THE EUROPEAN SOCIETY OF MEDICINE
Medical Research Archives, Volume 14 Issue 2

RESEARCH ARTICLE

Inheritance and impact of parental sex on clinical expression of Tourette syndrome

Ardiana Ala[1*], Camilla Groth[1], Liselotte Skov[1], Zeynep Tümer[2,3] and Nanette Debes[1,3]

[1]Department of Pediatrics, Copenhagen University Hospital - Herlev and Gentofte, Herlev, Denmark.
[2]Department of Clinical Genetics, Copenhagen University Hospital, Rigshospitalet, Copenhagen, Denmark.
[3]Department of Clinical Medicine, University of Copenhagen, Copenhagen, Denmark.

[*]ardiana-321@hotmail.com

OPEN ACCESS

PUBLISHED
28 February 2026

CITATION
Ala, A., Groth, C., et al., 2026. Inheritance and impact of parental sex on clinical expression of Tourette syndrome. Medical Research Archives, [online] 14(2).

ISSN
2375-1924

ABSTRACT

Background: Tourette syndrome (TS) is a highly heritable neurodevelopmental disorder characterized by multiple motor and vocal tics. While genetic factors play a major role, the potential influence of the sex of the affected parent on symptom expression and development of symptoms remains unclear.

Objective: This study aimed to examine whether maternal versus paternal disposition affects the clinical presentation and longitudinal course of TS in offspring.

Methods: A total of 310 children and adolescents with TS were included from the Danish National Tourette Clinic cohort (baseline 2005–2007; follow-up 2011–2013). Participants were categorized according to parental TS disposition (maternal, paternal, or none). Tic severity was assessed at both time points using the Yale Global Tic Severity Scale (YGTSS). Group comparisons were performed for age at onset, age at diagnosis, tic severity, and symptom progression.

Results: No significant or clinically meaningful differences were found in age at onset, age at diagnosis, or sex distribution between parental and sporadic cases, nor between maternal and paternal transmission. Patients with a parental disposition showed higher baseline tic severity but also slightly greater improvement in tic severity over time compared with sporadic cases. However, the magnitude of these differences was modest.

Conclusion: This study provides the first longitudinal evidence that maternal and paternal inheritance might not have a differential effect on the early clinical presentation of TS. Familial TS may modestly influence long-term symptom trajectories, offering new insight into the heritable mechanisms underlying TS progression.

Keywords: Tourette syndrome, tic disorders, parental inheritance, comorbidity, attention-deficit-hyperactivity disorder, obsessive compulsive disorder.

Introduction

Tic are defined as sudden, rapid, recurrent, non-rhythmic motor movements or vocalizations, called motor or vocal tics, respectively[1]. Tourette syndrome (TS) is characterized by the presence of multiple motor tics and at least one vocal tic, that can wax and wane in frequency, persist for at least one year and have an onset of symptoms before the age of 18 years[1]. The reported prevalence of TS varies between 0.3% and 0.9% in children[2,3]. There is a male predominance, with a male:female ratio being 3-4:1 in children, which decreases with age to a ratio of 2:1 in adults with TS[4]. Tourette syndrome is a multifactorial disorder, where environmental factors and multiple genes play a role in disease progression[5–7]. Recent advances in genetic research have identified potential genetic factors involved in TS. Key candidate genes include *DRD2, DRD4, 5-HT2C* and *SERT*, which are involved in multiple neuronal systems, including dopaminergic, histaminergic and serotonergic pathways[4,8]. While the heritability of TS suggests a strong genetic component, the genetic architecture is complex and current evidence points to a complex, polygenic rather than a monogenic cause. In addition, findings across studies remain inconsistent[4,9].

Tourette syndrome is often accompanied by comorbidities, with about 90% of individuals with TS experiencing at least one comorbid condition, such as attention deficit hyperactivity disorder (ADHD), obsessive compulsive disorder (OCD), sleep disturbances, rage attacks or depression[10,11]. The two most frequent comorbidities are OCD and ADHD, with reported frequencies of 13-66% and 33-91%, respectively, in patients with TS[12,13]. Studies examining co-occurring TS and OCD and/or ADHD in affected individuals and families, suggest an overlap in their etiological factors[4,6].

The familial aggregation of TS is notable, with first-degree relatives of affected individuals having a 10 to 100 times higher risk of developing TS compared to the general population[6].

While TS is highly heritable, little is known about the influence of sex of an affected parent on symptom expression in the offspring. In this study, we aimed to understand whether parental sex had an impact on the TS phenotype in the offspring, including age of symptom onset, age at diagnosis, tic severity, and progression of tic severity over time. Clarifying these patterns could enhance early identification, improve prognosis and support timely initiation of treatment.

Methods

PARTICIPANTS

This study is based on data of a large clinical cohort of children with TS recruited at the Danish National Tourette Clinic during the period 2005-2007 (baseline) and 2011-2013 (follow-up). At baseline 314 patients, aged 5-19 years were included and at follow-up, 227 patients. The longitudinal study was approved by the Scientific Ethical Committees (protocol H-2010-058) and the Danish Data Protection Agency (protocol HEH-2014-002).

All included participants fulfilled the DMS-IV-TR criteria for TS at time of inclusion. A limited number of senior child neurologists with experience in TS had verified the diagnosis at baseline.

CLINICAL ASSESSMENT

All patients were uniformly clinically examined at baseline and follow-up with a clinical interview and validated measurements to assess comorbidities. At baseline, the interview also gathered data on the diagnostic process including age and onset of symptoms, and a pedigree chart was drawn to evaluate whether TS or other comorbidities, i.e., ADHD or OCD, were present in the parents. Genetic disposition was categorized as maternal, paternal, or bilineal (both parents affected). Informed consent was obtained from all the families. Further details about the cohort can be found in Debes. et al.[10] and an overview with cohort characteristics can be seen in table 1.

Table 1. Parental disposition and cohort characteristics

	Paternal disposition (n=78)	Maternal disposition (n=43)	Bilineal disposition (n=11)	Parental disposition (maternal, paternal or bilineal) (n=132)	Sporadic TS (n=178)
Age at baseline (mean (SD))	12.5 (3.0)	12.2 (2.7)	12.2 (2.2)	12.4 (2.8)	12.5 (2.7)
Percentage boys (% (N))	75.6 (59)	83.7 (36)	81.8 (9)	78.8 (104)	84.3 (150)

DIAGNOSTIC TOOLS

Tic severity was rated at baseline and follow-up using the semi-structed validated interview the Yale Global Tic Severity Scale (YGTSS)[14]. The outcome scores total motor tic score (sum of five items (number, frequency, intensity, complexity, and interference) concerning motor tics), total vocal tic score (sum of the same five items concerning vocal tics), total tic score (sum of total motor tic score and total vocal tic score), impairment and global severity score (total tic score plus overall impairment rating) were used in this study.

PARENTAL DISPOSITION

Participants were categorized based on their parents' history of TS and comorbidities. The following groups were defined:

TS disposition refers to a combined group including all parents who reported symptoms of TS, regardless of whether they also reported OCD or ADHD (table 1).

Sporadic group was defined as having no reported symptoms of TS, ADHD, or OCD in either biological parent.

Paternal disposition was defined as a biological father with TS. Maternal disposition was defined as a biological mother with TS. Bilineal disposition was defined as both biological parents with TS. Parental disposition was defined as at least one biological parent affected by TS. Only those subjects with two biological parents were included.

Information regarding parental diagnosis of TS and comorbid conditions (OCD and ADHD) was based on self-reported, retrospective data. Specifically, parents were asked to report whether they had experienced symptoms consistent with TS, OCD, or ADHD at any point in their lives. These reports were not clinically confirmed, and thus do not reflect formal diagnoses but rather self-reported symptoms.

ANALYSIS

Overall, we compared the differences in maternal versus paternal disposition and the difference in parental disposition versus sporadic TS. We also compared the development of tic severity in the same patient categories at follow-up. All analyses were performed in SPSS Statistics (version 29.0. Inc., Chicago, IL, USA) in collaboration with a statistician. Descriptive analyses were used. Dependent on the outcome variable, Fisher´s exact, chi-square, or multivariate general linear model were used in the comparisons between groups. Age and sex were included as confounders in the relevant models. Statistical significance was set at the p=0.05 level. Due to considerable variation in age across participants, corrections for age and sex were applied in relevant comparisons. This adjustment was made to reduce the potential confounding effects of age distribution despite similar group means.

Results

A total of 310 patients diagnosed with TS were included in this study with 132 patients in the parental TS disposition group vs. 178 in the sporadic group. Table 1 shows cohort characteristics.

AGE OF TIC ONSET AND AGE AT DIAGNOSIS

No statistically significant difference in mean age at tic onset was found between the patients with a paternal and maternal disposition (p=0.888).

Patients with a paternal disposition (n=78) had a mean age of 5.2 years at onset, while patients with a maternal disposition (n=43) had a mean age of 5.5 years at onset (table 2).

Table 2. Paternal vs Maternal. Age of onset and diagnosis

	Paternal TS (n=78)	Maternal TS (n=43)	Bilineal TS (n=11)	P-value maternal vs paternal TS
Age of onset (mean (SD))	5.2 (2.9)	5.5 (2.5)	5.9 (2.9)	0.888[1]
Age at diagnosis (mean (SD))	9 (2.7)	8.8 (2.2)	8.9 (2.4)	0.973[1]

[1] Multivariate general linear model with sex as confounders

In terms of age at diagnosis, there was no significant difference between paternal (9 years) and maternal (8.8 years) disposition (p=0.973).

When comparing the parental TS group to patients in the sporadic group, mean age at onset was 5.4 years in the parental TS group and 5.3 years in the sporadic group. In addition age at diagnosis was the same for both groups 8.9 years (table 3).

Table 3. Parental vs. sporadic TS. Age of onset and diagnosis

	Parental disposition (maternal, paternal or bilineal) (n=132)	Sporadic TS (n=178)	P-value for parental TS vs. sporadic
Age of onset (mean (SD))	5.4 (2.8)	5.3 (2.4)	0.954[1]
Age at diagnosis (mean (SD))	8.9 (2.5)	8.9 (2.4)	0.996[1]

[1] Multivariate general linear model with sex as confounders

SEX DIFFERENCES

When comparing paternal disposition to maternal disposition, we found a higher percentage of boys in the maternal group (75.3 % vs 83.3 % boys). However, this did not reach statistically significance (p=0.220) (table 4).

Table 4. Sex distribution

	Paternal disposition	Maternal disposition	P-value maternal vs paternal disp	Bilineal disposition	Parental disposition	Sporadic disposition	P-value parental vs. sporadic TS
Percentage boys (% (N))	75.3 (58)	83.3 (35)	0.220[1]	81.8 (9)	78.8 (104)	69.3 (150)	0.138[1]

[1] Fisher´s exact test

SEVERITY AT BASELINE

A comparison of severity between patients with a parental disposition to TS and those with sporadic TS at baseline revealed a trend with higher severity in all outcome variables in the parental disposition group (table 5).

No significant differences in tic severity at baseline were found between the paternal and maternal disposition groups (table 5).

Table 5. Severity at baseline

	Paternal TS (n=78)	Maternal TS (n=43)	Bilineal TS (n=11)	Parental TS (n=132)	Sporadic TS (n=178)
Total motor tics T1 (median (IQR))	13 (11-16)	14 (12.5-16)	14 (14-15.5)	14 (12-16)	13 (8-15)
Total vocal tics T1 (median (IQR))	11 (0-14)	11 (0-12.5)	9 (0-13.5)	11 (0-13)	9 (0-12)
Total tics T1 (median (IQR))	24 (13-28)	24 (15-27)	27 (14-28.5)	24 (14-28)	20 (11-26.3)
Impairment T1 (median (IQR))	0 (0-10)	0 (0-10)	0 (0-20)	0 (0-10)	0 (0-0)
Global severity score T1 (median (IQR))	24 (13-35)	25 (19.5-34.5)	29 (14-42.5)	25 (15-35)	21.5 (11-29)

DEVELOPMENT OF TICS

At the follow-up assessment, which was done approximately 6 years later, patients with a parental disposition to TS exhibited more severe tics compared to those in the sporadic group.

When examining development in tic severity from baseline to follow-up, significant improvements were seen in vocal tics in the parental group (mean 3.1 (SD 7.5) vs. sporadic group (mean 2.9 (SD 7.1), p=0.048) and in total tics in the parental group

(mean 7 (SD 11.3) vs. sporadic group (mean 6.1 (SD 11.1), p=0.046). In general a trend towards better improvement was seen in the parental group although not all data reached statistical significant.

The same trend was observed in the difference between paternal and maternal disposition, with a trend towards slightly greater improvement in the paternal disposition group (table 6).

Table 6. Severity at follow-up

	Paternal TS (n=78)	Maternal TS (n=43)	P-value maternal vs paternal disposition	Bilineal TS (n=11)	Parental TS (n=132)	Sporadic TS (n=178)	P-value parental TS vs. sporadic
Change motor tic from T1 to T2 (mean (SD))	4.4 (6.1)	2.8 (5.5)	0.494[1]	4 (3)	3.9 (5.8)	3.2 (6.2)	0.299[1]
Change vocal tic from T1 to T2 (mean (SD))	3.2 (7.7)	2.7 (7.2)	0.186[1]	4.2 (7.7)	3.1 (7.5)	2.9 (7.1)	0.048[1]
Change total tic from T1 to T2 (mean (SD))	7.6 (12.3)	5.5 (9.8)	0.179[1]	8.2 (8.9)	7 (11.3)	6.1 (11.1)	0.046[1]
Change impairment from T1 to T2 (mean (SD))	1.1 (14.6)	0.4 (12.3)	0.345[1]	1.7 (14.7)	0.9 (13.8)	-1.5 (11.4)	0.479[1]
Change global severity score from T1 to T2 (mean (SD))	8.7 (23.4)	5.9 (18.1)	0.150[1]	9.8 (20.8)	7.9 (21.5)	4.6 (19.1)	0.109[1]

[1] Multivariate general linear model with age and sex as confounders

Discussion

In this study, we examined whether maternal versus paternal disposition affects the clinical presentation and longitudinal course of TS in offspring. We did not find any significant differences in age at onset, age at diagnosis, or sex distribution between parental and sporadic cases, nor between maternal and paternal transmission. Patients with a parental disposition had higher tic severity at baseline but also slightly greater improvement in tic severity over time compared with sporadic cases.

IMPACT OF PARENTAL DISPOSITION

Only a few studies have investigated the impact of parental sex on the clinical presentation of TS. One study found an earlier onset of TS with maternal transmission compared to paternal transmission and suggested that imprinting effects might be one of the modifying factors influencing the disease progression[15]. The authors also pinpointed that mothers with TS might identify early manifestations in their offspring more easily than fathers, who may be less likely to recognize early symptoms.

This could not be replicated in our study, in which we found that neither the presence of a parental disposition nor the sex of the affected parent was related to the age at symptom onset or the timing of diagnosis in TS.

Differences in inheritance patterns between maternal and paternal transmission have though been observed in other disorders. For example in myotonic dystrophy type 2, maternal inheritance was associated with earlier symptom-onset, independent of the patient's sex[16]. Similarly, in familial retinoblastomas, maternal inheritance has been associated with a significantly earlier presentation of symptoms compared to paternal inheritance[17]. However, these are monogenetic disorders with clearly defined genetic mechanisms. In contrast, TS is a complex, multifactorial disorder in which heritability likely results from the additive effects.

A previous study examining the parental impact of TS showed that parents of children with TS had a higher likelihood of having TS themselves, and in 82.4 % of the cases at least one of the parents had TS features[18]. Additionally, 25.5 % of the children with TS had a bilineal transmission. Compared to the parents of a healthy control group, parents of TS patients had a significantly higher frequency of TS and associated disorders. Our study could not replicate these findings.

A nationwide case-control study included N=1,120 patients with chronic tic disorder or TS and matched them to four controls (N=4,299). They found a correlation between parental psychiatric diagnoses and TS in offspring[19]. The association

between maternal psychiatric diagnoses and TS was stronger than paternal psychiatric diagnoses and TS. The maternal diagnoses included personality disorders (odds ratio 3.1), anxiety disorders (odds ratio 2.6), affective disorders (odds ratio 2.3), and addiction disorders (odds ratio 1.8). Paternal diagnoses included OCD and anxiety disorders, but these associations were not statistically significant[19]. The authors suggested that this may reflect environmental and/or genetic maternal influences.

SEX DIFFERENCES

Among patients with TS, there were more males in the predisposed groups compared to the sporadic group. Furthermore, in the maternal disposition group, a higher percentage of male off-spring was seen compared to the paternal group. These differences were though not statistically significant. Furthermore, our study does not include a longitudinal cohort of offspring to directly assess whether those with an affected parent are at increased risk of developing TS. However, this should be considered in future large-scale longitudinal studies.

Although TS is predominantly a male disorder, several studies have suggested sex differences in the clinical presentation of TS, including variations in the development and severity of tics and comorbidities[20,21].

SEVERITY AND DEVELOPMENT IN TIC SEVERITY

We found higher tic severity at baseline in the disposed group, but the same group also demonstrated greater improvement in severity of particularly vocal tics over time compared to the sporadic group. Our findings suggest that patients with a genetic disposition may experience greater improvement over time than those without a disposition.

One possible explanation for this pattern could be regression to the mean, whereby individuals with more severe symptoms at baseline may show greater relative improvement regardless of intervention. Another possibility is that children with a known familial risk are more likely to receive earlier or more intensive clinical attention and treatment, which could contribute to the observed improvement. However, we did not examine treatment differences between groups in the present study, and this would be an important area for further research to clarify.

No statistically significant differences in tic severity or tic development over time were seen between paternal and maternal groups, although the paternal group tended towards greater improvement.

Lichter et al.[22] also examined tic severity differences between maternal and paternal transmission. They found that paternal transmission was associated with increased vocal tic frequency and earlier onset of vocal tics relative to motor tics, and more prominent ADHD behaviors. On the other hand, maternal transmission was associated with greater motor tic complexity and more frequent non-interfering rituals. It was though a retrospective study based on a small sample size (25 subjects with paternal and 25 subjects with maternal inheritance), limiting its conclusions.

Although some data reached statistical significance across severity, the magnitude of these differences was modest, suggesting that the clinical impact may be limited and should be interpreted with caution.

LIMITATIONS

This study has some methodological considerations. The diagnosis of TS and comorbidities in the parent group was based on self-reported information rather than clinical verification. However, a thorough family history was obtained. Attrition bias must also be considered since the re-participation rate at follow-up was 72% compared to baseline. In this study comorbidities such as ADHD and OCD were not controlled for in the analysis. However, participants and non-participants did not differ significantly in any demographic or clinical characteristics at baseline, as described by Groth et al.[23]. A strength of the study is the use of standardized assessments for tic severity and the inclusion of a large cohort of TS patients.

Conclusion

This study is, to our knowledge, the first to directly compare longitudinal clinical trajectories in TS based on maternal vs. paternal familial disposition. We found no evidence for differences in age at onset, diagnosis, or sex distribution related to the sex of the affected parent, and could therefore not replicate finding from previous studies that have suggested parental disposition effects.

Moreover, our findings contribute novel insight into the longitudinal development of TS severity. Patients with a parental disposition showed higher baseline severity but also slightly greater improvement over time compared with sporadic cases. Although these differences were statistically modest and of uncertain clinical significance, they indicate that familial TS may influence symptom course rather than onset characteristics.

Overall, the results suggest that parental inheritance, whether maternal or paternal, does not meaningfully alter early clinical presentation of TS, while still potentially shaping long-term symptom trajectories. These findings expand current understanding of TS heritability and highlight the need for larger, genetically informed longitudinal studies to clarify the mechanisms underlying these patterns.

Conflict of Interest Statement:

The authors have no relevant financial or non-financial interests to disclose.

Funding Statement:

No funding was received to assist with the preparation of this manuscript

Acknowledgements:

None.

Author contributions

ND visualized the manuscript, conceptualization was attributed to ND and AA. Data analysis was contributed by ND. AA wrote the original draft. AA, CG, LS, ZT and ND reviewed and edited the draft. ND and CG collected clinical data.

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