Dermatoglyphic Evidence of Fluctuating Asymmetry in Schizophrenia

C. S. MELLOR

Fluctuating asymmetry provides a measure of an organism's capacity to buffer adverse factors that could disturb its development. It is estimated from the differences between theoretically identical right- and left-sided structures. Dermatoglyphic fluctuating asymmetry has been recently used to investigate developmental disorders. Fingerprints and palm prints of schizophrenic patients, which had been the subjects of an earlier report of conventional dermatoglyphic trait frequencies, were reanalysed to developmental their level of fluctuating asymmetry. A review of the diagnostic protocols and clinical records used in the original study indicated that most of the 482 subjects would have met DSM-III-R criteria for schizophrenia. The schizophrenic sample had significantly higher levels of fluctuating asymmetry on four dermatoglyphic traits, the finger-ridge counts, fingerprint patterns, the palmar at angles and palmar a-b ridge counts, than controls. This finding supports the results of two earlier studies, and its relevance to the roles of genetics, foetal insults, and developmental anomalies of the brain in the aetiology of schizophrenia is discussed.

Fluctuating asymmetry, random differences in size between supposedly identical right- and left-sided structures, is believed to be an indicator of developmental stability. Dermatoglyphic traits have been used recently to estimate fluctuating asymmetry and this provides a new way of looking at schizophrenic dermatoglyphics. This paper reports on such a study, the dermatoglyphic trait frequencies having been the subject of an earlier publication (Mellor, 1968).

Dermatoglyphics, the study of ridged skin on the palms and soles, is customarily used in the investigation of chromosomal and other congenital abnormalities. Dermatoglyphic analyses use either qualitative methods, based on the patterns formed by the dermal ridges, or are quantitative, a count of the dermal ridges in a pattern being the usual measure.

The scientific value of dematoglyphics largely derives from the fact that the dermal ridges appear in the third to fifth month of foetal development (Penrose & Ohara, 1973) and the patterns then formed never change. Therefore, dermatoglyphic abnormalities are due to genetic or other factors that express their effect before the end of the fifth month of foetal development.

Dermatoglyphic studies have, therefore, been used to investigate the effects of prenatal factors in the aetiology of schizophrenia (Mellor, 1968). Unfortunately, the results of such studies employing conventional qualitative and quantitative methods have yielded ambiguous results that are difficult to interpret. However, the use of dermatoglyphic traits to measure the degree of fluctuating asymmetry provides a new approach to this problem.

In order to explain this approach, a description of fluctuating asymmetry is first required. Van Valen (1962), in an extensive review, credits Ludwig with first describing fluctuating asymmetry in 1932. Fluctuating asymmetry has been used to assess developmental stability in various species including man. It can be defined as the random differences between corresponding morphometric characters on each side of the plane of symmetry. In practical terms, one estimates the level of fluctuating asymmetry from the differences between corresponding rightand left-sided structures. These differences are zero if each side is a perfect mirror image of the other. Fluctuating asymmetry requires that the differences between the two sides are random, that is, one side is never consistently bigger, or smaller, than the other. (If the differences are consistently in the same direction then this would be directional asymmetry.) The distribution of the signed, right minus left differences, in a population sample, approximates to a normal curve with a mean of zero. The variance of this distribution curve is a measure of fluctuating asymmetry.

The degree of fluctuating asymmetry is indicative of an organism's susceptibility to developmental noise. A high level signifies that the organism has a low capacity for buffering adverse environmental effects that could deflect the course of its genetically determined programme of development (Van Valen, 1962). There is probably a general buffering capacity for the whole organism as well as one for individual structures. Individuals who are relatively homozygous have inferior developmental stability when compared to those who are relatively heterozygous (Soulé, 1979). Soulé & Cuzin-Roudy (1982) have suggested, therefore, extreme phenotypes will have less general buffering capacity than average phenotypes.

The use of dermatoglyphic fluctuating asymmetry as an indicator of the human developmental process has been advocated by Livshits & Kobyliansky (1987). They found that subjects whose measurements on a group of anthropometric variables were close to the mean showed less dermatoglyphic fluctuating asymmetry than those with more outlying measurements. These findings support the hypothesis that fluctuating asymmetry is greater in extreme phenotypes. Dermatoglyphic fluctuating asymmetry has not been found to be related to sex (Micle & Kobyliansky, 1988), nor to handedness (Kobyliansky & Micle, 1986).

In their study of cleft lip, Woolf & Gianas (1976, 1977) used three dermatoglyphic traits, the palmar atd angle, the palmar a-b ridge count, and fingerprint patterns, to estimate the degree of fluctuating asymmetry in 'propositi' with congenital cleft lip. These subjects had significantly greater fluctuating asymmetry than normal subjects and subjects with non-congenital cleft lip. The unaffected parents and siblings of those with congenital cleft lip had fluctuating asymmetry levels between those of the propositi and normal subjects. Their findings indicated that cleft lip is genetically heterogeneous, that the familial form has a polygenic cause and that the genes responsible for cleft lip also raise the general level of fluctuating asymmetry.

In the first investigation of dermatoglyphic fluctuating asymmetry in schizophrenia, Markow & Wandler (1986) studied a sample of 81 subjects using two dermatoglyphic traits, the palmar a-b ridge count and fingerprint patterns. The fluctuating asymmetry was significantly higher for the schizophrenic group than the control group for both traits. Markow & Gottesman (1989) subsequently found that twins concordant for schizophrenia had higher levels of fingerridge count fluctuating asymmetry than discordant twin pairs.

There are cogent reasons for studying dermatoglyphic fluctuating asymmetry in schizophrenia. If fluctuating asymmetry is greater than in normal subjects, then this suggests that disturbances of foetal development may play some part in the later development of schizophrenia. Developmental disturbances inferred from this type of evidence must have occurred when the dermal ridges were forming, that is during the third to fifth months of intrauterine life.

Method

This investigation consisted of a new analysis, the estimation of the fluctuating asymmetry, of dermatoglyphic material that Mellor (1968) had previously described. This report included the method of obtaining the sample of schizophrenic subjects and their palm and fingerprints, together with the method of dermatoglyphic analysis. These will not be described again in detail, but will be outlined and given with additional information necessitated by recent developments in psychiatric diagnosis.

The subject sample, of 250 female and 232 male schizophrenic in-patients, consisted of acute and chronic subsamples. All subjects were white, of British parentage and resided in the English Midlands before their hospital admission. The acute sub-sample came from four different mental hospitals and consisted of 92 females and 77 males. All were assessed within 48 hours of admission for the inclusion criteria – the presence of first-rank symptoms of schizophrenia in the absence of an organic psychosyndrome. The operational definitions of these symptoms and their frequency within this sample were described in a subsequent publication (Mellor, 1970), as was their diagnostic specificity after an eight-year follow-up period (Mellor *et al*, 1981).

A retrospective examination of the clinical data collected at the same time as the dermatoglyphic data indicated that most of the acute subjects would have met the DSM-III-R criteria for schizophrenic disorder (American Psychiatric Association, 1987). Most first-rank symptoms are listed in the section A criteria and all met section B criteria by virtue of needing hospital admission. The Schneiderian criteria employed in the original study specifically excluded subjects with organic mental disorders. All were below 45 years of age at the onset of the first acute episode. Most subjects were readmissions with lengthy histories of mental illness; however, there were twelve first admissions for whom information about the duration of the illness was incomplete.

The chronic schizophrenics were a 1 in 3 random sample drawn from the chronic wards of three of the four mental hospitals referred to above. All had been in-patients for at least five years and had signs and symptoms that qualified them for membership of one of Leonhard's sub-categories of systematic schizophrenia, as described by Fish (1958). The 158 females and the 155 males who comprised this group were predominantly treatment-refractory patients with positive rather than negative symptoms. The checklist of symptoms used to assign each subject to the Leonhardian categories of systematic schizophrenia was reexamined in the light of the DSM-III-R diagnostic criteria for schizophrenia. All the subjects met the required symptom criteria listed in section A. Subjects with organic brain disease were excluded in the original selection criteria, and all were obviously qualified in the area of severity and duration of illness.

The chronic subjects in one hospital were not palm printed, reducing the numbers available for palm-print analysis to 177 males and 189 females.

Dermatoglyphic fluctuating asymmetry

The methods of estimating dermatoglyphic fluctuating asymmetry will be described in relation to each of the four traits studied. The same method could not be used for each trait, because the data from which the fluctuating asymmetry levels of the control groups were estimated were only available in different formats. In the original study, the control data were obtained from the extensive work on dermatoglyphics in the British population published in a series of papers by workers at the Galton Laboratories (Mellor, 1968). Unfortunately, there was little interest in dermatoglyphic fluctuating asymmetry at that time, but information was published from which the degree of fluctuating asymmetry could be estimated for the fingerridge counts and the atd angles.

Finger-ridge counts

Fluctuating asymmetry was estimated from comparisons made between the ridge counts on the homologous fingers of the right and left hands, i.e. the ridge count of the right first finger was compared with that of the left first finger. As part of a series of publications on the finger-ridge counts in the British population, Holt (1959) provided productmoment correlations (r) of the ridge counts for each pair of fingers and from this a commonly used measure of fluctuating asymmetry, $1-r^2$ (Micle & Kobyliansky, 1988), can be derived. The square of the product-moment correlation coefficient (r^2) of the two variables is a measure of their common variance and $1-r^2$, sometimes known as the coefficient of indetermination (Sokal & Rohlf, 1981), is an estimate of their unshared variance and thus of fluctuating asymmetry.

No correction was made for directional asymmetry as this was small, the mean of the right minus left ridge counts for pairs of digits ranging from -1.88 to -0.466.

Fingerprint patterns

Fingerprint patterns are not morphometric traits, but the types of pattern tend to be identical on homologous fingers; therefore the degree of pattern discordance can be used as a measure of fluctuating asymmetry. Woolf & Gianas (1977) used three basic types of pattern for the purpose of comparison, arches, loops and whorls. Their normal data were used for comparison as none was available for the normal British population.

Paimar atd angles

Instructions for determining the atd angle of the palm were given by Penrose (1954). It is the angle subtended at the most distal t triradius of the palm by the a and d triradii, and is an indirect method of measuring the distal displacement of the t triradius. He also provided data for the normal British population from which estimates of atd angle fluctuating asymmetry could be made. The coefficient of indetermination, $1-r^2$, derived from the productmoment correlations between the right and left atd angles was used as the measure of fluctuating asymmetry, in conformity with the method used for the finger-ridge counts. The variances of the signed right minus left differences could also have been used. No correction for directional asymmetry was made as this was small; the mean of the differences between atd angles was +0.14 for the schizophrenic sample and +0.29 for the normal sample.

Palmar a-b ridge count

The palmar a-b ridge count, the number of ridges lying between the a and b triradii of the palm, was described by Fang (1949) who published data for the normal British population, but not in a form that allowed fluctuating asymmetry to be estimated. The normal data provided by Woolf & Gianas (1977), therefore, had to be used for comparison. This consisted of the variance of the signed right minus left differences. The mean of these differences in the schizophrenic sample was small and no correction for directional asymmetry was required.

Results

The product-moment correlation coefficients for the individual pairs of fingers of male and female subjects, both schizophrenic and normal, are given in Table 1.

The schizophrenic subjects have lower correlations than normal subjects for nine of the ten pairs of digits, and in four pairs the difference is statistically significant.

The estimates of fluctuating asymmetry $(1 - r^2)$ derived from the correlation coefficients are given in Table 2, together with the differences between the schizophrenic and normal groups. As expected from the correlations, the estimate of fluctuating asymmetry was greater in nine of the ten pairs of schizophrenic digits. This pattern of differences between pairs of digits is statistically significant (P = 0.02), calculated directly using the randomisation test for matched pairs (Siegel, 1956, p. 88).

The measures of fluctuating asymmetry are greatest in the second digit pairs of both male and female normal subjects, while it is greatest in the third digit pairs of both groups of schizophrenics.

Fingerprint patterns

The numbers and percentages of right-left fingerprint pairs that did not have the same type of pattern on each finger pair are compared in Table 3. The proportion of discordant pairs belonging to the schizophrenic subjects is significantly

Table 1 Correlations (r) between the right and left ridge counts of individual fingers, normal and schizophrenic subjects

Finger pairs	Normal	Schizophrenia	Difference between r	
Female	(n = 825)	(n = 250)		
i i	0.780	0.741	NS	
11	0.742	0.795	NS	
111	0.768	0.734	NS	
IV	0.830	0.795	NS	
v	0.825	0.741	P<0.005	
Male	(n = 825)	(n = 232)		
1	0.775	0.680	<i>P</i> <0.01	
11	0.768	0.741	NS	
III	0.797	0.720	P<0.025	
IV	0.833	0.757	P<0.01	
V	0.790	0.773	NS	

Table 2Fluctuating asymmetry measure $(1 - r^2)$ of ridge count foreach pair of fingers, difference between normal and
schizophrenic subjects

Finger pairs	Normal (N)	Schizophrenia (S)	Difference (S-N)
Female			-
I I	0.391	0.452	+0.061
II	0.449	0.368	-0.081
III	0.410	0.461	+0.051
IV	0.311	0.367	+0.056
V	0.320	0.450	+0.130
Male			
I	0.399	0.452	+0.139
11	0.410	0.452	+0.042
III	0.365	0.482	+0.117
IV	0.307	0.428	+0.121
v	0.377	0.403	+0.026

-			-
Γ.	ы	-	- 7

Proportion of homologous fingers with discordant fingerprint patterns: differences between normal and schizophrenic subjects, and schizophrenic sexes

Subjects	Fingerprint pairs			
•	Total	Discordant	(%)	
Schizophrenia (n = 482)	2410	556	(23.1) ¹	
Normal (n = 166)	830	149	(18.0)1	
Male schizophrenics				
(n = 232)	1160	268	(23.1)	
Female schizophrenics				
(n = 250)	1250	288	(23.0)	

1. Significance of difference between proportions, z = 3.03, P < 0.005.

higher than that of the normal subjects, using the statistical test advocated by Fleiss (1981, p. 23) for proportions with a specified characteristic in two independent samples.

There was no significant difference between male and female schizophrenics in the proportions of discordant patterns.

Palmar atd angle

The product-moment correlations (r) between the right and left atd angles are given in Table 4 together with the measure of fluctuating asymmetry $(1-r^2)$ for both normal and schizophrenic subjects. The means and variances of the right minus left palmar atd angles are also given. The correlation coefficient for the schizophrenic female sample is significantly smaller than that of the normal female subjects, which means that the former have a greater degree of fluctuating asymmetry of the atd angle. The correlation between the atd angles in the male schizophrenic sample is also lower than that of normal male subjects, but not significantly so. It is of similar magnitude to that of the female schizophrenics, but the normal male subjects have a greater degree of fluctuating asymmetry of the atd angle than the normal females.

Table 4

Palmar atd angle, mean and variance of differences between right and left hand, and correlation (r) and asymmetry estimate $(1 - r^2)$, for schizophrenic and normal subjects

Subjects	п	Difference mean variance		r	1 – <i>r</i> ²
 Eemale					
remaie					
schizophrenia	186	-0.42	(112.9)	0.507'	0.743
normal	485	+0.01	(42.7)	0.717 ¹	0.486
Male					
schizophrenia	177	+0.40	(71.5)	0.548	0.700
normal	510	+0.24	(51.3)	0.627	0.607

1. Significant difference between r, P<0.001.

Palmar a-b ridge counts

The mean and standard deviation of the differences between the right and left palmar a-b ridge counts for the schizophrenic sample was -0.27 (s.d. 4.28). The mean is close to zero and, therefore, no correction for directional asymmetry was made. The variance of the schizophrenic group (18.32) was significantly greater than that of the normal control group (3.30) ($F \pm 5.55$, d.f. = 362/164, P < 0.01), indicating that the schizophrenics had a greater degree of fluctuating asymmetry of the a-b ridge count. There was no significant difference between the males and females who constituted the schizophrenic sample.

Acute v. chronic schizophrenia

All the measures of dermatoglyphic fluctuating asymmetry were examined for differences between the acute and chronic schizophrenic sub-samples. It was predicted that fluctuating asymmetry would be greater in the chronic group as the members were more severely affected by schizophrenia. The findings were in the predicted direction for the finger ridge counts, fingerprint patterns and a-b palmar ridge counts, but not for the palmar atd angles. None of these differences between the acute and chronic subjects reached statistical significance.

Discussion

The schizophrenic subjects exhibited a greater degree of fluctuating asymmetry than the control groups on four dermatoglyphic traits that are largely independent of one another. This study lends support to the findings of Markow & Wandler (1986) and Markow & Gottesman (1989) that a sample of subjects with schizophrenia, on the dermatoglyphic evidence, does have a higher level of fluctuating asymmetry than is found in a normal sample. This increase in fluctuating asymmetry in schizophrenia is relatively small; a rough estimate, based upon the finger-ridge count and the atd angle findings, would be that it is greater by 10 to 20%. Such a small increase is perhaps to be expected as manifest congenital malformations are not part of the schizophrenic syndrome.

However, before discussing the significance of this finding, certain reservations about this study should be declared. The most obvious is the question of the control groups for the a-b ridge count and the fingerprint pattern component of the investigation, for which no normal British controls were available. Despite this, the decision to include these two dermatoglyphic traits was made because they were the only two studied by Markow & Wandler (1986). Although there are racial differences in the frequencies of dermatoglyphic characters and the sizes of ridge counts, there is little information about fluctuating asymmetry.

Another reservation about these results is the inconsistency between sexes of the dermatoglyphic findings, even though the trends are in the same direction. The level of fluctuating asymmetry of the finger-ridge counts is significantly greater for more pairs of fingers in the male schizophrenic sample than in the female. The significant finger pairs are also different. The converse is seen for the atd angle, the fluctuating asymmetry of which is significantly increased in the female schizophrenics, but not in the male. It is difficult to find an adequate explanation for these findings without having more information about normal sexual differences in dermatoglyphic fluctuating asymmetry.

Clinical features of schizophrenia associated with fluctuating asymmetry have not been identified. Markow & Wandler (1986) found it was associated with the severity of the illness. In this study there was no significant evidence to support this finding. This could have been due to the narrow range of severity within this schizophrenic sample; all the subjects, including the acute admissions, were severely ill.

The use of $1-r^2$ as a measure of fluctuating asymmetry was imposed by the form in which the normal data were available. An advantage of this measure is that it is not affected by directional asymmetry. Its major disadvantage, found by Palmer & Strobeck (1986), is that it is the least sensitive of nine different methods of measuring fluctuating asymmetry and is, therefore, more likely to result in type II errors.

Evidence of increased fluctuating asymmetry has relevance for aetiological theories of schizophrenia in the areas of genetics, the prenatal environment and developmental anomalies. These aetiological possibilities are not mutually exclusive and could even be interdependent.

The genetic implications of an increased level of fluctuating asymmetry being found in schizophrenia

were interpreted by Markow & Wandler (1986) in the same way as those for cleft lip (Woolf & Gianas, 1977). The level of fluctuating asymmetry is indicative of the capacity to buffer adverse environmental factors during development and the efficacy of this buffering process is determined by polygenic inheritance. An association between fluctuating asymmetry and a particular disorder suggests that the same multiple alleles play a part in the aetiology of both. The polygenic theory of schizophrenic inheritance proposed by Gottesman & Shields (1967) and elaborated by McGue et al (1983) requires the number of adverse genes in the polygenic system to increase until a threshold is crossed and the trait for schizophrenia develops. Alternatively, the polygenic system may reduce the general buffering capacity of the organism and enhance the aberrant developmental effects produced by a specific gene in a specific organ. It would be compatible with an increased liability to the anomalous development of cerebral asymmetry postulated by Crow et al (1989).

The second possibility is that the fluctuating asymmetry could be due to adverse environmental effects that overwhelm an otherwise adequate buffering system. There is presently no direct evidence linking such intrauterine insults to the subsequent development of schizophrenia. The evidence of an association between the month of birth and schizophrenia is well known and has been recently reviewed in relation to maternal age at birth by Dalén (1988). This association is open to many causal interpretations and linking it to an inimical intrauterine environment is as plausible as any other.

The third possibility is that this aberration of dermatoglyphic development is associated with a deviation of the normal process of brain development. The dermatoglyphic findings would, therefore, lend indirect support to views that minor abnormalities of brain development play a role later in life in the development of schizophrenia. Such views, developing from newer techniques in brain imaging and neuropathology, have been the subject of recent reviews (Murray & Lewis, 1987; Roberts, 1988). The evidence of structural abnormalities of the temporal lobe in the absence of gliosis suggests that neurodevelopmental deviations may play a part in the subsequent development of schizophrenia in some patients. Jakob & Beckman (1989), on the basis of neurohistological findings, have claimed that the parahippocampal gyrus is affected by a failure of cell migration, during the third to sixth months of foetal development, which is the time when the dermal ridges form. Crow et al (1989) have found both in their own and other studies that the abnormalities are predominantly in the dominant temporal lobe

and, as mentioned above, postulated an anomalous development of normal cerebral asymmetry attributable to the effect of a specific gene.

Dermatoglyphic fluctuating asymmetry holds promise as a method of studying schizophrenia. Several questions require further investigation. Are there specific clinical features associated with fluctuating asymmetry which may be identified by using more sensitive measures of fluctuating asymmetry? Family studies may help to clarify, as in cleft lip, the question of the genetic heterogeneity. Fluctuating asymmetry may also help in assessing the relationship between prenatal environmental influences and structural brain changes. Finally, it may help to explain the somewhat perplexing findings in schizophrenia when conventional methods of dermatoglyphic analysis are used.

Acknowledgements

I wish to acknowledge my debt to the late Dr Eliot Slater, Professor L. S. Penrose and Professor E. W. Anderson, whose advice and prescience 25 years ago, in the areas of schizophrenic sampling, dermatoglyphics and clinical diagnosis, respectively, enabled this work to be done. My thanks are due to Justin Mellor for assistance with the computing.

References

- AMERICAN PSYCHIATRIC ASSOCIATION (1987) Diagnostic and Statistical Manual of Mental Disorders (3rd edn, revised) (DSM-III-R). Washington, DC: APA.
- CROW, T. J., BALL, J., BLOOM, S. R., et al (1989) Schizophrenia as an anomaly of development of cerebral asymmetry. Archives of General Psychiatry, 46, 1145-1150.
- DALEN, P. (1988) Schizophrenia, season of birth and maternal age. British Journal of Psychiatry, 153, 727-733.
- FANG, T. C. (1949) A comparative study of the a-b ridge count on the palms of mental defectives and the general population. Journal of Mental Science, 96, 780-787.
- FISH, F. J. (1958) Leonhard's classification of schizophrenia. Journal of Mental Science, 104, 34-54.
- FLEISS, J. L. (1981) Statistical Methods for Rates and Proportions. New York: John Wiley & Sons.
- GOTTESMAN, I. I. & SHIELDS, J. (1967) A polygenic theory of schizophrenia. Proceedings of the National Academy of Science, 58, 199-205.
- HOLT, S. B. (1959) The correlations between ridge-counts on different fingers estimated from a population sample. Annals of Human Genetics, 23, 459-460.
- JAKOB, H. & BECKMAN, H. (1989) Gross and histological criteria for

developmental disorders in the brains of schizophrenics. Journal of the Royal Society of Medicine, 82, 466-469.

- KOBYLIANSKY, E. & MICLE, S. (1986) Handedness and dermatoglyphic direction and fluctuating asymmetry. Zeitschrift fur Morphologie und Anthropologie, 76, 313-329.
- LIVSHITS, G. & KOBYLIANSKY, E. (1987) Dermatoglyphic traits as possible markers of developmental processes in humans. American Journal of Human Genetics, 26, 111-127.
- MARKOW, T. A. & WANDLER, K. (1986) Fluctuating dermatoglyphic asymmetry and the genetics of liability to schizophrenia. Psychiatry Research, 19, 323-328.
- & GOTTESMAN, I. I. (1989) Fluctuating dermatoglyphic asymmetry in psychotic twins. Psychiatry Research, 29, 37-43.
- MCGUE, M., GOTTESMAN, I. I. & RAO, D. E. (1983) The transmission of schizophrenia under a multifactorial threshold model. American Journal of Human Genetics, 35, 1161-1178.
- MELLOR, C. S. (1968) Dermatoglyphics in schizophrenia. Part I: Qualitative aspects. Part II: Quantitative study. British Journal of Psychiatry, 114, 1387-1397.
- (1970) First rank symptoms of schizophrenia: I the frequency in schizophrenics on admission to hospital. II differences between individual first rank symptoms. British Journal of Psychiatry, 117, 15-23.
- , SIMS, A. C. P. & COPE, R. V. (1981) Change of diagnosis in schizophrenia and first rank symptoms: an eight year followup. Comprehensive Psychiatry, 22, 184-188.
- MICLE, S. & KOBYLIANSKY, E. (1988) Sex differences in the intra individual diversity of finger dermatoglyphics: pattern types and ridge counts. Human Biology, 60, 123-134. MURRAY, R. M. & LEWIS, S. W. (1987) Is schizophrenia a neuro-
- developmental disorder? British Medical Journal, 295, 681-682.
- PALMER, A. R. & STROBECK, C. (1986) Fluctuating asymmetry: measurement, analysis, patterns. Annual Review of Ecology and Systematics, 17, 391-421.
- PENROSE, L. S. (1954) The distal triradius t on the hands of parents and sibs of mongol imbeciles. Annals of Human Genetics, 19, 10-38
- & OHARA, P. T. (1973) The development of the epidermal ridges. Journal of Medical Genetics, 10, 201-208.
- ROBERTS, G. W. (1988) Abnormalities in brain structure in schizophrenia. Current Opinion in Psychiatry, 1, 83-89.
- SIBOEL, S. (1956) Nonparametric Statistics. New York: McGraw-Hill.
- SOKAL, R. R. & ROHLF, F. J. (1981) Biometry (2nd edn). New York: W. H. Freeman.
- SOULE, M. (1979) Heterozygosity and developmental stability: another look. Evolution, 33, 396-401.
- & CUZIN-RUDY, J. (1982) Allomeric variation. 2. Developmental instability of extreme phenotypes. American Naturalist, 120, 765-786
- VAN VALEN, L. (1962) A study of fluctuating asymmetry. Evolution, 16, 125-142.
- WOOLF, C. M. & GIANAS, A. D. (1976) Congenital cleft lip and fluctuating dermatoglyphic asymmetry. American Journal of Human Genetics, 26, 400-403.
- (1977) A study of fluctuating dermatoglyphic · & asymmetry in the sibs and parents of cleft lip propositi. American Journal of Human Genetics, 29, 503-507.

Clive S. Mellor, MD, PhD, FRCPC, FRCPsych, Professor of Psychiatry, Memorial University of Newfoundland; *Department of Psychiatry, Health Sciences Centre # 1171, St John's, Newfoundland, Canada A1B 3V6

*Correspondence