Original article

The relation of severe malocclusion to patients’ mental and behavioral disorders, growth, and speech problems

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Summary

Background: Severe malocclusions appear in up to 20 per cent of the population. Many neuropsychiatric diseases are likely to have a neurodevelopmental, partially genetic background with their origins as early as fetal life. However, the possible relationship between neurodevelopmental disorders and severe malocclusions is unclear. The aim of this study was in a population-based setting (270 000 inhabitants) to investigate whether patients with severe malocclusions have more mental and behavioural disorders and growth or speech problems than controls without severe malocclusion.

Material and Methods: The study group consisted of patients from the Espoo Health Care Center, Finland, born in year 2000, who were retrospectively screened for their medical and dental records, including their possible mental and behavioural disorders (i.e. attention deficit hyperactivity disorder, Asperger’s syndrome, autism, mood disorder, or broadly defined behavioural abnormalities, learning problems, mental disorders, sleep disturbances, anxiety symptoms, depressive symptoms, and eating-related symptoms) and their need of orthodontic treatment according to the Treatment Priority Index (TPI). The study group consisted of a severe malocclusion group (n=1008; TPI 8–10) and a control group (n=1068) with no severe malocclusion (TPI 0–7).

Results: Patients with severe mandibular retrognatia (P<0.000), lip incompetence (P=0.006), or neurodevelopmental disorders (mental and behavioural; P=0.002) were found to have significantly more speech problems than the controls. The patients with severe malocclusions were leaner, that is, body mass index (kg/m²) <17, underweight; 17–25, normal weight; >25, overweight) than controls (P=0.003), and underweight patients had a significant association with retrognathic maxilla (P < 0.000) compared to normal or overweight patients. No significant relationship between neurodevelopmental disorders and severe malocclusions, that is, retrognatia of maxilla, hypodontia, and severe dental crowding was observed.

Conclusion: Our results indicate that patients with severe mandibular retrognatia, lip incompetence,
Introduction

Severe malocclusions appear in up to 29 per cent of the population (1). Malocclusion is a dento-skeletal disorder that may affect both function and aesthetics of patients and, thereby, cause impairments in their quality of life and social interactions. They may worsen or even cause health problems, such as eating problems, headaches, speech problems, and sleep apnoea (2–6). The recording of malocclusions in the population should be based on an efficient and objective method. According to the Treatment Priority Index (TPI), malocclusions are divided into Classes 1–10. Twenty-nine per cent of patients belong to group 7–10. TPI is an objective method that enables epidemiological surveys of malocclusion without undue cost and energy (7–9). In Finland, patients in TPI groups 8–10, about 20 per cent, are treated at the communal health care centres.

Many neuropsychiatric diseases are likely to have a neurodevelopmental, partially genetic etiology, and their appearance can be visible as early as in utero. Key to the treatment and potential interventions of many neuropsychiatric disorders is early diagnosis. Children with non-syndromic intellectual disability, that is, without existing or diagnosed other comorbid conditions like autism or Down syndrome, might be difficult to discern as other symptoms may be subtle and often missed by clinicians (10). Very few studies exist that link dental development or oral health with neurodevelopmental or neuropsychiatric disorders. An example of comorbidities among individuals with intellectual disabilities is that children with intellectual disabilities have usually been reported to have poorer oral hygiene and higher prevalence of dental caries than their peers (11). To add to the perplexity in diagnosis and management, non-syndromic intellectual disability may be due to genetic alterations at various levels of the genome, which still needs to be identified. A major flaw, for example, in autism spectrum disorder (ASD) management is late diagnosis (12). The role of dentists and especially orthodontists in the early detection of these disorders should be developed due to their access and role in regular follow-up and treatment of large numbers of children and adolescents. The development of the dentition and jaws might serve as a valuable indicator in the early detection of mental and behavioural disorders. The connection between jaw development and mental and behavioural disorders is not clear. On the other hand, malformation of the cranial base structure and individual bones of the upper midface has been shown to be characteristic in many syndromes, for example, in Apert’s syndrome (13). Thus, milder developmental abnormalities may also affect jaw growth and development. However, the possible link between mental and behavioural disorders to severe malocclusions is unclear.

Systematic, unbiased studies that would analyze the potential co-occurrence of neurodevelopmental and neuropsychiatric traits and dental development in unselected samples are hard to conduct. Most of the studies reported so far are relatively small and focused on special, specifically ascertained samples. Finland has, since 1972, had a primary health care system that is based on local health centres and funded by the local public authorities (14). The Finnish health care system follows the development and growth of every child by systematically monitoring every resident from birth to at least age 16 according to TPI (Table 1) (15). This provides a unique opportunity for study designs where entire age groups can be analyzed. Here, we analyzed the potential association of malocclusions with neurodevelopmental or neuropsychiatric traits in one entire age cohort in a mid-size city of approximately 270 000 inhabitants in Southern Finland. There are only minor regional differences in the implementation of orthodontic screening within the city. The socio-economic status of Espoo city is good, usually around the Top 3 in the country.

The aim of the study was to investigate patients with severe malocclusions, determined by TPI 8–10 (1), whether they have more mental and behavioural disorders and growth or speech

| Table 1. The criteria for severe malocclusions (Treatment Priority Index [TPI] 8–10) |
|-----------------------------------------------|-----------------|---|
| Malocclusion | Criteria | TPI |
| Traumatic deep bite | Lower incisors are in contact with palatium | 8 |
| Severe crowding | Crowding of at least one upper canine in one dental arch | 8 |
| Mandible retrognatia | Horizontal overjet is at least 8 mm | 8 |
| Anterior crossbite | Crossbite in the anterior teeth | 8 |
| Posterior crossbite | Crossbite in the posterior teeth | 8 |
| Jaw asymmetry | Severe asymmetry of the upper and/or lower jaw (asymmetry of >5 mm of the midline or on the occlusal plane) | 8 |
| Mandible prognatia | Skeletal prognatia of mandible with anterior crossbite | 8 |
| Maxilla retrognatia | Skeletal retrognatia of maxilla with anterior crossbite | 8 |
| Hypodontia | Hypodontia of one to six teeth causing severe occlusal problems | 8 |
| Maxilla prognatia | Horizontal overjet is at least 8 mm | 8 |
| Scissors bite | Scissors bite in several teeth | 8 |
| Impacted tooth | One or several impacted canines or incisors | 8 |
| Open bite | Severe open bite with maximum molars and second premolars in contact | 8 |
| Oligodontia | Missing at least seven teeth | 9 |
| Syndrome | Syndrome affecting face and or dentition (hemifacial microsomia, etc.) | 8–10 |
| Trauma | Trauma that has affected face/occlusion severely | 8–10 |
problems than controls without severe malocclusion in a unique health care setting where all school age children are screened until at least the age of 16.

**Patients and methods**

**TPI screening for the need of orthodontic treatment**

In Finnish health care centres, a dentist screens the children and adolescents usually every 1–2 years. When the dentist notices that the patient seems to have severe malocclusion, the patient will be sent to a screening by an orthodontist who makes the final diagnosis of the patient’s occlusion according to the TPI score (Table 1). In borderline cases, the orthodontist can follow the patient to see how the occlusion develops. In Finland, dental students are trained nationwide to screen for malocclusion using TPI. In addition, the specialist in orthodontics regularly trains dental hygienists and dentists at his or her health centre to use the TPI index accurately. The financing of Finnish health centres comes from municipal tax money. Thus, family wealth does not affect whether they have access to orthodontic treatment.

**Patients**

Every child living in the Espoo city in Finland is screened using her/his lifelong dental and medical records, including obligatory check-ups to monitor growth and development. Thus, this provides a true population-based register of the entire age group. This retrospective register study consisted of all adolescent patients, born in the year 2000 (study group 1008: boys 49 per cent, girls 51 per cent; controls 1068: boys 51 per cent, girls 49 per cent, age 16 years), who have been screened between 3 and 16 years of age for the need of orthodontic treatment by TPI in the Espoo Health Care Center, Espoo, Finland. All 16-year-old patients from Espoo that had been diagnosed with severe malocclusion (TPI 8–10) were included in the study group. The control group consisted of age-matched patients who did not have severe malocclusion (TPI 0–7). Thus, the research group consisted of 66.7 per cent of the total amount of 16-year-old adolescents in Espoo at 2016 (Table 2). All patients’ medical and dental records were evaluated. All speech disorders from small sound problems to severe speech problems detected in the study were diagnosed and treated by speech therapists. Patients with mental and behavioural disorders were analyzed separately. The data was also divided into two groups: 1. a broad group of neurodevelopmental disorders, where there is well-established evidence for a genetic component (i.e. attention deficit hyperactivity disorder [ADHD], Asperger’s syndrome, autism, mental disorder, mood disorder, behaviour disorder, and learning problems) and 2. disorders or symptoms where the genetic component is less well established (i.e. sleep disturbances, anxiety symptoms, depressive symptoms, and eating-related symptoms).

**Statistical analysis**

Results were generated using a computerized statistical package (IBM SPSS® Statistics 19.0, Armonk, New York, USA). Significance was calculated using Pearson’s chi-square test ($P \leq 0.05$) or a two-sided $t$-test. Presented $P$-values are uncorrected; the significance threshold for each table is presented in the Results section.

**Ethical permission**

The protocol for the retrospective study was approved by the Espoo Health Care Center, Espoo, Finland (13 June 2016). The study did not fulfill the characteristics of a medical study according to the Medical Research Act and did not require ethical permission. The principles outlined in the Declaration of Helsinki were followed.

**Results**

**Patients**

The study group, that is, population with severe malocclusion consisted of 1008 patients (32.4 per cent) out of a total of 3111 patients. The control population consisted of patients without severe malocclusion but receiving dental care at Espoo Health Care Center, that is, 1068 individuals (34.3 per cent). These 2076 patients represent 66.7 per cent of the age group of 16-year-old adolescents in a mid-size city in Finland (Table 2).

The gender, weight, height, and body mass index (BMI) of the study ($s$) and control ($c$) groups at the age of 16 years are shown in Table 3. The BMI in the study group was slightly lower compared to controls ($P = 0.014$, females; $P = 0.0017$, males). Patients, including both sexes, were divided into three groups based on their BMI (<17 underweight ($s = 6.6$ per cent, $c = 3.5$ per cent), 17–25 normal weight ($s = 79.4$ per cent, $c = 78.3$ per cent), >25 overweight ($s = 14.0$ per cent, $c = 18.2$ per cent)). Patients with severe malocclusions were leaner (<17 underweight group) than controls ($P = 0.003$) and being overweight was significantly associated with retrognathic maxilla ($P = 0.000$) compared to normal or overweight patients.

The malocclusion data collected according to TPI are presented in Table 4. The most frequently detected malocclusions were traumatic deep bite (19 per cent), severe crowding (13 per cent), and mandible retrognathia (12 per cent), and anterior crossbite (10 per cent). To adjust for the multiple testing in Table 4, we set the statistical significance level to $P < 0.05/16 = 0.0031$.

The general health variables are present in Table 5. Significantly more speech problems were detected in the study group ($P < 0.001$) compared to controls. All other variables were equally represented in both groups. To adjust for the multiple testing in Table 5, we set the statistical significance level to $P < 0.05/22 = 0.0023$.

| Table 2. The total study and control population of 16-year-old adolescents and the total number of 16-year-old inhabitants in Espoo in 2016 |
|---|---|---|---|
|   | Study population | Boys | Girls | %a |
|   | 1008 | 494 (49%) | 514 (51%) | 32.4 |
|   | 1068 | 514 (51%) | 523 (49%) | 34.3 |
|   | 2076 | 1039 (50%) | 1037 (50%) | 66.7 |
|   | 272 192 |

*aPercentage of the total population of 16-year-old inhabitants in Espoo in 2016.*
The relationship between speech problems, severe mandibular retrognatia, lip incompetence, BMI, and a combined broad group of neurodevelopmental disorders with possible genetic component \( (n = 232; \text{i.e. ADHD, Asperger's syndrome, autism, mental disorder, mood disorder, behaviour disorder, and learning problems}) \) are shown in Table 6. Patients with severe mandibular retrognatia \( (P < 0.000) \), lip incompetence \( (P = 0.006) \), or neurodevelopmental disorders \( (P = 0.002) \) were found to have significantly more speech problems than controls. To adjust for the multiple testing in Table 6, we set the statistical significance level to \( P < 0.05/41 = 0.0012 \). No significant relationship between a broad group of neurodevelopmental disorders with well-established evidence for a genetic component and the studied severe malocclusions, that is, retrognatia of maxilla, hypodontia, and severe dental crowding, was detected.

**Discussion**

We studied the potential association between severe malocclusions, as determined by the TPI (8–10), mental and behavioural disorders and growth and speech problems. In Finland, almost all children are systematically followed by the communal health care system during regular visits from birth to the age of 18 years. During these visits, data on their growth and general and mental health data are collected. In addition, dental check-ups are similarly arranged, where patients are also screened for malocclusions, and those with signs of severe occlusal problems are guided to orthodontic treatment. Thus, our study cohort was representative of children's general and dental health records from birth to 16 years of age in a Finnish mid-sized city of 272 192 inhabitants. The health records of 67 per cent (2076) patients of the age group were included. Despite these comprehensive screening programs, there are shortcomings. The dental check-ups are not every year and the missing 33 per cent of patients consist most likely of patients who have used private dental care or moved to other parts of Finland.

Almost all general health variables were equally represented in both groups. The exceptions were speech problems that were detected significantly more often in patients with severe malocclusion compared to controls. Patients with severe mandibular

### Table 3.
The mean values and standard deviations (SD) by gender (f = female \( n = 1036 \), m = male \( n = 1040 \)), weight (kg), height (cm), and body mass index (BMI; kg/m\(^2\)) of the study and control population at the age of 16 years

| Variable             | Study (f) | Control (f) | Study (m) | Control (m) | \( P \) |
|----------------------|-----------|-------------|-----------|-------------|--------|
| \( n \)              | 514       | 522         | 494       | 546         | 0.483  |
| Height (f)           | 166.2 cm  | 165.8 cm    | 178.1 cm  | 171.7 cm    | <0.0001|
| SD                   | 9.10      | 9.24        | 11.95     | 9.23        | 0.029  |
| Weight (f)           | 59.6 kg   | 61.5 kg     | 67.5 kg   | 68.5 kg     | 0.244  |
| SD                   | 14.42     | 13.52       | 13.92     | 13.73       | 0.014  |
| BMI (f)              | 21.5 kg/m\(^2\) | 22.3 kg/m\(^2\) | 21.2 kg/m\(^2\) | 23.2 kg/m\(^2\) | 0.0017 |
| SD                   | 6.34      | 3.77        | 5.9       | 4.06        |        |

The analysis procedure used was a two-sided \( t \)-test.

### Table 4.
Collected malocclusion variables and their percentage in the research group [the study and the control group], study group, the control group, and \( P \)-values

| Variable                | Research g \% (\( n \)) | Study g \% (\( n \)) | Control g \% (\( n \)) | \( P \) |
|-------------------------|-------------------------|---------------------|------------------------|--------|
| Traumatic deep bite     | 19.2 (398)              | 18.9 (395)          | 0.3 (3)                | <0.000 |
| Severe crowding         | 13.1 (270)              | 13.0 (269)          | 0.1 (1)                | <0.000 |
| Mandible retrognatia    | 12.1 (252)              | 12.1 (252)          | 0 (0)                  | <0.000 |
| Anterior crossbite      | 10.4 (217)              | 10.1 (209)          | 0.3 (8)                | <0.000 |
| Posterior crossbite     | 9.3 (194)               | 8.9 (185)           | 0.4 (9)                | <0.000 |
| Jaw asymmetry           | 8.4 (175)               | 8.3 (173)           | 0.1 (2)                | <0.000 |
| Lip incompetence        | 7.7 (159)               | 7.7 (158)           | <0.1 (1)               | <0.000 |
| Mandible prognatia      | 2.3 (56)                | 2.3 (56)            | 0 (0)                  | <0.000 |
| Maxilla retrognatia     | 5.2 (109)               | 5.2 (108)           | <0.1 (1)               | <0.000 |
| Hypodontia              | 5.5 (115)               | 4.5 (94)            | 1.0 (21)               | <0.000 |
| Maxilla prognatia       | 4.4 (91)                | 4.3 (90)            | 0.1 (1)                | <0.000 |
| Scissors bite           | 4.1 (84)                | 3.6 (73)            | 0.5 (11)               | <0.000 |
| Impacted tooth*         | 3.6 (75)                | 3.6 (75)            | 0 (0)                  | <0.000 |
| Anterior open bite      | 1.8 (38)                | 1.7 (36)            | 0.1 (2)                | <0.000 |
| Severe open bite        | 0.3 (6)                 | 0.3 (6)             | 0 (0)                  | 0.003  |
| Oligodontia             | 0.1 (2)                 | 0.1 (2)             | 0 (0)                  | 0.089  |

All percentages were calculated relative to the entire research group.

*Wisdom teeth were excluded.
Table 5. Collected general health variables and their percentage in the research group, study group, and control group and P-value

| Variable          | Research g % (n) | Study g % (n) | Control g % (n) | P  |
|-------------------|-----------------|--------------|----------------|----|
| JIA*              | 0.3 (7)         | 0.2 (4)      | 0.1 (3)        | 0.648 |
| Type I diabetes   | 0.7 (15)        | 0.2 (4)      | 0.5 (11)       | 0.089 |
| Epilepsy          | 0.4 (9)         | 0.2 (4)      | 0.2 (5)        | 0.505 |
| Sleep apnoea      | 0.2 (4)         | 0.2 (4)      | <0.1 (1)       | 0.289 |
| Cancer            | 0.1 (3)         | 0.1 (2)      | < 0.1 (1)      | 0.092 |
| Syndrome**        | 0.1 (3)         | 0.1 (2)      | < 0.1 (1)      | 0.530 |
| Tourette syndrome | 0.1 (2)         | 0.1 (2)      | 0 (0)          | 0.145 |
| ADHD              | 1.5 (31)        | 0.8 (16)     | 0.7 (15)       | 0.731 |
| Asperger          | 0.2 (5)         | <0.1 (1)     | 0.2 (4)        | 0.184 |
| Autism            | 0.3 (6)         | 0.1 (2)      | 0.2 (4)        | 0.456 |
| Speech problems   | 28.2 (587)      | 16.0 (333)   | 12.2 (254)     | <0.001 |
| Developmental disorders | 1.7 (35)   | 0.8 (16)   | 0.9 (19)   | 0.7 |
| Sleep problems    | 1.9 (39)        | 1.0 (20)     | 0.9 (19)       | 0.731 |
| Mental health trait | 8.0 (167) | 4.1 (85)    | 3.9 (82)      | 0.527 |
| Anxiety disorder  | 4.5 (93)        | 2.0 (41)     | 2.5 (52)       | 0.378 |
| Mood disorder     | 1.0 (21)        | 0.7 (14)     | 0.3 (7)        | 0.095 |
| Depression        | 3.9 (82)        | 1.6 (34)     | 2.3 (48)       | 0.190 |
| Other psychosis   | 0.1 (3)         | 0.0 (1)      | 0.1 (2)        | 0.399 |
| Substance abuse   | 0.6 (13)        | 0.3 (7)      | 0.3 (6)        | 0.702 |
| Behavioural problems | 2.6 (54)   | 1.5 (32)    | 1.1 (22)      | 0.110 |
| Learning problems | 2.7 (57)        | 1.4 (29)     | 1.3 (28)       | 0.722 |
| Eating problems   | 4.5 (92)        | 2.3 (47)     | 2.2 (45)       | 0.619 |

All percentages are calculated relative to the entire research group. To adjust for the multiple testing, we set the statistical significance level to P < 0.05/22 = 0.0023.

* Juvenile rheumatoid arthritis.
** Tourette excluded.

Table 6. The relationship between speech problems and patients with or without severe mandibular retrognatia, with or without lip incompetence, and the combined group of neurodevelopmental disorders with possible genetic component (i.e. ADHD, Asperger’s syndrome, autism, mental disorder, mood disorder, behaviour disorder, and learning problems)

| Variable                      | Speech p. % (n) | No recorded speech p. % (n) | P % (n) | P-value |
|-------------------------------|----------------|-----------------------------|--------|---------|
| Mandibular retrognatia. (232) | 40.9 (103)     | 59.1 (149)                  | <0.000 |         |
| No mandibular retrognatia. (1824) | 26.5 (484)    | 73.5 (1340)                 |        |         |
| Lip incompetence (159)        | 37.7 (60)      | 62.3 (99)                   |        | <0.000  |
| No lip incompetence (1914)    | 27.5 (526)     | 72.5 (1388)                 |        | 0.006   |
| Neurod. disorders (232)       | 37.1 (86)      | 62.9 (146)                  |        | 0.002   |
| No neurod. disorder (1844)    | 27.2 (501)     | 72.8 (1343)                 |        |         |

P-values are uncorrected.

Kovalenko et al. (16) found that orthognathic patients with severe malocclusions and facial deformities had significantly higher prevalence of emotional instability, introversion, anxiety, and unsociability. They suggested that patients with severe facial deformities might be more prone to psychological distress, depression, and adverse psychological reactions.

The results of Cabrita et al. (17) strengthen the connection between malocclusion and intellectual disability. In their small study of 123 patients with intellectual disability and 79 with no impairment, they showed that Class III cases were present almost exclusively in the intellectual disability group (91.7 per cent). Linear regression indicated that having an intellectual disability seem to have a tendency to severe or very severe malocclusion.

The role of dental professionals, especially orthodontists, is an important position from which to detect children with neurodevelopmental disorders due to the regular dental check-ups. Children with intellectual disabilities have usually been reported to have poorer levels of oral hygiene and higher prevalence of dental caries than their peers (18). In a cross-sectional study, Makkar et al. (11) studied the prevalence of caries in 269 children (52 with cerebral palsy, 35 with Down syndrome, 30 with autism, and 152 with non-syndromic intellectual disability without any known comorbid conditions) using the intelligence quotient as an indication. They found that the level of caries in permanent teeth and oral hygiene deteriorated with increasing severity of non-syndromic intellectual disability. Children with non-syndromic intellectual disability, that is, without existing or diagnosed other comorbid conditions like autism or Down syndrome, might be difficult to discern as other symptoms may be subtle and often missed by the clinicians (10).

Many studies have shown the prevalence of malocclusion among children with ASD to be higher when compared to the normal population. Fontaine-Sylvestre et al. (19) found a significantly higher
prevalence of malocclusion in Canadian children aged between 5 and 18 years with ASD (n = 99) compared with controls (n = 101). Children with ASD were significantly more likely to have malocclusion than non-ASD, especially posterior crossbite.

A major challenge in ASD management is late diagnosis. The role of dentists seems to be important also in the early diagnosis of ASD. The eruption schedule of primary teeth has been shown to differ in ASD. ASDs and related developmental conditions are typically diagnosed between 2 and 4 years of age (16, 20). Gozes et al. (12) found in a survey of 54 families with various activity dependent neuroprotective homeobox gene mutations early deciduous teething among 81 per cent of children with an almost fully erupted dentition, including molars by 1 year of age, and only 10 of the children had teeth within the normal developmental time range. Usually at 12 months, children rarely display more than the incisor teeth (21).

**Conclusion**

Our results indicate that patients with severe mandibular retrognatia, lip incompetence, or a broad group of neurodevelopmental disorders with well-established evidence for a genetic component were found to have significantly more speech problems than controls. Patients with severe malocclusions were likely to be leaner than controls, and underweight patients had more often retrognathic maxilla. Patients with speech problems and severe malocclusion should receive special attention during orthodontic treatment to detect signs of possible neurodevelopmental disorders as early as possible.

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**Conflict of interest**

The authors declare that they have no conflict of interest.

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