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Tics and Heredity
A Study of the Relatives of Child Tiqueurs

D. M. ZAUSMER and M. E. DEWEY

The limited literature on the pedigrees of tiqueurs, including those with Gilles de la Tourette's syndrome, is reviewed. Most statistical analyses have been restricted to affected family members without specifying the unaffected ones. The present statistical analysis of a series of child tiqueurs, including 91 probands and 1293 first- and second-degree relatives, 46 of whom were tiqueurs, predicts the odds on being a tiqueur for individuals, and establishes how those odds are affected by certain explanatory variables using log-linear models. The data do not confirm a familial pattern beyond reasonable doubt, but if the suggested prevalence of tics in the population is 10% then the figure for parents is large enough to support a familial hypothesis. The pedigrees do not indicate a simple mode of genetic transmission. Further research is needed to confirm that there is a connection between childhood tics and Gilles de la Tourette's syndrome, to establish that the predisposition to tics is familial, and, if so, whether there is a complex genetic mechanism involved, or some other environmental aetiology so far undisclosed.

Throughout the literature on tics and Gilles de la Tourette's syndrome (henceforth referred to as Tourette's syndrome) there are innumerable case-reports containing family histories of members with tics, but few large-scale investigations of the pedigrees of child tiqueuers have been published (Mahler *et al*, 1945—34 cases; Zausmer, 1954—96 cases; Torup, 1962—220 cases; Corbett *et al*, 1969—180 cases). The modest literature on the familial nature of Tourette's syndrome and multiple tics is summarised by Kidd *et al* (1980). There are even fewer genetic studies and these relate to patients with Tourette's syndrome (Shapiro *et al*, 1978; Friedhoff & Chase, 1982). It is evident that the debate regarding the inheritance of tics continues. Shapiro *et al* (1972) investigated 34 patients with Tourette's syndrome and found no relationship between this disorder and a family history of tics; they concluded that genetic factors were not involved in the aetiology. This view was later modified (Shapiro *et al*, 1978) and they recorded that the syndrome occurs with increased frequency in relatives of the patients but that there is no discernible pattern of transmission. Moldofsky *et al* (1974) studied 15 patients with Tourette's syndrome and found nine who had at least one first-degree relative with tics; although no specific sex-linked dominant forms of inheritance were seen, a recessive mode of genetic influence was suggested.

A valuable contribution is that of Kidd *et al* (1980), who collected data on Tourette's syndrome and tics for relatives of 75 patients with the syndrome as part of a pilot questionnaire survey. The frequencies of Tourette's syndrome and/or tics among relatives were significantly heterogeneous in a familial pattern. This suggests (a) that the disorder is transmitted, with tics alone being a milder manifestation, and that this severity difference is a threshold phenomenon related to transmission, (b) that the sex difference in prevalence is real because it was found among relatives of both male and female probands, and (c) that the sex difference is related to transmission as a threshold effect since female probands, although less common than male probands, had a higher proportion of affected relatives. These pilot data provided evidence for the transmission of Tourette's syndrome and can be qualitatively explained by a genetic model of transmission. Kondo & Nomura (1982) agreed that these conclusions are fundamentally compatible with their own; however, in an investigation of 43 patients (41 Japanese and 2 Koreans) with Tourette's syndrome they found that among a total of 428 known relatives only nine (2%) showed evidence of Tourette's syndrome or chronic multiple tics. Kidd & Pauls (1982) discussed the genetic hypotheses for Tourette's syndrome and chronic multiple motor tics. They analysed a combined sample containing 421 first-degree relatives of 118 probands using three different models, and concluded that such models can explain the complex transmission pattern observed in families of patients with the syndrome, but that discrimination between

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the quite different models for the inheritance of a complex trait is extremely difficult if the only information available is the trait phenotype.

Other recent genetic research includes the study of HLA typing and red blood cell choline levels in Tourette's syndrome (Comings *et al*, 1982*a*,*b*). Reporting the former investigation, the authors state that the available data are compatible with a two-locus hypothesis, with a major gene for Tourette's syndrome and a modifying gene which decreases the threshold for penetrance. However, their findings from a preliminary study of HLA A and B antigens in 12 patients with Tourette's syndrome and their family members do not point to a predominant HLA A or B type associated with the disease. In the latter investigation, to determine whether red blood cell choline could be used as a marker for Tourette's syndrome, 16 patients and their relatives were studied. The findings confirmed an earlier observation of raised levels in Tourette's syndrome, which seem to be genetically controlled. The authors conclude that although the elevated levels do not appear to be a direct marker for Tourette's syndrome, they may be a marker for genetic or environmental influences that serve to enhance the expression of the major gene(s) for the syndrome.

There is therefore a diversity of hypotheses to explain the familial predisposition to these disorders and a paucity of data to support a genetic theory. In view of this we carried out further statistical analysis of the first author's research findings relating exclusively to child tiqueurs (Zausmer, 1954), both for its historical interest and because of the hypothesised connection between chronic multiple motor tics and Tourette's syndrome (Kidd & Pauls, 1982).

Historical background

During the period 1952–1954 the first author studied 96 child tiqueurs (63 boys and 33 girls) as a research project at the University of Liverpool. The sample is described elsewhere (Zausmer, 1954). In 32% of the families there were relatives who had had tics. There were no recorded members exhibiting features of Tourette's syndrome. The figure of 32% tallies with that of Torup (1962), who found a family history of tics in 30–40% of close relatives, but not with that of Corbett *et al* (1969) who noted it in only 10% of cases. Interestingly, Shapiro *et al* (1978) found that the percentage of family members with tics in their sample of Tourette patients was 34.5.

In 1965 the first author had the good fortune to interest Dr C. A. Clarke (at that time Reader in Medicine at the University of Liverpool) in the analysis of the pedigrees of the research sample of tiqueurs to determine if it was possible to explain the familial transmission of tics on a genetic basis. Clarke summarised his findings as follows (5 of the 96 pedigrees were omitted from the analysis because of insufficient background information). (a) There was a highly significant sex difference (87 males as against 50 females). (b) The impression of family size was that it did not differ significantly from that of the general Liverpool population. (c) Most of the tiqueurs were not the first or last born in the families (see also Table V). (d) The mean ages of the parents (where the ages of both were known) at the birth of the propositus were fathers 31.3 (s.d. 5.7) and mothers 28.3 (s.d. 5.1) years, which was probably typical for the population.

Clarke concluded that no simple genetic system could explain the pattern of cases, assuming they were a homogeneous group. (If they were clinically heterogeneous, the numbers in each class would be too small for statistical analysis). The nearest approach to simplicity would be to invoke a single dominant gene with incomplete penetrance, but the lack of penetrance would be so great as to make it valueless from a practical point of view (56 out of 91 tiqueurs had no family history of tics). Clarke then considered the possibility of a single dominant gene plus a multifactorial background. One might expect that where the sex of the tiqueur was the unusual one (that is, female) there would be more likelihood of her parents (mother) or offspring being affected than would be the case in a male tiqueur. There was no evidence that this was so. Thus, where the propositus was a female there was a positive family history in 11 cases and a negative one in 18 cases. Where the propositus was a male there was a positive family history in 24 cases and a negative one in 38 cases.

This was not statistically significant and our more recent analysis described below confirms this finding. (For this analysis illegitimate births, adoptions and incomplete family histories were omitted.)

Method

Wider availability of more advanced statistical techniques since 1965 has meant that we are now able to explore the pedigree data in more detail. As mentioned above there are 91 families with sufficient detail for us to be able to include them in the analysis: 62 of these index cases were boys and 29 girls. We have at least some data on 1533 relatives of these tiqueurs: 178 siblings, 36 half-siblings, 178 parents, 342 grandparents, and 799 siblings of the parents. We have used the following potential explanatory variables: subject's own sex, sex of the index case, age (classified into five groups—0–4, 5–9, 10–14, 15–19 and 20+ years), relationship to the index case (sibling, half-sibling, parent, grandparent, parental sibling), and parental type (whether or not at least one parent of the tiqueur was affected). Examination of the raw data revealed that some of our 1533 cases had missing values on so many of these variables that including them in the analysis would have been impractical, and the results that follow are based on 1293 cases with almost complete data: 178 siblings, 22 half-siblings, 176 parents, 342 grandparents, and 575 parental siblings. As can be seen, the parental siblings are the group for which data are most often missing on the main explanatory variables. Among the 1293 relatives in our main sample there were 46 who were tiqueurs, 25 males and 21 females.

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TABLE I
Proportion of tiqueurs classified by sex and degree of relationship to index case

	Sex of index case					
	Male		*Female*		*Total*	
Index cases	62		29		91	
	Sex of relative					
	Male		*Female*			*Proportion of*
	Observed	*Expected*	*Observed*	*Expected*	*Total*	*tiqueurs*
Sibling						
Tiqueurs	8	5.0	1	4.0	9	0.05
Total	93	—	85	—	178	—
Half-sibling						
Tiqueurs	0	0.0	0	0.0	0	0.00
Total	9	—	13	—	22	—
Parent						
Tiqueurs	11	13.4	14	11.6	25	0.14
Total	88	—	88	—	176	—
Grandparent						
Tiqueurs	2	3.8	5	3.2	7	0.02
Total	171	—	171	—	342	—
Parental sibling						
Tiqueurs	4	2.8	1	2.2	5	0.01
Total	299	—	276	—	575	—
All relatives						
Tiqueurs	25	—	21	—	46	0.04
Total	660	—	633	—	1293	—

Expected frequencies calculated from the model with sex and degree of relationship included

In Table I we present the raw frequencies broken down by degree of relationship and sex.

Clarke's preliminary analysis (reported above) followed the practice of much of the literature on tics by examining the data in terms of number of families affected. As Wilson *et al* (1978) point out, the fact that a certain proportion of families have at least one affected member tells us relatively little unless we know the size of families concerned. We have therefore analysed the family data using the information on individuals; our prevalence figures may thus be compared with population values directly. We have concentrated on predicting the odds on being a tiqueur for individuals, and analysing how those odds are affected by the explanatory variables. We have used log-linear models (Bishop *et al*, 1975) to examine the effects of the explanatory variables on the odds. The main advantage of this more complex analysis is that we are examining all the possible explanatory variables simultaneously. This means that when we claim that a variable is an important predictor of the odds on being affected by a tic, we are examining the effect of that variable over and above the effect of the other explanatory variables in our model, and we are also able to examine interactions between explanatory variables.

Two of the variables give rise to problems in fitting, parental type and age. Both of these variables have a strong but artefactual association with degree of relationship. In the case of parental type it makes no sense to use this variable in the analysis of the parent data, but we have examined it for the other sets. We wished to include age as an explanatory variable, not because of its scientific interest, but to deal with the problem that younger relatives would not have passed completely through the period of risk and so would show a lower prevalence of tics than a group which was beyond a critical age (hence the choice of age strata). For this reason we only considered age as an explanatory variable for the sibling data. In order to deal with the problem of these two variables we conducted four separate preliminary analyses looking at the different degrees of relationship separately, rather than performing one large analysis. In each of these we fitted a model and then iteratively removed terms from the model, starting from the highest order interactions. Only non-significant terms were deleted, and all lower order terms forming a significant interaction were retained. This is essentially the method outlined by Brown (1976). When it became clear in these analyses that neither parental type nor age would need to be included in the final model, the motivation for the separate analyses disappeared, and we then combined all the data and proceeded with a single analysis using the same approach.

The use of log-linear models makes our method comparable with the methods used by Kidd *et al* (1980). We have used the algorithms of Haberman (1973) and Lustbader & Stodola (1981) to perform the fitting.

Results

The preliminary analyses revealed that age and higher order interactions involving age were not significant even for the

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TABLE II
Proportion of tiqueurs among siblings classified by age

Age	*Tiqueurs*	*Non-tiqueurs*	*Proportion*
0–4	1	49	0.02
5–9	3	43	0.07
10–14	3	38	0.08
15–19	0	19	0.00
20+	2	19	0.11
All	9	168	0.05

(one sibling omitted — missing age)

siblings. This can perhaps be attributed to the small number of tiqueurs in each age group. There is in fact a deficiency of tiqueurs in the younger groups (Table II) but this fails to reach significance in any of our analyses.

Parental type

Parental type and higher order interactions involving parental type also failed to reach significance in the analyses of siblings, grandparents and parental siblings combined. The results are presented in Table III.

TABLE III
Proportion of tiqueurs classified by parental type

Degree of relationship	*Parent of child affected*	*Neither parent of child affected*
Sibling		
Tiqueurs	4	5
Total	36	142
Half-sibling		
Tiqueurs	0	0
Total	7	13
Grandparent		
Tiqueurs	3	4
Total	90	250
Parental sibling		
Tiqueurs	0	5
Total	119	454

Sex and degree of relationship

Sex of the proband and higher order interactions involving sex of proband failed to reach significance in any of the analyses conducted. The final simplest model which provides a good fit to the data is one in which the probability of developing a tic is seen as dependent on degree of relationship to the index case and possibly on an interaction between degree of relationship and sex of the relative. The dominant effect is that of degree of relationship (χ^2 test for including degree of relationship in the model = 54.45; d.f. 4, $P<0.001$). There are more tiqueurs among the parents of the index cases, and also among the siblings. There is some suggestion in the data of an interaction between sex and degree of relationship (χ^2 test for including the interaction when degree of relationship and sex are already in the model = 9.05; d.f. 4, $0.05<P<0.1$). From inspection of the expected values calculated from the model with degree of relationship and sex included it is clear that the main cause of this apparent interaction is the different sex ratio for siblings. Just fitting sex and the component of the interaction due to siblings was significant ($\chi^2=6.15$, d.f. 2, $P<0.05$). Our own feeling is that this interaction will probably not turn out to be a substantive finding, but for completeness we present the χ^3 values and the odds in Table IV.

Odds on being a tiqueur for first- and second-degree relatives

Table IV contains a summary of the two final log-linear models which we have considered. The results are in the form of the odds on being a tiqueur (the probability of being a tiqueur divided by the probability of not being a tiqueur). The table gives the odds for each degree of relationship, and also shows the relative odds (relative to siblings) for the other categories. These can then be interpreted as the increase in relative risk for each degree of relationship. So, for example, we see that the odds for parents are more than three times greater than for siblings. (The odds are conditional on the index case having been identified in the family; they cannot be interpreted as estimates of population risk.)

Birth order

In addition to the other variables mentioned we have some information on the birth order of some of the relatives. Many of the cases have missing data on this variable, but we do have usable information on a subset of the data. Table V shows the relationship between birth order and being affected by a tic for the 418 individuals for whom we have sufficient data. Adding this variable to the log-linear model reveals that there is no relationship between birth order and being affected by a tic, and there is no interaction between birth order and the other variables.

Some characteristics of the pedigrees

Direct transmission through three generations was reported in three families. In one case the male proband had an affected mother and maternal grandmother; in the second case the male proband had an affected mother and maternal grandfather; and in the third case the male proband had an affected father and paternal grandfather.

Of the 28 probands with an affected first-degree relative, 11 indicated at least one other relative who was affected. Within the 20 male probands there were eight instances of the disease being transmitted from an affected male to another male, and the affected relatives were both male and female.

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TABLE IV
Odds estimated from log-linear models using degree of relationship and degree of relationship and sex

Degree of relationship	*Odds on being a tiqueur for a given degree of relationship: likelihood ratio $\chi^2 = 9.33$, d.f. 9*	
	Odds	*Odds relative to sibs*
Sibling	0.053	—
Parent	0.165	3.108
Grandparent	0.021	0.392
Parental	0.009	0.165

Degree of relationship	*Odds calculated from model with degree of relationship and sex and the sibling by sex component of the interaction fitted $\chi^2 = 3.18$, d.f. 3*				
	Male odds			*Female odds*	
	Odds	*Odds relative to sibs*	*Multiplier for females*	*Odds*	*Odds relative to sibs*
Sibling	0.094	—	0.126	0.012	—
Parent	0.150	1.592	1.211	0.023	1.928
Grandparent	0.019	0.200	1.211	0.003	0.243
Parental sibling	0.008	0.085	1.211	0.001	0.103

TABLE V
Proportion of tiqueurs classified by birth order

Birth order	*Non-tiqueurs*	*Tiqueurs*	*Total*
First	91	10	101
Second	91	8	99
Third	77	5	82
Fourth	54	1	55
Fifth or later	73	8	81
Total	386	32	418

There were three cases of tics in both sides of the family.

Among 91 probands there were 21 with one parent affected and two with both parents affected. Only three of the probands with an affected parent had affected siblings. Of the affected siblings, five cases came from a family in which neither parent was a tiqueur.

Discussion

The reliable identification of tiqueurs among relatives of patients is fraught with difficulties. The majority of children's tics disappear before puberty and, as in the case of enuresis, the symptom is frequently forgotten or suppressed when the history is recorded many years later. In spite of careful history-taking it is likely that many relatives who had had tics in childhood were missed. The total familial incidence was probably much higher than the figures obtained.

In the absence of trustworthy estimates of the prevalence of tics in the population it is difficult for us to justify calculating coefficients of hereditability from our data, and indeed we cannot demonstrate beyond reasonable doubt that the data show a familial pattern. If we assume that the prevalence in the population is 10%, as Wilson *et al* (1978) estimate, then the figure for parents is large enough to support the suggestion that tics are familial in origin.

Our results do not appear to indicate any straightforward genetic hypothesis. If the transmission were a simple recessive one we would have expected that the frequency of occurrence of tics would have been higher for siblings than for parents, whereas we found the reverse, even allowing for the fact that the siblings had not yet passed completely through the period of risk.

If there were two sorts of tics, hereditary and environmental, it would seem plausible that we would find differences between the families when we compared members of families where at least one parent of the child was also affected with members of the other families. In fact we found no such differences.

If transmission were multifactorial, with different thresholds for males and females, we would expect that probands of the more rarely affected sex would have more affected relatives. Our current analysis has shown that the relatives (male or female) of the girls are no more likely to be affected, thus confirming for individuals Clarke's original analysis of families. It thus seems unlikely that a single dominant gene with a multifactorial transmission can account for the results.

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TABLE VI

Comparison of the numbers of relatives affected in the present study with those in the Tourette syndrome families studied by Shapiro et al (1978)

	Shapiro et al study						*Present study*					
	Total		*Male*		*Female*		*Total*		*Male*		*Female*	
	n	*Ratio*[1]	*n*	*Ratio*	*n*	*Ratio*	*n*	*Ratio*	*n*	*Ratio*	*n*	*Ratio*
Total number of patients	139	—	—	—	—	—	91	—	—	—	—	—
Patients without Family history of tics	91	65.5	—	—	—	—	56	61.5	—	—	—	—
Patients with Family history of tics	48	34.5	—	—	—	—	35	38.5	—	—	—	—
Family members with tics												
Parents	33	23.7	15	10.8	18	12.9	25	27.5	11	12.1	14	15.4
Grandparents	3	2.2	3	2.1	0	0.0	7	7.7	2	2.2	5	5.5
Siblings (full and half)	8	5.8	6	4.3	2	1.4	9	9.9	8	8.8	1	1.1
Parental siblings	10	7.2	6	4.3	4	2.9	5	5.5	4	4.4	1	1.1
Total relatives	54	—	30	—	24	—	46	—	25	—	21	—

1. Ratio of number of affected relatives to number of index cases.
The figures do not sum in columns because some patients had more than one affected relative

If there were a dominant single gene transmission (even with a reduced penetrance) we would expect to find many pedigrees with direct transmission through three generations. In fact we found only three instances in our sample. The occurrence of male parents of male probands effectively rules out the possibility of the inheritance of tics as an X-linked characteristic.

In Table VI we present a comparison of our findings with those of Shapiro *et al* (1978). They studied the relatives of 139 Tourette syndrome patients and present their results in terms of number of families with affected relatives. We have already argued that this is not the method of presentation of choice, but for comparative purposes we present our own data in the same format. Exact comparison is difficult without information on the number of relatives not affected in each category, but it is clear that the two sets of figures show good agreement. As is the case with our study, Shapiro and his colleagues found a substantial sex difference among patients, but not among relatives.

A comparison of the significant findings of the present study with those of Kidd *et al* (1980), whose sample included relatives of adults affected by Tourette's syndrome, indicates marked differences. They found own sex, sex of index case and parental type to be significant but we did not. Our own study showed that degree of relationship was significant, and that there was a possible interaction between sex and degree of relationships, but Kidd *et al* did not examine these variables. We have no convincing explanation for the resemblance between our findings and those of Shapiro *et al*, or for their joint dissimilarity from those of Kidd *et al*, other than that there are of course considerable differences between the populations, sampling methods and statistical techniques employed.

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David M. Zausmer, MB, BS, MRCPsych, BSc, ABPsS, *Retired Consultant Child Psychiatrist*; *Michael E. Dewey, BA, ABPsS, *Lecturer in Psychological Statistics, Department of Psychiatry, University of Liverpool, Liverpool L69 3BX.*

**Correspondence*

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