^a\) blood group one can assign, in informative cases, the extra or the missing X chromosome as paternal or maternal, thereby locating the source of error at gametogenesis before zygote formation. Race and Sanger (1968, 1969) have recently pointed out the significance of this method. We present four 45,X Turner's syndrome cases and one 47,XXY Klinefelter's syndrome case, which are informative as to the source of the missing or extra X chromosome. In the 48,XXYY condition Xg\(^a\) blood grouping has been found to be useful in only two cases in assigning the source of the extra sex chromosomes (de la Chapelle et al., 1964; Pfeiffer et al., 1966).

Price (1968) reported some interesting electrocardiogram data on patients with sex chromosome aneuploidy. We present data on nine XXY and four XYY males in this report.

Materials and Methods

The patients with sex chromosome aneuploidy reported in this study attended the genetics and/or the endocrine clinics of this hospital. Diagnostic laboratory tests were performed to substantiate the clinical findings in the majority of cases. Sex chromatin...
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studies were done on buccal smears and chromosome preparations were obtained from leucocyte cultures and in some cases from skin fibroblast cultures. The control subjects with normal karyotype are medical students and hospital personnel.

Dermatoglyphic prints were obtained by Faurrot's material or Hollister's equipment, and read with the aid of a magnifying lens.

Xg* blood grouping was performed by Dr. Wilma Bias in the Immunogenetics Laboratory of this Division.

Results and Discussion

Dermatoglyphs

45,X Turner's syndrome. Total finger ridge counts on 20 patients averaged 175-8 (S.E. 12-92) (Table I), confirming earlier findings of Holt and Lindsten (1964), Penrose (1966), and Pfeiffer and Kierra (1968) of a higher value than that obtained for normal males and females. Since most of our patients were seen at advanced ages, we were unable to collect prints from sufficiently large numbers of parents and sibs in many families. However, the data obtained on the few relatives of Turner's syndrome patients are presented, and on comparison it appears that in general they are similar to those in the general population. The effect of absence (i.e. XO) or presence of the Y chromosome in the karyotype (i.e. XY and XYY) on total finger ridge count was tested and was found to be significant at p<0-001 level.

Palmar triadius was found to be distally placed bilaterally in 24% of the patients (Table II), and this appears to be a deviation (significant at p<0-001 level) from the normal population (9-5%) and parents. A pattern in the hypothenar area was present in 66% of the patients and simian crease was found in 12% of the palms (Table II). These frequencies also are high when compared with the general population which have frequencies of 25% and 1-5%, respectively (significant at p<0-001 and 0-05 levels, respectively).

47,XXY Klinefelter's syndrome. The average total finger ridge count for our 21 patients is 105-24 (S.E. 9-93) as compared to 121-7 obtained for 12 patients by Hunter (1968), 114-8 obtained for 25 patients by Penrose (1968), and 122-17 obtained for 23 patients by Uchida et al. (1964) (Table III). The frequency of arches on the fingers appears to be higher than in the general population (Table II). For 25 patients the frequency of arches was 8% compared with 3-5% for 138 normal 46,XY males and 7-0% for 20 normal 46,XX females (significant

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<td>166-10 (S.E. 8-62)</td>
<td>Holt and Lindsten (1964)</td>
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**TABLE II**

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<td>91-05</td>
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<td>10-52</td>
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<td>17-3</td>
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<td>Whorl</td>
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<td>31-2</td>
<td>25-5</td>
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<td>25</td>
<td>24</td>
<td>16</td>
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* Data on 15 patients only.
at \( p < 0.001 \) level). Therefore, the low total finger ridge count obtained on Klinefelter’s syndrome can be attributed at least in part to the increased frequency of arches.

Palmar triadius was distally placed in 16% and intermediate in 50% of the cases studied compared with 13.6% and 21.8% for the control group (significant at \( p < 0.001 \) level). Hypothenar pattern was found in 32% of the cases. Simian crease was seen in 4 of the 25 patients (16%).

The extra X chromosome appears to affect the dermatoglyphic patterns considerably. As in 45,X Turner’s syndrome, the total finger ridge count is affected. While in the former it is increased, the presence of an extra X chromosome in Klinefelter’s syndrome reduces the total finger ridge count and is significantly different (at \( p < 0.025 \) when compared with normal females and 48,XXXY males. As the cases of Klinefelter’s syndrome were detected at or after puberty, it was often difficult to obtain prints from relatives in the family. They had either moved away or had died, or it was difficult to get their cooperation, since the patients did not want the parents to be approached for any studies.

Forbes (1964) studied 24 patients with Klinefelter’s syndrome and found that mean ridge counts on right index fingers were 8.5 and on an average less than normals. The average on our 25 patients was 10.18, and only the right middle finger has a lower ridge count. Forbes’ observations on simian crease, hypothenar pattern, and palmar triadius differ from ours in the sense that she did not find any patient with a simian crease, and other patterns were more in the normal range. But she did find loops with lower ridge counts and more arches on the fingers.

47,XXY males. Penrose (1966, 1967) has compiled information on total finger ridge count. Mavwalwa, Parker, and Melnyk (1969) reported on one patient and reviewed four earlier cases, and concluded that there were no consistent patterns associated with this group of patients. Average total finger ridge count is 109.85 (S.E. 11.29) for 28 patients (Appendix*). This figure is higher than that obtained for 47,XXY Klinefelter’s syndrome patients but lower than that obtained for normal males and females. The average total finger ridge count is significantly different (\( p < 0.001 \)) from normal males and patients with Turner’s syndrome, indicating the effect of the extra Y chromosome. The extra Y chromosome reduces the ridge counts but not so much as the extra X chromosome does, thus confirming the findings of Hunter (1968) and Penrose (1966, 1967).

The hallucal patterns do not appear to be different from those in the general population, and consist of whorls and loops in 90% of the cases studied. Palmar triadius was distally placed in 12% of the cases studied and hypothenar pattern was present in 31% of the cases. Simian crease was found on only one hand of a single patient out of a total 15 studied (Table II).

Archs are present in higher frequency (17.7%) and probably contribute towards reducing the ridge counts. We had used the criterion of one or more arches on the fingers and a total finger ridge count of less than 80, along with height of over 183 cm., as one of the prerequisites for karyotyping inertmates in a screening survey for ascertaining XYY males. However, the sample of 18 such inmates had normal 46,XY karyotype. Even though the over-all frequency of arches is 17.7%, it should be noted that there are 15 47,XXY patients with no arches on any one of the digits, and there are 2 47,XXY patients with 10 arches.

48,XXXY males. There are several reports of this condition with no mention of dermatoglyphs. The average total finger ridge count for 21 patients is 91.05 (S.E. 10.52) compared to 106.1 for 7 patients of Penrose (1967), 88.71 (S.E. 23.87) for 7 patients of Uchida et al. (1964), and 103.6 (S.E. 6.9) for 5 patients of Alter et al. (1966). The frequency of arches on the fingers is 17.3% and is similar to that of XYY males (Table II); it does not appear to be the sole reason for the reduced finger ridge counts as suggested by Alter (1965) and by Uchida et al. (1964). Palmar triadius was intermediate in 75%, and distally placed in 9% of the cases studied. A hypothenar pattern was present in 70% of the cases. A simian crease was present in 22% of the cases. There appears to be a significant deviation from the normal, especially when comparison is made with

### TABLE III

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<tr>
<th>No. of Patients Studied</th>
<th>Average Total Finger Ridge Count</th>
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</tr>
<tr>
<td>12</td>
<td>121.7 (S.D. 55-8)</td>
<td>Hunter (1968)</td>
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<td>114.8 (S.E. 11-02)</td>
<td>Penrose (1967)</td>
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<tr>
<td>23</td>
<td>122.17 (S.E. 11-02)</td>
<td>Uchida et al. (1964)</td>
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</table>

* For the Appendix, order document NAPS 01006 from ASIS National Auxiliary Publications Service, c/o CCM Information Sciences, Inc., 909 Third Avenue, New York, 10022, U.S.A.; remit $2.00 for microfiche or $5.00 for photocopies.

A copy of the Appendix is also deposited in the B.M.A. library (address B.M.A. House, Tavistock Square, London W.C.1), and access to it can be made by arrangement with the librarian.
parents and normal sibs (Ferrier and Ferrier, 1968), and dermatoglyphic peculiarities may be useful in distinguishing this syndrome from 47,XXY Klinefelter's and XXY males. We are unable to confirm the earlier conclusion drawn by Uchida et al. (1964) and by Alter et al. (1966). They concluded that these patients had characteristic configurations of the hypothenar area involving the presence of an ulnar triradius with a loop carpal, loop radial, or arch radial pattern, and that the hypothenar patterns appear to be specific for this chromosome complement and not for other double Y combinations or XXY.

Data on one case of 49,XXX female (Borgaonkar and Leger, 1969), two cases of 48,XXXY males (Ferguson-Smith, Johnston, and Hardemaker, 1960), and one case of 49,XXXXY male (Walker and Borgaonkar, 1969) are presented (Appendix). Total finger ridge count was 150 in the triple-X patient with no arch pattern on any of the fingers. Both parents of the XXX female were not available for any studies, as the mother was dead and the father could not be traced. The two males with 48,XXXY did not have any arches on the fingers, and the total ridge count on one of them was 128. The finger ridge count could not be determined on the other patient because the patients' hands and fingers could not be straightened out sufficiently to obtain good and clear prints. The patient with 49,XXXXY karyotype had a total finger ridge count of 88 with an arch on the right index finger, and a simian crease and distally placed triradius. This patient and both of his parents were Xg⁺ positive.

**Parental age**

45,XO Turner's syndrome. The average paternal age of 27 patients, 27-48 (S.E. 1·12) years was very similar to the average of 27-74 (S.E. 0·72) obtained on 63 patients by Boyer, Ferguson-Smith, and Grumbach (1961), but it is low when compared to the average of 34-40 obtained by Soltan (1968) on his 20 patients (Table IV). In 4 of our patients the missing sex chromosome was found to be paternal and the average age of these 4 fathers is 29 years. The combined average paternal age of our 4 XMO cases and of 4 XMO cases of Soltan (1968) is 31-88 (S.E. 2·63) years. It is significantly (at p < 0·05 level) higher than the average paternal age of XO patients obtained by us and Boyer et al. (1961). The lower frequency of XO cases as compared to XMO cases has already been pointed out by Edwards et al. (1966) and Fraser (1963).

A total of 14 45,X patients were typed for Xg blood group, and 8 were Xg positive and 6 were Xg negative. Among the 11 fathers typed for Xg, 5 were positive, 6 were negative, and 2 were dead. One mother was dead, and among the 14 mothers typed for Xg, 9 were positive and 5 were negative.

**TABLE IV**

<table>
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<th>No. of Patients Studied</th>
<th>Average Maternal Age (yr.)</th>
<th>Standard Error</th>
<th>No. of Patients Studied</th>
<th>Average Paternal Age (yr.)</th>
<th>Standard Error</th>
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<td>60</td>
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47,XXY Klinefelter’s syndrome. The average maternal age for 20 patients was 28-45 (S.E. 1-53) years as compared to the average paternal age of 30-32 (S.E. 1-70) for 21 patients. These are slightly lower than those reported by Ferguson-Smith et al. (1964), Frölund, Sanger, and Race (1968), and Soltan (1968) (Table IV). In only one of our patients could we determine that the extra X chromosome was paternal in origin, and the father of this patient was 25. The average paternal age for 10 X^MY patients was 30-90 (S.E. 2-0) and was found not to be significantly different when compared to the average for our XXY patients, but is higher than the average paternal age of 26-42 years for the U.S. population in 1960 and the average paternal age of 27-02 years for seven X^MY cases. The average paternal age for 7 X^MX^P^Y cases was 29-71, and is not significantly different from the average paternal age of our Klinefelter’s syndrome patients. As most of these patients were detected at or past puberty, it was not always possible to find both parents living. Not many of our cases typed informative, but this was not surprising in view of the experience obtained by other investigators. A total of 19 XXY patients were typed for Xg blood group and 8 were Xg negative and 11 were Xg positive. Two fathers were dead, 2 were Xg negative, and 2 were Xg positive. One mother was dead, 2 were Xg negative, and 4 were Xg positive.

48,XXXXY males. In 2 patients (de la Chapelle et al., 1964; Pfeiffer et al., 1966), the source of the extra X chromosome was detected: the ages of the fathers were 36 and 24 respectively at the time of the patients’ births. The average maternal age for 24 cases is 27-63 (S.E. 1-59) years and the average paternal age for 23 cases is 32-57 (S.E. 2-22) years (Table IV). It may be that there is a trend towards higher paternal age, but many more cases need to be studied (Borgaonkar, Mules, and Char, 1970).

47,XYY males. As the non-disjunction in this case probably takes place at the second meiotic division in the spermatogenesis of the father, it seemed worth while to see what the paternal age is at birth of XYY males. If, on comparison with the average for the general population differences were to be found, this would indicate whether there was any relation between paternal age and non-disjunction of the Y chromosome in the second meiotic division. Average paternal age appears to be higher with sporadic cases of single gene disorders with a dominant mode of inheritance (Murdoch et al., 1970). The average age of 72 fathers of XYY males is 31-08 years (S.E. 0-85) (Table IV). A histogram (Fig.) has been prepared which shows the age distribution of the 89 fathers in whose spermatogenesis an error had occurred. We have included the 72 XYY cases, 8 cases of X^MY, 2 cases of X^MX^PY, and 7 cases of X^MX^PY, and the distribution here does appear to be slightly slanted towards an increased paternal age, though not significantly different. The over-all average is 31-02 (S.E. 0-76) years and is a year more than the average of 29-89 for the U.S. population in 1960. However, the 89 cases included in this tabulation have been reported from all over the world, and the range is from 18 to 53. It would be desirable to have a uniform population for comparison, including birth order and time period.

Electrocardiogram. Price (1968) obtained a mean PR interval of 0-169 (S.E. 0-013) sec. for 13 XXY males. Average PR interval on our 6 XXY cases was 0-154 sec., and the range was 0-13 to 0-18, and 3 other patients were reported to have normal electrocardiograms (Appendix). We do not consider these as variations from the normal. Further, Price examined the electrocardiograms of 20 males with 47,XXY chromosome complement, and found that they had prolonged PR intervals of 0-184 (S.E. 0-035) sec. Our 4 patients had a mean PR interval of 0-177 sec. (Borgaonkar et al., 1968b).

Summary

Dermatoglyphic data on 45,X patients are presented and compared with previous reports. These patients usually have high total finger ridge count, distally placed palmar triradius, hypothenar pattern, and rarely a simian crease. Average paternal
age for 27 patients was 27-48 (S.E. 1·12) years and in 4 XXYO cases 29 years.

Dermatoglyphic data on 47,XXY patients are presented and compared with earlier reports. Total finger ridge count is low in these patients. ECG findings on 9 patients were normal. Average maternal age of 20 cases was 28·45 (S.E. 1·53) years, and average paternal age for 21 cases was 30·32 (S.E. 1·70) years. Only one case of XXYXY is reported.

Dermatoglyphic data have been compiled and reported for the first time on 28 47,XXY patients. Mean total finger ridge count is 109·84 (S.E. 11·29). Hypothenar pattern was present in 31% of the cases. Arches on digits were present with 17·7% frequency. Average paternal age of 72 cases is 31·08 (S.E. 0·85) years.

Dermatoglyphic data on 21 48,XXXY patients have been compiled. Average total finger ridge count is 91·05 (S.E. 10·52), and frequency of arches is 17·3%. Hypothenar pattern was present in 70% and a similar increase in 22% of the cases, respectively. Average maternal age of 24 cases is 27·63 (S.E. 1·59) and average paternal age of 23 cases is 32·57 (S.E. 2·22). Data on one case with 47,XXX, two cases with 48,XXXX, and one case with 49,XXXXY chromosomes are also presented.

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References


Comments on patients with sex chromosome aneuploidy: dermatoglyphs, parental ages, Xg a blood group.

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