Genetic and environmental effects on stuttering: A twin study from Finland

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ABSTRACT

The present study explored the prevalence of self-reported stuttering in a Finnish twin population and examined the extent to which the variance in liability to stuttering was attributable to genetic and environmental effects. We analyzed data of 1728 Finnish twins, born between 1961 and 1989. The participants were asked to complete a questionnaire on speech, language, and voice. In two of the questions they were asked to report the occurrence of childhood and present stuttering of their own and that of their sibling. According to the results, 2.3% (52) of the participants were reported to have stuttered as children and 28.8% of them (15) were reported to continue to stutter in adulthood. There was no significant gender difference in the prevalence of stuttering in either childhood or adulthood. For childhood stuttering, the tetrachoric correlation was higher for monozygotic pairs (r = .74) than for dizygotic pairs (r = .27). By means of structural equation modeling it was found that 82% of the variance in liability to childhood stuttering was attributable to additive genetic effects, with the remaining 18% due to non-shared environmental effects. In conclusion, the results of the present study confirm findings from prior studies and support a strong genetic and only a moderate non-shared environmental effect on stuttering. Potential small differences in the prevalence of stuttering in different populations are suggested by our data.

Educational objectives: The reader will be able to recognize the contribution of genetic and environmental effects on stuttering.

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1. Incidence and prevalence of stuttering

According to a review by Guitar (2006), the incidence of stuttering is about 5%. However, there is some variation in the incidence of stuttering in different studies depending on the population and the method used. The incidence of stuttering in three year old children, investigated by speech screening, was 5.2% in Månsson’s (2000) study, while the overall incidence of stuttering was 2.2% in a population study carried out by telephone interviews by Craig, Hancock, Tran, Craig, and Peters (2002).

In twin studies, the reported incidence and prevalence rates vary to some extent. When the population was of adults and self-reports were used, the incidence was 3.2% for males and 1.2% for females in a study by Andrews, Morris-Yates, Howie, and Martin (1991), 8.8% in a study by Felsenfeld et al. (2000) and 5.6–5.8% in younger groups and 4.5% in the oldest group in the study by Fagnani, Fibiger, Skytte, and Hjelmborg (2010).

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When parental reports for childhood stuttering have been used in twin studies, prevalence rates of 7% (Dworzynski, Remington, Rijsdijk, Howell, & Plomin, 2007), 6.7% for males and 3.6% for females (Ooki, 2005) and 4% (van Beijsterveldt, Felsenfeld, and Boomsma, 2010) have been reported. According to Guitar (2006) the overall prevalence of stuttering in kindergarten age is about 2.4%.

1.1. The etiology of stuttering

Historically, there have been a variety of etiological explanations of stuttering. In spite of various approaches, including physiological, behavioral, and psychological aspects, no single theory sufficiently explains the origin of stuttering. Several theories have focused on physiological factors (see e.g., Guitar, 2006). Because various brain areas are involved in the planning and production of speech, there is naturally a risk of disruptions in these processes. According to a review by Yairi (2007), deficient coordination between hemispheres due to deviant brain dominance might be a contributory cause of stuttering. Disturbances in auditory feedback have also been suggested as a possible cause. For example, Foundas et al. (2004) have reported an association between planum temporale asymmetry and auditory feedback disturbance.

There is also strong evidence to support the notion that disturbances in motor control could cause stuttering; that is, stuttering might be a result of deficiencies in neural connections controlling speech (Kent, 2000). When Fox et al. (1996) examined individuals who stutter using positron emission tomography, they found evidence that stuttering could be a disorder where multiple neural systems involved in the speech production may be affected. In a review, Alm (2004) concluded that stuttering might be the result of an impaired ability of the basal ganglia to give motor timing cues which initiate speech. There is also evidence as to how a brain injury in the basal ganglia can cause stuttering for adults (Tani & Sakai, 2011). According to the review by Smits-Bandstra and De Nil (2007), dysfunction in cortico-striato-thalamo-cortical connections can cause difficulties in sequence learning and automatization and be one of the etiological components in the development of stuttering.

Possible explanations of the physiological and psychological factors behind the stuttering may include both environmental and genetic effects (Alm, 2004; Guitar, 2006). Environmental factors, such as growing up in the same family, are considered to be shared environmental factors. On the other hand, factors that influence people on an individual level are considered to be non-shared environmental factors. They can be, for example, illness, injury, or harmful events during prenatal or perinatal development. Premature birth is known as one perinatal risk factor for stuttering (Ajdacic-Gross et al., 2010; Stromswold, 2006).

1.2. The role of genetics

According to a review by Yairi, Ambrose, and Cox (1996) stuttering is far more frequent among relatives of stuttering probands than in the general population. In the reviewed studies the percentages of stutterers with a positive family history of stuttering has ranged from 20% to 74%. In the non-stuttering control groups, the corresponding percentages have ranged from 1.3% to 42%. The variation in the results is likely to depend on differences in definitions of stuttering, the effect of spontaneous recovery, and the imbalanced gender ratio (Yairi et al., 1996).

One way to study genetic predisposition of speech disorders is a twin study. In contrast to family studies, twin studies can help in determining whether familial aggregation is genetic or environmental in nature. Monozygotic (MZ) twins have the same gene collection while dizygotic (DZ) twins share about 50% of their segregating genes (Carey, 2003). Twins often grow up in a shared environment and by making the equal environment assumption (EEA), that is, that the environmental influences are shared to the same extent by MZ and DZ twin pairs, differences between the two twin groups are attributable to genetic variance (Rutter, 2006).

In many twin studies of stuttering the concordance rate – an estimate of the probability that both twins are affected by stuttering if it is known that one of the twins is affected – has been reported to be higher in MZ than in DZ twins (Andrews et al., 1991; Dworzynski et al., 2007; Felsenfeld et al., 2000; Howie, 1981). If significantly more MZ twins than DZ twins are concordant for a certain disorder, it is probable that the difference depends on genetic factors (Rutter, 2006).

In studies from different parts of the world, the results of genetic model fitting analyses of stuttering vary slightly (Table 1). Approximately 70–85% of the variance in liability to stuttering has been found to be attributable to genetic effects, with the remainder due to non-shared environmental effects. The recent results of van Beijsterveldt et al. (2010) indicate that shared environmental factors may be significant for stuttering-like behaviors and high levels of normal nonfluency in young children.

Recent studies in molecular genetics have focused on finding susceptibility genes that might contribute to stuttering. The results of these studies differ from each other, suggesting that stuttering is likely to be a polygenic disorder for which several genes may increase susceptibility (Kang et al., 2010; Lewis, Ricci, Lukong, & Drayna, 2004; Raza, Riazuddin, & Drayna, 2010; Riaz et al., 2005; Shugart et al., 2004). The results of the study of Suresh et al. (2006) and Wittke-Thompson et al. (2007) support linkage on multiple loci throughout the genome. The results of a recent study by Kang et al. (2010) found that a mutation in gene GNPTAB, GNPTG and NAGPA occurred more often in stuttering probands relative to non-stuttering controls. These structures are associated with, for example, emotion and motor function. Expression of these three genes in the human brain is not known and it has to be examined further, but there are data that they have high levels of expression in the hippocampus, hippocampal formation, and cerebellum in mice (Kang et al., 2010). As support to the multiple loci...
### Table 1

Previous studies on genetic and environmental influences on stuttering.

<table>
<thead>
<tr>
<th>Study</th>
<th>Instrument</th>
<th>Measure of stuttering</th>
<th>Twins N</th>
<th>Age</th>
<th>Men</th>
<th>Women</th>
<th>Additive genetic effects (A)</th>
<th>Dominant genetic effects (D)</th>
<th>Shared environment (C)</th>
<th>Non-shared environment (E)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Andrews et al. (1991)</td>
<td>Questionnaire</td>
<td>“Ever stuttering”</td>
<td>7020</td>
<td>Adults</td>
<td>2745</td>
<td>4875</td>
<td>71%</td>
<td>0</td>
<td>0</td>
<td>29%</td>
</tr>
<tr>
<td>Felsenfeld et al. (2000)</td>
<td>Questionnaire and an telephone interview for some of the participants</td>
<td>Stuttering before 14 and older</td>
<td>3768</td>
<td>17–29 years</td>
<td>39.4%</td>
<td>60.6%</td>
<td>70%</td>
<td>0</td>
<td>0</td>
<td>30%</td>
</tr>
<tr>
<td>Ooki (2005)</td>
<td>Parent questionnaire</td>
<td>The frequency of stuttering</td>
<td>3792</td>
<td>3-15 years</td>
<td>1849</td>
<td>1943</td>
<td>9% (males) 56% (females)</td>
<td>71% (males) 29% (females)</td>
<td>0</td>
<td>20% (males) 15% (females)</td>
</tr>
<tr>
<td>Dworzynski et al. (2007)</td>
<td>Parental report</td>
<td>A question concerning stuttering</td>
<td>12,892</td>
<td>Same participants in age 2, 3, 4 and 7 years</td>
<td>Recovered group</td>
<td>.67</td>
<td>Persistent group</td>
<td>.60</td>
<td>Not tested</td>
<td>0</td>
</tr>
<tr>
<td>van Beijsterveldt et al. (2010)</td>
<td>Mother's interview</td>
<td>Questions concerning nonfluency</td>
<td>20,445</td>
<td>5 years</td>
<td>Stuttering</td>
<td>42%</td>
<td>High nonfluency</td>
<td>45%</td>
<td>Stuttering</td>
<td>44%</td>
</tr>
<tr>
<td>Fagnani et al., 2010</td>
<td>Questionnaire</td>
<td>“Do you stutter or have you stuttered?”</td>
<td>33,317</td>
<td>20–71 years</td>
<td>15,138</td>
<td>18,179</td>
<td>82% for males 81% for females</td>
<td>2% for males 0% for females</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>
through the genome Suresh et al. (2007) found, after performing gender-specific analyses, linkage for men and women on different chromosomes.

The estimations of genetic and environmental effects on the liability of stuttering, as well as the prevalence of stuttering vary in some extent depending on the population and the method used in the study. Therefore studies in different populations are indicated. The purpose of the present study was to explore the prevalence of stuttering and to estimate genetic and environmental effects on the liability of stuttering in a Finnish twin population.

2. Method

2.1. Sample

A total of 1728 Finnish twins (555 men and 1173 women), born between 1961 and 1989 completed an Internet-based questionnaire on speech, language, and voice in spring 2006. These participants had initially participated in a study addressing sexuality and aggression (see, for example, Santtila et al., 2007). The invitations to the original study were sent to all Finnish twins in the aforementioned age range. The sample for the present study was recruited in two different ways from those who completed the original questionnaire. The 1289 twins born between 1961 and 1971, had provided the authors their addresses after completing a questionnaire concerning sexuality and aggression. They were all invited to participate in the present study by means of an e-mail and 53% (n=684) of them accepted the invitation and filled in the Internet-based “Speech, language, and voice” questionnaire. The twins born between 1972 and 1989 were asked to fill in the same Internet-based questionnaire after they had completed another questionnaire about sexuality and aggression. There were 5026 participants in this second sample, and 20.7% of them (N = 1044) completed the questionnaire. Thus, the total sample was 1728 participants and the response rate was 27.4%. More information about the data collection of the original study concerning sexuality and aggression can be found in Santtila et al. (2007).

2.2. Measurement

The Internet-based self-report questionnaire on speech, language, and voice, designed by the third author, was intended to give information about self-reported speech, language, and voice disorders. The questionnaire consisted of 69 statements related to speech, language, and voice (Simberg et al., 2009). The answers concerning stuttering are reported in the present study. The questionnaire included a question as to whether the participant had been found to stutter as a child and if he or she still stuttered. The participants also reported whether their co-twin stutters or had stuttered. The first question was “Were you found to be stuttering as a child?” The alternative answers were “Yes”, “No”, and “I still stutter”. The other question was “Was your twin found to be stuttering?” The possible answers to this question were “Yes”, “No”, “He/She still stutters”, and “I do not know/remember”. The Finnish word used in the questionnaire “todettu” means either a professionally made diagnosis or it means that someone other than the respondent has also identified that person as an individual who still stutters or ever stuttered. If the participants answered I do not know/I do not remember, the answers were excluded and coded as missing. In the present study, participants who had recovered from stuttering were defined as having childhood stuttering, while those who still stuttered in adulthood were defined as having persistent stuttering.

In order to examine cross-twin correspondence, a comparison was made between self-reports and co-twin responses for the stuttering items. The percentage of agreement between the answers of the target persons and his or her co-twin was 98.2%, with a κ-value of .585, and p < .001. Because of this exceptionally high cross-twin agreement, the response of the co-twin concerning the occurrence of stuttering in his or her sibling was used in cases where the siblings had not answered the questionnaire themselves. This proxy procedure, called the family history (FH) method, has been used in other behavioral genetic studies; for example, by Ellingson and Slutske (2010) when examining pathological gambling and gambling involvement. This procedure increased the sample to 2948 participants.

2.3. Pairing of twins and decision of zygosity

The participants born between 1972 and 1989 had received individual codes in an unrelated study, which made the pairing of twins possible. The participants born between 1961 and 1971 had taken part in another twin study, but had no individual codes and were paired based on a set of criteria. The most important criteria were date of birth, gender, and reported gender of co-twin. Most of the twins were paired according to this information. In a few cases, several individuals were born on the same day and it was thus not possible to classify the twin pair based on the information mentioned above. In these cases, information from other questions in the “speech, language, and voice” questionnaire was used. Questions related to voice, speech, and communication disorders, as well as medication and allergies, were used as indicators. The pairing of the twins resulted in 1474 complete twin pairs. Zygosity of these participants was determined based on responses to questions about similarity. These questions concerned, for example, difficulty distinguishing between the twins and comments that they are alike as two peas in a pod. The questions are standard questions for twin studies developed by Sarna, Kaprio, Sistonen, and Koskenuvo (1978). The zygosity determination resulted in 470 MZ, 716 DZ, and 288 unclassified twin pairs. Both the same
and opposite sex DZ twins were included. After excluding the twin pairs without known zygosity, the size of the sample was 2372 individuals in 1186 twin pairs.

2.4. Statistical analyses

Statistical analyses were conducted using the software PASW Statistics 18.0 (SPSS Inc., 2010). Chi-square tests were used to compare differences in the prevalence of stuttering between men and women. A Pearson correlation coefficient was calculated in order to determine the relationship between year of birth and stuttering. Significance tests examining the potential effects of gender and year of birth on both childhood and persistent stuttering were derived from the Generalized Estimating Equations module of SPSS. This module appropriately takes into account the dependence between observations of family members.

Chi-square tests were computed to determine potential differences between MZ and DZ twins in relation to the occurrence of stuttering. Tetrachoric correlation and structural equation model fitting procedures were used to estimate the extent to which childhood stuttering was due to genetic and environmental influences. It was not possible to estimate the genetic and environmental influences on persistent stuttering because of the small number of participants with persistent stuttering. The number of affected twin pairs was also not large enough to enable separate analyses for men and women. The model fitting analysis and the estimations of values for the tetrachoric correlations were carried out using Mx Statistical Package (Neale, Boker, Xie, & Maes, 2002).

The tetrachoric correlation analysis estimates the correlation between two variables assuming an underlying continuous distribution (Uebersax, 2009). A threshold divides the continuous variable into two dichotomous variables, affected and unaffected, so that there is an equal probability to exceed the threshold and to be classified as affected instead of non-affected (Reuter, Hüppe, Netter, & Henning, 2003). Assuming a continuous liability to a disorder enables the genetic and environmental contribution to the dichotomous variable to be estimated (Gottesman & Shields, 1967). An equal threshold for both twin groups suggests no between-group differences in regard to the distribution.

Structural equation models were then fitted to the tetrachoric correlations to provide estimates of the proportion of variance in liability to stuttering due to additive genetic effects (A), shared environmental effects (C), and non-shared environmental effects (E). Additive genetic effects (A) refer to the combined influence of different alleles throughout the genome. Shared environmental effects (C) refer to influences that enhance the similarity of individuals growing up in the same family, in contrast to non-shared environmental effects (E), which refer to the effects that are unique for every individual and make them different within the same family. Non-shared environmental factors also include possible measurement errors. As the cohort effect and gender effect can inflate estimates of the shared environment the year of birth and gender were removed from the variables used in the model fitting analysis.

When interpreting the results of the correlation, a higher MZ than DZ correlation suggests that additive genetic effects (A) contribute to the variance. If the DZ correlation is less than half the MZ correlation, dominant genetic effects (D) can also explain at least part of the variance. A DZ correlation less than half the MZ correlation also indicates that there is no influence of shared environmental effects (C). Finally, an MZ twin correlation that is lower than 1.00 indicates that non-shared environmental effects (E) and possible measurement errors contribute to the results.

Different models were compared with each other using the differences in significance (p-values) and the differences in fit (−2 × log-likelihood of data) functions to determine how well each model fit the data. If the tests could not distinguish between different models, these were further compared using Akaike’s information criterion (AIC) (Akaike, 1987). A lower AIC-value indicates a better model fit.

3. Results

3.1. Epidemiological results

The use of both self-reports and the reports of the co-twins, if they had not produced their own answer, enriched the total sample size from 1729 to 2948. Therefore a combination of self and co-twin reports was used for all of the analyses. After excluding the twin pairs without known zygosity, the size of the sample was 2372. Of these, 1385 respondents had answered one or both of the stuttering items and 904 had a co-twin report. There were 83 participants who had neither their own nor a co-twin report and were therefore excluded. Thereby, the final sample size used in the analysis was 2289 individuals (782 men and 1507 women).

The relationship between year of birth and stuttering was not statistically significant either in childhood (r = −.018, B = 0.000, SE = 0.0005 (95% CI, −.001, .001), χ²(1) = .523, p = .469) or in adulthood (r = −.008, B = 0.000, SE = 0.0002 (95% CI, −.000, .000), χ²(1) = .139, p = .709). Similarly, there were no significant differences in the prevalence of stuttering as a function of zygosity (MZ versus DZ) either in childhood (χ²(1) = .001, p = .972) or in adulthood (χ²(1) = 1.142, p = .285). According to the reports 2.3% of the participants (n = 52) had stuttered as children and 0.7% (n = 15) still stuttered (Table 2). This suggests that 71.2% of those who had reported stuttering in childhood had eventually recovered.
Table 2
Occurrence of stuttering after combining the self-reports and the answers of the co-twin in cases where the other twin’s report was missing (N=2289, missing reports 83).

<table>
<thead>
<tr>
<th></th>
<th>Childhood stuttering</th>
<th>Persistent stuttering</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Male MZ</td>
<td>274</td>
<td>2</td>
</tr>
<tr>
<td>Male DZ</td>
<td>481</td>
<td>15</td>
</tr>
<tr>
<td>Female MZ</td>
<td>624</td>
<td>19</td>
</tr>
<tr>
<td>Female DZ</td>
<td>848</td>
<td>16</td>
</tr>
<tr>
<td>Total</td>
<td>2237</td>
<td>52 (2.3%)</td>
</tr>
</tbody>
</table>

Table 3
Model fitting results for childhood stuttering (with 95% confidence intervals).

<table>
<thead>
<tr>
<th>Model</th>
<th>Compared with model</th>
<th>Additive genetic effects (A)</th>
<th>Dominant genetic effects (D)</th>
<th>Shared environment (C)</th>
<th>Non-shared environment (E)</th>
<th>−2 LL</th>
<th>AIC</th>
<th>Δχ²</th>
<th>Δdf</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. ACE</td>
<td></td>
<td>0.82 (0.17, 0.95)</td>
<td>0.00 (0.00, 0.54)</td>
<td>0.18 (0.05, 0.43)</td>
<td>469.45</td>
<td></td>
<td></td>
<td>4102.55</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. AE</td>
<td>1</td>
<td>0.82 (0.57, 0.95)</td>
<td></td>
<td>0.18 (0.05, 0.43)</td>
<td>469.45</td>
<td>4104.55</td>
<td>0.00</td>
<td>1</td>
<td>1.000</td>
<td></td>
</tr>
<tr>
<td>3. CE</td>
<td>1</td>
<td></td>
<td>0.62 (0.38, 0.80)</td>
<td>0.38 (0.20, 0.62)</td>
<td>475.09</td>
<td></td>
<td></td>
<td>4098.91</td>
<td>5.64</td>
<td>0.018</td>
</tr>
<tr>
<td>4. E</td>
<td>1</td>
<td></td>
<td></td>
<td>1.00 (1.00, 1.00)</td>
<td>496.41</td>
<td></td>
<td></td>
<td>4079.59</td>
<td>26.96</td>
<td>0.000</td>
</tr>
</tbody>
</table>

AIC, Akaike Information Criterion; df, degrees of freedom. Models with best model fit are indicated in bold. −2 LL = −2 × log-likelihood of data.

During childhood 2.2% (n = 17) of men and 2.3% (n = 35) of women had reported stuttering. For persistent stuttering, we found that 0.8% (n = 6) of the men and 0.6% (n = 9) of the women were reported to continue to stutter in adulthood. No significant difference was observed between men and women regarding childhood stuttering (B = −0.001, SE = 0.0069 (95% CI, −.015, .012), χ²(1) = .047, p = .829) or persistent stuttering (B = 0.002, SE = 0.0037 (95% CI, −.006, .009), χ²(1) = 0.212, p = .645). Although alternative explanations are possible, the most likely reason for the over-representation of females with stuttering in our sample is the fact that there were twice as many women in the parent sample as men. It should be noted, however, that the confidence intervals for the prevalence estimates of childhood stuttering go from 1.2% to 3.2% for men and from 1.5% to 3.2% for women. This means that given the number of participants in our sample, we cannot exclude that the previously in other samples observed gender difference actually also exists in the Finnish population.

3.2. Tetrachoric correlations

Tetrachoric correlations were calculated for childhood stuttering. The thresholds for MZ and DZ twins were ranked equally without a decrease in model fit, suggesting that there were no differences between zygosity groups in the distribution (χ²(3) = 2.15, p = .542). The MZ tetrachoric correlation r = .74 (95% CI, .48, .89) was higher than the DZ tetrachoric correlation r = .27 (95% CI, −.22, .65), suggesting that genetic effects are involved in childhood stuttering. Because the MZ correlation of .74 is less than 1.0, some influence of the non-shared environment and possible measurement error are also implicated.

3.3. Model-fitting results

Formal model fitting procedures were performed to estimate the extent to which childhood stuttering was due to environmental and genetic influences (Table 3).

Initially, a model including A, C and E components was tested. The components were then omitted one by one. The component for additive genetic effects (A) could not be omitted without a significant decrease in model fit. The component for shared environment (C) could, however, be excluded from the model without significant decrease in model fit. Therefore, the best model in this analysis was the AE model where the additive genetic component accounted for 82% of the variance and the non-shared environment accounted for 18% of the variance.

4. Discussion

The purpose of the present study was to explore the contribution of genetic and environmental effects on stuttering, as well as the prevalence of stuttering in a Finnish twin population. The results confirm findings from prior studies and support a strong genetic and only a moderate non-shared environmental effect for this phenotype.
4.1. Genetic and environmental effects

The genetic modeling results of the present study, that the additive genetic component accounted for 82% of the variance and the non-shared environment accounted for 18% of the variance in stuttering differ slightly from the results of the twin studies by van Beijsterveldt et al. (2010) and Ooki (2005). They found that also shared environmental influences have some effects on stuttering. On the other hand, their methodology differed from that of the present study. They used parental reports regarding the occurrence of stuttering in their children. The results of the present study are, however, very much in line with the results of previous twin studies that have applied self-reporting methodology as was done here (Andrews et al., 1991; Fagnani et al., 2010; Felsenfeld et al., 2000). Fagnani et al. (2010) found in the Danish study the additive genetic effect to be 82% for males and 81% for females and the unique environmental effect to be 16% for men and 19% for females. The studies by Andrews et al. (1991) and Felsenfeld et al. (2000), both done in Australia, found the respective figures to be 71% and 29%, and 70% and 30%. Shared environmental effects did not seem to explain any of the variation in any of these studies applying self-reports of adult participants. It is possible that the method used to collect the data, parental or self-report, influences the results and should be taken into consideration when the results of different studies are compared.

One interesting finding is that the results from the studies carried out elsewhere (Andrews et al., 1991; Felsenfeld et al., 2000) are very similar to one another, as are the results of the present study carried out in Finland and the results of the Danish study (Fagnani et al., 2010). These latter two studies were carried out in Nordic countries, which are situated quite near each other. It is possible that these small variations in heritability reflect cultural differences in response to stuttering items that occur in different populations.

Interestingly the prevalence rates found in the present study were slightly lower than in other self-report studies (Andrews et al., 1991; Fagnani et al., 2010; Felsenfeld et al., 2000). A possible reason for the lower rate of reported stuttering in the present study could be a memory or an “ignorance” factor. All of the participants in the present study were 17 years of age or older. It is possible that some participants who stuttered as children have forgotten it or were never made aware of the problem. This possibility is weakened, however, by the findings of Fagnani et al. (2010) who reported higher prevalence rates than were found here using young adult self-report methodology.

Another possibility for the lower prevalence rates found in the present study may be the way in which the affected persons were identified. The participants in the present study did not receive a stuttering diagnosis by a speech-language pathologist (SLP), largely due to a shortage of SLPS within Finland at that time. Moreover, it is likely that childhood stuttering was never discussed within the family. These factors may have resulted in some under-reporting of stuttering in this sample, particularly for cases where the problem resolved during childhood and were never formally diagnosed or treated.

Finally, though presently very speculative, it is possible that the lower prevalence rates for stuttering could be due to the character of the Finnish language. There are results in the literature that younger speakers show significantly more dysfluency on function words such as articles and prepositions than on content words and with adults the situation is the opposite (Au-Yeung, Gomez, & Howell, 2003; Au-Yeung, Howell, & Pilgrim, 1998; Howell, Au-Yeung, & Sackin, 1999). This has been observed both in English and Spanish speakers. The Finnish language, for example, does not have articles and has very few prepositions. These linguistic variations might, in some respect, facilitate fluency. The effect of language differences on the prevalence of stuttering is an area that should possibly be examined further in a cross-disciplinary study.

4.2. Limitations of the study

It is necessary to examine the results of the present study with a certain caution. One potential limitation is the low response rate. Altogether, 6315 twins received a request to answer a questionnaire concerning speech, language, and voice after they had answered a separate questionnaire on sexuality and aggression. However, only 1385 self-reports and 904 cotwin reports were available. The reason for the attrition could be that 5026 individuals received the invitation to this study just after they had completed a 20 pages long Internet-based questionnaire for another study. It is probable that participants were not so eager or did not have time to continue with another Internet-based questionnaire. It is not very likely that the drop out was due to difficulty accessing the Internet, since, 86% of the adult Finnish population use the Internet (Official Statistics of Finland, 2010).

One limitation of the study was the way how the stuttering was diagnosed. Only two self-reported items formed the basis of the definition if the person had stuttered or continued to stutter. However, this methodology has been used in other genetic studies concerning stuttering (Andrews et al., 1991; Fagnani et al., 2010) and for example dysphonia (Simberg et al., 2009).

Because the sample size was relatively small for a statistical genetic study, the estimates of the model parameters had relatively wide confidence intervals. However, the effects were statistically significant, indicating that there was adequate statistical strength. Similarly, because the number of stuttering affected-cases in our population-based study was small, it was not possible to perform subgroup analyses that were of interest (e.g., examining recovered versus persistent cases or doing gender-specific analyses).
CONTINUING EDUCATION

Genetic and environmental effects on stuttering: A twin study from Finland

QUESTIONS

1. What kind of etiological explanations for stuttering have been presented?
   a. Deficient coordination of hemispheres.
   b. Disturbances in auditory feedback.
   c. Difficulty in motor control.
   d. Disturbances in the functions of basal ganglia.
   e. All the above.

2. Why are twin studies a common method when studying genetic predisposition for speech disorders?
   a. Twin studies can help determine whether familial aggregation is genetic or environmental in nature.
   b. Both monozygotic and dizygotic twins share about 50% of their genes.
   c. Monozygotic twins have the same genetic collection, while dizygotic twins share about 50% of their segregating genes.
   d. Dizygotic twins have the same genetic collection, while monozygotic twins share about 50% of their segregating genes.
   e. Both (a) and (c).

3. When structural equation models are fitted to the tetrachoric correlations of twin pairs they provide estimates of the proportion of variance in liability of stuttering due to
   a. Additive genetic effects (A).
   b. Dominant genetic effects (D).
   c. Shared environmental effects (C).
   d. Non-shared environmental effects (E).
   e. All the above.

4. What effects were found in the present study to account for variance in the liability of childhood stuttering?
   a. Only genetic effects.
   b. Only non-shared environmental effects.
   c. Only shared environmental effects.
   d. Genetic and non-shared environmental effects.
   e. Genetic and shared effects.

5. Which statement is true?
   a. Genetic effects were found to account for 18% and non-shared environmental effects for 82% of the variance in liability of childhood stuttering.
   b. Genetic effects were found to account for 82% and non-shared environmental effects for 18% of the variance in liability of childhood stuttering.
   c. Genetic effects were found to account for 18% and shared environmental effects for 82% of the variance in liability of childhood stuttering.
   d. Genetic effects were found to account for 82% and shared environmental effects for 18% of the variance in liability of childhood stuttering.
   e. None of the above.

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