# A Multiple Stepwise Logistic Regression Analysis of Trauma History and 16 Other History and Dental Cofactors in Females With Temporomandibular Disorders

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*The simultaneous contribution of 11 occlusal factors, dental attrition severity, orthodontic history, trauma (motor vehicle accident [MVA] and non-MVA), and age in defining two independent large populations of females diagnosed with five mutually exclusive temporomandibular disorders was tested through multiple stepwise logistic regression analysis. Non-MVA trauma was significant in both groups in defining disc displacement (DD) with and without reduction, and osteoarthrosis (OA) (both primary and following DD). Anterior open bite was also a significant factor in defining OA in both groups. Much smaller contributions were also made by missing teeth in one of the populations with OA following DD, and by retruded contact position–intercuspal position slide lengths and overjet in one of the primary OA populations. Motor vehicle accident trauma was significant in defining myofascial pain (MP) in both populations, and laterotrusive attrition mildly defined MP in one population. Only a minority of total variance was explained: 6% to 8% of DD with reduction; 10% to 14% of DD without reduction; 11% to 20% of OA following DD; 17% to 38% of primary OA; and 4% to 10% of MP. Non-MVA trauma was the major defining feature of the temporomandibular joint intracapsular disorders, and MVA trauma explained a very small percentage of the MP patients. Implications are discussed and recommendations are made for future research.*
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**key words:** temporomandibular disorders, trauma, occlusion, age

The investigation of potential etiologic factors in temporomandibular disorders (TMD) has yielded very little evidence that any one factor is a significant contributor to disease development. The failure to identify a consistent set of major contributing etiologic factors has been explained by the multifactorial hypothesis: that TMD is caused by so many factors acting simultaneously and in such a wide variation of individualized patterns, that no one single factor is able to explain more than a minor proportion of the disease development.

Despite the paucity of studies designed to examine a direct etiologic link to TMD, a great deal of circumstantial evidence supports direct trauma to the mandible as an etiologic factor in some subjects. Trauma was shown to be the strongest factor differentiating symptomatic from asymptomatic nonpatients,[1] and a direct blow

Table 1 Age Characteristics of the Study I Population*

| Group | n | Mean age | SD | Range |
|---|---|---|---|---|
| Asymptomatic control | 52 | 40.02 | 15.66 | 21–74 |
| DD with reduction | 64 | 33.32 | 10.39 | 17–63 |
| DD without reduction | 41 | 31.56 | 8.96 | 18–59 |
| OA with DD prior history | 66 | 34.84 | 8.51 | 21–54 |
| Primary OA | 81 | 44.96 | 14.60 | 21–78 |
| Myofascial pain | 84 | 35.88 | 12.47 | 13–72 |

*All females. DD = disc displacement; OA = osteoarthrosis.

Table 2 Age Characteristics of the Study II Populations*

| Group | n | Mean age | SD | Range |
|---|---|---|---|---|
| Asymptomatic control | 32 | 33.78 | 10.44 | 21–62 |
| DD with reduction | 34 | 33.32 | 10.39 | 17–63 |
| Asymptomatic control | 28 | 33.04 | 12.13 | 21–62 |
| DD without reduction | 19 | 32.06 | 11.22 | 20–59 |
| Asymptomatic control | 30 | 32.07 | 9.43 | 21–54 |
| OA with prior DD history | 31 | 32.52 | 8.00 | 21–54 |
| Asymptomatic control | 30 | 49.23 | 13.46 | 24–74 |
| Primary OA | 33 | 50.44 | 12.64 | 24–72 |
| Asymptomatic control | 28 | 30.43 | 7.25 | 21–47 |
| Myofascial pain | 30 | 30.23 | 7.58 | 18–47 |

*All females. DD = disc displacement; OA = osteoarthrosis.

to the mandible can result in a temporomandibular joint (TMJ) effusion.[2,3] A scintigraphy study by Harris et al[4] describes posttraumatic changes within the TMJ. Direct blows to the mandible from contact sports[5] or by physical abuse or accidents[6] have also been reported to initiate TMD symptoms.

Although hyperextension injury to the head and neck with no direct blow to the face is suggested as a possible cause of TMD,[7,8] most of the evidence for this hypothesis is anecdotal,[7] and a direct etiologic role for indirect trauma has yet to be established.[9,10] One study of TMD incidence following nondirect impact showed no significant TMD development, even when the cervical spine demonstrated whiplash.[11] Other studies[12,13] failed to demonstrate TMJ injury based on models of indirect extension-flexion trauma to the head and neck. Thus, previous studies suggest a selective role for direct trauma and a doubtful role for indirect trauma in TMD development.

Pairwise study of trauma provides a very limited perspective that is also not very biologic because single factors can rarely act in isolation in biology. Especially because of the medicolegal implications of injury, it is extremely important to describe any relationship according to its strength of association. Information about the TMJ in particular has been largely lacking from most published pairwise medical studies examining trauma, and this brings greater relevance to this multifactorial study. To the knowledge of the authors of the present study, no earlier studies have attempted to simultaneously evaluate trauma as a cofactor with other history, demographic, or occlusomorphologic dental variables in characterizing differentiated groups of TMD from asymptomatic control subjects and estimate the strengths of association. The authors were also fortunate in this study to be able to carry out the modeling on one data set (Study I) and validate it on a second data set (Study II). Thus, the analysis was performed on two independent patient populations, which lends greater credence to agreements between the results.

The null hypothesis tested was the following: differentiated TMD in females is not explained by trauma or orthodontic history, age, or dental attrition and other dental occlusal factors.

## Materials and Methods

### Populations

**Control Sample (Asymptomatic Subjects).** A pool of 52 females without any current or past symptoms or signs of TMD or orofacial pain was screened from a previously described population of dental and dental hygiene students[14] supplemented by consecutive patients across a wider age range from a general dental practice (DAS), using an established protocol.[14] All of these control subjects were used in Study I (Table 1). In Study II, between 28 and 32 of these control subjects were selected through age matching to the diagnostic groups (Table 2). Age matching was necessary in Study II because age proved to be a significant cofactor in three of the five patient diagnostic groups studied in Study I.

**Experimental Sample (Patients With TMD).** The experimental population consisted of consecutive female patients attending the University of California at Los Angeles Pain Management Center Orofacial Pain Clinic, and a private practice setting (DAS). These patients were assigned to five mutually exclusive diagnostic groups employing

previously discussed inclusion and exclusion criteria[15,16] that closely compare with those published later by the American Academy of Orofacial Pain.[17] The female patient sample used in Study I was taken from an original large sample population of males and females previously analyzed for disease association with occlusal variables.[18] The female patient sample used in Study II was taken from an original large sample population of males and females previously analyzed for disease association with attrition severity.[19]

*Disc displacement with reduction* (Study I: n = 64; Study II: n = 34) was defined by clinical signs of TMJ reciprocal clicking whereby the joint sounds occurred at different positions on the mandibular movement pathways, and the clicks were reduced through chairside trial mandibular repositioning. There was an absence of clinical signs (TMJ pain, crepitation) and radiographic signs (condylar erosion, regressive or proliferative changes) associated with osteoarthrosis.

*Disc displacement without reduction* (Study I: n = 41, Study II: n = 18) was defined by a history of TMJ clicking prior to the sudden occurrence of a restriction in mouth opening, and an absence of clinical and radiographic signs of osteoarthrosis. All cases were examined during the acute locking stage.

*Osteoarthrosis of the TMJ with a prior history of TMJ derangement* (Study I: n = 66; Study II: n = 31) was defined by the clinical presence of crepitation and/or localized TMJ pain (osteoarthritis), plus radiographic signs of significant intracapsular osseous changes. The patients had to have reported a history of a prior mandibular movement restriction associated with a TMJ interference characterized by sudden sustained locking. Although this classification depended partly on the remembrance of sometimes distant sudden locking history, it was believed to be reliable because sudden onset locking not resolving for months or years is not likely to be forgotten over time.

*Primary osteoarthrosis* (Study I: n = 81; Study II: n = 33) was defined as in *osteoarthrosis of the TMJ with a prior history of TMJ derangement*, but without any known earlier history of mandibular movement interference because of TMJ locking.

*Myofascial pain* (Study I: n = 84; Study II: n = 30) was defined by the presence of four or more sites of moderate to severe tenderness in the masticatory musculature on palpation[14] and the absence of any symptom or sign of an intracapsular TMD.

A hierarchical system was used for the diagnoses, so that the presence of criteria for an intracapsular classification took precedence over a myofascial pain diagnosis. Thus, the intracapsular patients may also have had myofascial tenderness, but none of the classified myofascial pain patients had clinical evidence of intracapsular disease (disc displacement or osteoarthrosis). In all cases, the subjects' rights were protected and informed consent was granted.

### Procedures

**Clinical Examination.** All subjects were examined according to the same strict criteria.[14] All completed a questionnaire about their symptoms and history, and they were given a lengthy interview followed by a complete clinical examination.[20]

**Radiographic Examination.** To identify patients with osteoarthrosis, the clinical groups with TMJ dysfunction were also examined by bilateral serial tomographic imaging in the sagittal and frontal planes. The scans were blindly interpreted by an experienced independent oral radiologist. Only those patients with radiographic changes judged by the radiologist to be extensive enough for a diagnosis of osteoarthrosis were included in the two osteoarthrosis classifications.

The possibility of misclassification in the other groups was considered by the authors, but a recent magnetic resonance imaging (MRI) study concluded that an accurate diagnostic assignment can be expected without radiographs or MRI from a clinical examination and history alone.[21] The authors estimated from the evidence of past studies[21] that no more than 5% of subjects, or 19 of 388, might be misdiagnosed, which is not a large enough risk to warrant the cost of 388 MRI studies or radiation exposure in subjects without intracapsular disease.

**Occlusomorphologic Data.** The 11 occlusal variables studied in the females were the same factors evaluated in an earlier study population of both males and females[18]: anterior open bite; unilateral maxillary lingual crossbite; retruded contact position–intercuspal position (RCP-ICP) slide length; RCP-ICP slide asymmetry; overbite; overjet; dental midline discrepancy; unilateral RCP contact; number of missing posterior teeth; the greater of the mesiodistal interarch relationship discrepancies (right or left) at the first molar location; and first molar interarch relationship asymmetry (right versus left). In Study II, a population was available with additional data on dental attrition collected in an original study sample of both males and females and data on anterior tooth attrition severity; mediotrusive and laterotrusive attrition severity in the posterior dentition were in-

cluded with the 11 other occlusal factors examined in Study I.

Length and direction of occlusal RCP-ICP slides were measured clinically. The remaining occlusomorphologic data were collected by measurements using calipers or rule, or by counts from dental casts registered in nonreversible hydrocolloid impressions. Nonextracted third molars were only counted when they replaced a more mesial missing posterior tooth. Overbite was recorded as the greatest central incisor vertical overlap, while open bite relationships were recorded as negative. Overjet was recorded as the least horizontal overlap between incisors. The dental midline discrepancy between the opposing arches was measured with a millimeter rule. Unilateral maxillary lingual crossbite was recorded from observations of the dental casts, and this was the only type of crossbite with sufficient prevalence to permit statistical analysis. The mesiodistal discrepancy in the intercuspal relationship between the buccal groove of the mandibular first molar and the mesiobuccal cusp tip of the maxillary first molar was measured as a continuous representation of Angle Class, with zero as neutro-occlusion, negative for a distal relationship, and positive for a mesial relationship. The greater of the right- or the left-side discrepancies was used in the analysis. The difference between the right- and left-side measurements was also utilized as a measure of intra-arch dental asymmetry.

Dental casts were graded for severity and location of wear facets as described in a previous publication.[22] All scoring was performed by one calibrated observer after consensus evaluation by both authors of 10 casts selected at random. The severity scoring of dental attrition was a contraction of the method by Richards and Brown[23] abbreviated to fewer groups because of the inability to absolutely identify dentin exposure on dental casts. All scoring was performed according to the following five-point scale: 0 = no wear; 1 = minimal wear; 2 = noticeable flattening parallel to the occluding planes; 3 = flattening of cusps or grooves; and 4 = total loss of contour and/or dentinal exposure when identifiable. The facets were graded in seven zones: incisor; right canine; left canine; right premolar and molar laterotrusion; left premolar and molar laterotrusion; right premolar and molar mediotrusion; and left premolar and molar mediotrusion. The worst score finding was recorded in each zone. The maximum possible anterior score was thus 1 incisor and 2 canine zones × 1 facet location × maximum severity score of 4 = 12. The maximum possible laterotrusion score for the posterior dentition was thus 2 premolar and 2 molar zones × 2 facet locations × maximum severity score of 4 = 32. The maximum possible mediotrusion score for the posterior dentition was thus 2 premolar and 2 molar zones × 1 facet location × maximum severity score of 4 = 16. Examiners were blinded to patient identity and disease status.

**Age and History Factors.** Age was included as a variable in Study I, but not in Study II, where age matching between the patients and the control subjects made the inclusion of age redundant. In Study II, a very close age match between the experimental groups and each respective control population was achieved (Table 2).

Three history factors were also included in the analysis in both Study I and Study II: prior orthodontic treatment; notable trauma from a motor vehicle accident (MVA trauma); and notable trauma history not from a motor vehicle accident (non-MVA trauma). These factors were originally screened from a questionnaire blinded as to diagnosis, but an interview was used to confirm the questionnaire answers. Orthodontic treatment history was considered positive if the subject experienced a complete treatment intervention with an orthodontist. Motor vehicle accident trauma was considered positive if the subject remembered the onset of significant pain or disability following a traumatic incident related to a motor vehicle, and non-MVA trauma was considered positive if the subject recalled the onset of significant pain or disability following one or more traumatic incidents not related to a motor vehicle.

### Statistical Analysis

Only female subjects participated so as to control for gender. To control for age effects, age was tested as a variable in Study I (see Table 1), and the experimental and control groups were matched for age in Study II (see Table 2). A multiple stepwise logistic regression model was used to simultaneously assess the relative odds of each of 18 potential contributing age, morphologic, and history factors while controlling for the other 17. The outcome was always the disease classification versus the asymptomatic control subjects.

Multiple stepwise logistic regression is a method for deciding which of a list of potential predictors are associated with presence or absence of disease. This method identifies which of the potential predictors discriminate between diseased subjects and nondiseased control subjects. The stepwise logistic procedure first determines the subset of potential predictors that simultaneously discriminate. For

Table 3 Disc Displacement With Reduction: Significant Contributing Factors From Multiple Stepwise Logistic Regression Analysis*

| Factor | Est | OR | $R$ | $P <$ | Entry OR | $R^2$ | $G^2$ |
|---|---|---|---|---|---|---|---|
| Study I | | | | | | | |
| Non-MVA trauma | –0.200 | 1.96:1.00 | .286 | .028 | | | |
| Age | 0.821 | 1.18:1.00 | –.009 | .010 | 32 years | | |
| | | | | | | 11.3% | 7.7% |
| Study II | | | | | | | |
| Non-MVA trauma: | 0.916 | 5.00:1.00 | .422 | .014 | | | |
| | | | | | | 7.6% | 5.7% |

*Est = estimate; OR = odds ratio; Entry OR = measure where the OR is 1:1. $R^2$ and $G^2$ represent adjusted values.

each selected factor in this subset, the model also provides an estimate of the odds ratio that the factor can differentiate the patient from the control subject while simultaneously controlling for the other selected factors. The odds ratio does not explain the risk for new disease, which would require incidence data.

Intervariable correlations were determined to identify potential interactions among the 18 predictor variables, but none of these correlations were strong. A few potential interactions with trauma were suspected through stratifying an array of the means (continuous variables) or prevalences (nominal variables) of the other variables according to the presence or absence of trauma history, and these were included as additional factors in the logistic model.

Two measures of model fit were used in this study to determine the proportion of disease variation that is accounted for by the selected variables. The $G^2$ measure is the proportion of the log likelihood accounted for in the logistic model, and the $R^2$ measure is the squared correlation between the observed disease status (yes or no) and that predicted by the model.

It is important to emphasize that the kind of data analysis utilized in this study cannot support any etiologic associations. Thus, while some of the associations were significant, this does not imply any etiologic role for the identified factors in the disease process. The reader is thereby cautioned not to infer any etiologic relationship to factors described as "defining," "contributing," "explaining," "characterizing," "differentiating," or "predicting." Similarly, "odds ratios" only serve to estimate the strength of any significant associations for factors identified as contributing through the multifactorial analysis, and this concept should not be interpreted to suggest any etiologic role for these factors.

## Results

### Disc Displacement With Reduction

Non-motor vehicle accident trauma history was a significant factor in both Study I and Study II in differentiating the control subjects from patients with the disease classification of disc displacement with reduction (Table 3), with the odds ratio for disease in Study I at 1.96:1.00 ($P < .028$), and in Study II, 5.00:1.00 ($P < .014$). Age was a significant factor in Study I with an odds ratio of 1.18:1.00 and a very small though significant negative correlation of –.009 ($P < .01$). This means that this disease is correlated with younger age ranges with the 2.00:1.00 odds ratio recommended by the authors as the minimally discernible clinical threshold being exceeded at 27 years and younger. No other factors remained in the regression equation. The amount of explained variance in both studies was small (Study I: $R^2 = 11.3\%$, $G^2 = 7.7\%$ ; Study II: $R^2 = 7.6\%$, $G^2 = 5.7\%$).

### Disc Displacement Without Reduction

As with disc displacement with reduction, non-MVA trauma history was a significant factor in both Study I and Study II in differentiating the control subjects from the patients (Table 4). In Study I, the odds ratio for disease with non-MVA trauma was 1.94:1.00 ($P < .004$); in Study II, it was 6.45:1.00 ($P < .002$). In addition, age was a significant factor in Study I, but with an imperceptibly small odds ratio and a very small negative correlation of –.011 ($P < .003$), meaning that this disease is correlated with younger age ranges. However, the 2.00:1.00 odds ratio threshold for age was never reached. The contribution of non-MVA trauma history in accounting for the variance in

Table 4 Disc Displacement Without Reduction: Significant Contributing Factors From Multiple Stepwise Logistic Regression Analysis*

| Factor | Est | OR | $R$ | $P <$ | Entry OR | $R^2$ | $G^2$ |
|---|---|---|---|---|---|---|---|
| Study I | | | | | | | |
| Non-MVA trauma | −0.032 | 1.94:1.00 | .398 | .004 | | | |
| Age | 0.991 | 0.00:1.00 | −.011 | .003 | 32 years | | |
| | | | | | | 18.9% | 14.0% |
| Study II | | | | | | | |
| Non-MVA trauma | 1.171 | 6.45:1.00 | .522 | .002 | | | |
| | | | | | | 17.6% | 10.4% |

*Est = estimate; OR = odds ratio; Entry OR = measure where the OR is 1:1. $R^2$ and $G^2$ represent adjusted values.

Table 5 Osteoarthrosis With a Prior Disc Displacement History: Significant Contributing Factors From Multiple Stepwise Logistic Regression Analysis*

| Factor | Est | OR | $R$ | $P <$ | Entry OR | $R^2$ | $G^2$ |
|---|---|---|---|---|---|---|---|
| Study I | | | | | | | |
| Non-MVA trauma | −0.988 | 5.37:1.00 | .311 | .011 | | | |
| Anterior open bite | 4.354 | 155.00:1.00 | .491 | .006 | | | |
| Missing posterior teeth | 0.172 | 1.15:1.00 | .064 | .004 | 1 tooth | | |
| Age | −0.991 | 0.98:1.00 | −.008 | .018 | 32 years | | |
| | | | | | | 20.4% | 20.1% |
| Study II | | | | | | | |
| Non-MVA trauma | 1.021 | 5.55:1.00 | .429 | .005 | | | |
| | | | | | | 10.9% | 10.6% |

*Est = estimate; OR = odds ratio; Entry OR = measure where the OR is 1:1. $R^2$ and $G^2$ represent adjusted values.

both studies was small, although slightly greater than with disc displacement with reduction (Study I: $R^2$ = 18.9%, $G^2$ = 14.0%; Study II: $R^2$ = 17.6%; $G^2$ = 10.4%).

### Osteoarthrosis With Prior Disc Displacement

As with both disc displacement groups, non-MVA trauma was a significant contributor in differentiating the control subjects from the patients in Study I (odds ratio for disease = 5.37:1.00, $P$ < .011), and it was the only significant contributor in Study II (odds ratio for disease = 5.55:1.00, $P$ < .005) (Table 5). In Study I, the presence of an anterior open bite was a powerful additional predictor (odds ratio for disease = 155.00:1.00, $P$ < .006). The number of missing posterior teeth was also a significant differentiator (entry odds ratio for disease at one tooth missing = 1.15:1.00, $P$ < .004, with the 2.00:1.00 threshold of significance reached at five or more missing teeth), as was age (entry odds ratio for disease at 32 years = 0.98:1.00, $P$ < .018, with the 2.00:1.00 threshold of significance reached at age 27 years and younger). The amount of variance accounted for by the combined factors was similar to the disc displacement groups (Study I: $R^2$ = 20.4%, $G^2$ = 20.1%; Study II: $R^2$ = 10.9%, $G^2$ = 10.6%).

### Primary Osteoarthrosis

As with the other intracapsular disease groups, non-MVA trauma was a significant contributor in differentiating the control subjects from the patients in both Study I (odds ratio for disease = 18.98:1.00, $P$ < .0001), and in Study II (odds ratio for disease = 5.45:1.00, $P$ < .007) (Table 6). The presence of anterior open bite was also a powerful predictor in both Study I (odds ratio for disease = 8.42:1.00, $P$ < .037) and Study II (odds ratio for disease = 101.20:1.00, $P$ < .031). Two other factors also contributed significantly in Study I: namely the length of the RCP-ICP occlusal slide (entry odds ratio for disease = 1.31:1.00, $P$ < .011, at 0.43 mm of slide, with the 2.00:1.00 threshold of significance reached at 4 mm or more of slide), and overjet

**Table 6** Primary Osteoarthrosis: Significant Contributing Factors From Multiple Stepwise Logistic Regression Analysis*

| Factor | Est | OR | $R$ | $P <$ | Entry OR | $R^2$ | $G^2$ |
|---|---|---|---|---|---|---|---|
| Study I | | | | | | | |
| Non-MVA trauma | 0.250 | 18.98:1.00 | .650 | .0001 | | | |
| Anterior open bite | 1.438 | 8.42:1.00 | .327 | .037 | | | |
| Non-MVA trauma slide | −1.077 | 1.28:1.00 | .312 | .010 | 0.05 mm | | |
| RCP-ICP occlusal shift | 0.588 | 1.31:1.00 | .211 | .011 | 0.43 mm | | |
| Overjet | 0.289 | 1.28:1.00 | .073 | .017 | 2.38 mm | | |
| | | | | | | 26.0% | 38.2% |
| Study II | | | | | | | |
| Non-MVA trauma | 1.003 | 5.45:1.00 | .447 | .007 | | | |
| Anterior open bite | 3.924 | 101.20:1.00 | .435 | .031 | | | |
| | | | | | | 13.6% | 16.8% |

*Est = estimate; OR = odds ratio; Entry OR = measure where the OR is 1:1. $R^2$ and $G^2$ represent adjusted values.

**Table 7** Myofascial Pain: Significant Contributing Factors From Multiple Stepwise Logistic Regression Analysis*

| Factor | Est | OR | $R$ | $P <$ | Entry OR | $R^2$ | $G^2$ |
|---|---|---|---|---|---|---|---|
| Study I | | | | | | | |
| MVA trauma | 0.651 | 3.83:1.00 | .276 | .002 | | | |
| | | | | | | 6.6% | 4.2% |
| Study II | | | | | | | |
| Laterotrusion attrition | 0.086 | 1.29:1.00 | .035 | .009 | 15.036 pts | | |
| MVA trauma | 0.664 | 3.88:1.00 | .273 | .042 | | | |
| | | | | | | 13.5% | 9.5% |

*Est = estimate; OR = odds ratio; Entry OR = measure where the OR is 1:1. $R^2$ and $G^2$ represent adjusted values.

(entry odds ratio for disease = 1.28:1.00, $P < .017$, at 2.38 mm of overjet, with the 2.00:1.00 threshold of significance reached at 6 mm or more of overjet). One interaction was also a significant contributor, RCP-ICP occlusal slide length when there was a history of non-MVA trauma history (entry odds ratio for disease = 1.28:1.00, $P < .010$, at 0.05 mm of slide length, with the 2.00:1.00 threshold of significance reached at 4 mm or greater of slide length). The amount of variance accounted for by the combined factors was moderate for Study I ($R^2 = 26.0\%$, $G^2 = 38.2\%$) and lower for Study II ($R^2 = 13.6\%$, $G^2 = 16.8\%$).

### Myofascial Pain

Motor vehicle accident trauma was a significant factor in both Study I (odds ratio for disease = 3.83:1.00, $P < .002$) and Study II (odds ratio for disease 3.88:1.00, $P < .042$) (Table 7). Laterotrusive attrition severity, which could only be evaluated in Study II, was an additional significant factor in Study II (entry odds ratio for disease = 1.29:1.00, $P < .009$, at an attrition score of 15.036 of a total of 32 possible points, with the 2.00:1.00 threshold of significance reached at a score of 19 or greater). The contribution of MVA trauma history in accounting for the variance in Study I was small ($R^2 = 6.6\%$, $G^2 = 4.2\%$), and the additional contribution of laterotrusive attrition severity in Study II only increased the explained variance modestly ($R^2 = 13.5\%$, $G^2 = 9.5\%$).

## Discussion

### Trauma History

The present study demonstrates that non-MVA trauma history is a moderate differentiating feature of patients with intracapsular TMD—namely, disc displacement with and without reduction, and

osteoarthrosis in both patients with primary disease or with disease preceded by a derangement history. This conclusion is supported by the consistently elevated odds ratios for disease and high levels of significance with non-MVA trauma history in both of the two large independent populations (see Tables 3 to 7). The odds ratios, which are proxies for the relative risk for disease, reached or exceeded the 2.00:1.00 threshold, the level at which clinically meaningful relationships are considered demonstrable. The amount of variance explained by non-MVA trauma history ranged from moderate ($R^2$ = 42% in osteoarthrosis with a disc displacement history in Study I) to modest ($R^2$ = 8% for disc displacement with reduction in Study I). These levels were higher than most of the dental morphologic variables presented in a previous study.[18]

We hypothesize that a notable proportion of nonvehicular head trauma in females includes direct head injuries sustained in physical or sexual abuse, a suspicion supported by recent evidence that such histories are common. Curran et al[24] described that in an anonymous survey 28.9% of children and 23.3% of adults experienced multiple instances of sexual abuse. The figures for physical abuse were also notable: 42.2% of children and 22.2% of adults. The Curran et al[24] study and our present study employed nonanonymous means for data collection of history variables; therefore, the prevalence was probably underreported. The patient admissions of non-MVA trauma history are thereby probably conservative, and the factor may be more influential than what we are reporting.

In contrast to many opinion articles about trauma etiology, the contribution of motor vehicular accident history studied in the present article as a cofactor among multifactorial variables was absent from intracapsular disorders, but it was significant in the myofascial pain samples in both the present study populations (see Table 7). Nevertheless, the MVA trauma history as a cofactor in the myofascial pain groups only accounted for about 4% of the variance ($G^2$). In a random population, the percentage contribution of MVA trauma would be expected to be even less than what we reported, because of several factors. We presume that we evaluated treatment-seeking populations whose thresholds for disease acquirement were low enough to permit disease onset, and who probably contained a psychologic predisposition to seek treatment in our tertiary orofacial pain management center. Furthermore, we presume that a portion of these patients would have had lowered thresholds because of latent or pre-existing disease. Just as for MVA cervical injury, it is expected that most stresses and strains to the mandibular system will heal. The recovery rate at 2 years for cervical injury is reported to be 82%.[25] However, no similar data are available for TMD as yet, and the recovery rate needs to be built into studies of the prevalence of TMD-related MVA injury.

An earlier pairwise study by Pullinger and Seligman[26] who investigated trauma history has been overinterpreted and misapplied in the medicolegal arena. There is an inherent difficulty in interpreting prevalence data, especially when the analysis utilizes pairwise testing. This is because of the inability both to estimate strengths of association and to control for a host of potential confounding variables. More importantly, *prevalence does not prove or explain etiology.* Although differences in the prevalence of trauma history had been shown in TMD groups, these differences do not establish a causal relationship to the TMD, and chi square analysis is unable to address the level of contribution of trauma in defining the samples. Thus, it is important to not exaggerate the results in this earlier study[26] in testimony proposing an etiologic relationship between a specific traumatic event and the onset of a TMD. In contrast, the authors stressed the enhanced relevance of the kind of analysis used in the present study that was able to determine odds ratios and strengths of association. A causation study, however, would require an incidence model.

As a cofactor, the contribution of MVA trauma to myofascial pain was very low (less than 4%), and did not remain in the regression equation in the intracapsular TMD. In trauma, the energy of impact has to go somewhere, and its biologic consequences are not zero. However, the effects on the TMJ and its associated musculature are weak when studied in group analysis. This deduction from grouped data cannot, however, be applied so precisely to individual case histories.

### Attrition

The amount of the variance for myofascial pain in Study II that is accountable by laterotrusive posterior tooth attrition was exceedingly small, 0.1% ($R^2$, Table 7). The authors consider this level of contribution to be clinically irrelevant and note that attrition dropped out from the regression equations in intracapsular TMJ disease. The present study, as well as other recent studies by Pullinger and Seligman[19] and Seligman et al,[22] supports the conclusion that TMJ symptoms and intracapsular disease may have no meaningful relationship to the cumulative attrition record, which

is a cumulative record of functional and parafunctional wear. It is now accepted that myofascial pain is a multifactorial problem with notable central components, and, in our study, 99.9% of the variance was unexplained by this single factor. According to the present study, most myofascial pain patients would remain unidentified according to their attrition record, and there is no convincing evidence to date that bruxism is a disease, per se.

The concept of endogenous trauma through bruxism producing disease is certainly not validated in the present study. This does not, however, imply that a sudden parafunctional event combined with a newly introduced occlusal interference or instability might not produce adverse loading. Nevertheless, this study points out that the majority of heavy bruxers do not experience muscle pain, and it supports the model that symptomatology in bruxers is mostly dependent on a variety of other host factors.

### Occlusal Factors

A recent study of a sample population of both males and females by Pullinger et al[18] showed a clinically limited but statistically significant contribution of several occlusal factors in differentiating TMD groups. In the present analysis, some of the previously identified factors became nonsignificant, although most (anterior open bite and missing posterior teeth in osteoarthrosis with a derangement history, and anterior open bite, RCP-ICP occlusal slide, and overjet in primary osteoarthrosis) remained at similar odds ratios as previously reported (see Tables 5 and 6). The notable absence of posterior crossbite appearing as a significant predictor in the present study may be explained by the all-female population, which showed much lower prevalences of this occlusal condition than did males, who were well represented in the earlier study.[18]

The overall message in both of these present studies limited to females (Studies I and II) and in the earlier study[18] with males and females remains the same, namely that the contribution of occlusal variables as cofactors is very small.

### Orthodontic History

Orthodontic treatment history never remained significant at any level of the regression analysis for any of the disease groups in both samples, and it was dropped in early iterations. Thus, this group analysis study supports the conclusion of many other studies[27] that the relative risk of orthodontic treatment in precipitating TMD in general is minimal. Nevertheless, it is reasonable to consider that the application of force on articular tissues over time, or inducing a sudden orthopedic change or instability in specific individuals with low thresholds for disease acquirement or latent or pre-existing disease, might still increase the chance for TMD onset. Thus, the authors believe that it remains important to screen for latent TMD symptomatology in all patients who present for orthodontic treatment.

### Limitations of This Study and Future Research

The present study cannot comment directly on etiology because it is a cross-sectional study based on prevalence data. Consequently, some of the factors that were not statistically significant may still have an etiologic influence in the disease process; others that were significant may not be etiologic. Thus, although some factors did not appear to be significant overall, this does not mean that they cannot influence disease in certain instances and it is important to not overinterpret the results when evaluating a specific individual.

The most important finding from the present study is the consistent contribution of non-MVA trauma in differentiating patients with intracapsular TMD. Apparently the prevalence of non-MVA trauma is very high in our society, and the link to TMD onset is a reasonable avenue of future investigation in developing the trauma model. The authors suggest that a more refined model be developed for evaluating trauma as a potential etiologic factor. However, the contribution of trauma in accounting for the variance was still only between 8% and 42%, which supports the multifactorial profile of TMD.

The present study suggests that MVA trauma and bruxism have been overemphasized in the past, so the authors recommend that future research concentrate on other head trauma. The recently exposed high prevalence of physical and sexual abuse[24] suggests that these kinds of trauma should receive closer scrutiny.

Because this study is based on cross-sectional retrospective prevalence data, it suffers from the same shortcomings of most past TMD investigations, despite the sophisticated statistical analysis. Thus, the etiology of TMD remains unknown, and this study should be viewed as a preliminary analysis for suggesting directions for future research in the development of the multifactorial disease model for TMD through longitudinal, prospective incidence studies. The information from this prevalence study should be helpful in identifying the factors that might be included in an incidence study.

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## Resumen

Análisis de Regresión Logística Escalonado Múltiple de Antecedentes Traumáticos y Otros 16 Antecedentes y Cofactores Dentales en Mujeres con Desórdenes Temporomandibulares

Se examinó la contribución simultánea de 11 factores oclusales, la severidad de la atrición dental, los antecedentes ortodóncicos, el trauma (accidentes automovilisticas [AA] y accidentes no automovilisticos [ANA]), y la edad para definir dos poblaciones grandes e independientes de mujeres diagnosticadas con cinco desórdenes de la articulación temporomandibular mutuamente exclusivos; por medio del análisis de regresión logística escalonado múltiple. El trauma generado por ANA fue significativo en ambos grupos al determinar el desplazamiento del disco (DD) con y sin reducción, y la osteoartrosis (OA) (tanto primaria como después del desplazamiento del disco). La mordida abierta anterior fue también un factor significativo al determinar la osteoartrosis en ambos grupos. La contribución de los dientes ausentes en una de las grupos con osteoartrosis luego del desplazamiento del disco, como también la longitud de los deslizamientos entre Relación Céntrica y Oclusión Céntrica, y la sobremordida horizontal en una de las poblaciones con OA primaria fue mucho menor. El trauma automovilístico fue significativo al determinar el dolor miofacial (DM) en ambas poblaciones, y la atrición laterotrusiva definió ligeramente el DM en una población. Sólo se explicó una minoría de la variación total: 6% a 8% de los desplazamientos de disco con reducción; 10% a 14% de los desplazamientos de disco sin reducción; 11% a 20% de las osteoartrosis luego de los desplazamientos de disco; 17% a 38% de las osteoartrosis primarias; y 4% a 10% del dolor miofacial. El trauma por ANA fue la mayor característica determinativa de los desórdenes intracapsulares de la articulación temporomandibular; y el trauma por AA definió un porcentaje muy pequeño de los pacientes con DM. Se discuten las implicaciones y se dan recomendaciones para investigaciones futuras.



## Zusammenfassung

Eine multiple schrittweise logistische Regressionsanalyse der Traumaanamnese und 16 anderer anamnestischer und dentaler Kofaktoren bei Frauen mit Myoarthropathien

Der Beitrag von 11 okklusalen Faktoren, des dentalen Attritionsgrads, der orthodontischen Anamnese, von Traumata (Verkehrsunfälle und Nichtverkehrsunfälle) und des Alters bei 2 unabhängigen weiblichen Patientengruppen mit 5 Myoarthropathiediagnosen wurde untersucht. Als statistischer Test wurde die multiple schrittweise logistische Regressionsanalyse verwendet. Die nicht durch Verkehrsunfälle verursachten Traumata waren bei den Gruppen mit Diskusluxationen und Arthrosen signifikant. Ein anteriorer offener Biß trat bei beiden Gruppen signifikant oft zusammen mit Arthrosen auf. Viel kleiner war der Beitrag durch fehlende Zähne bei einer Gruppe mit Arthrose nach Diskusluxation. Ein kleiner Zusammenhang bestand auch zwischen primärer Arthrose und der RK-IK-Differenz und Overjet. Verkehrsunfälle waren in beiden Gruppen für Muskelschmerzen signifikant. Laterotrusive Attrition war nur bei einer Gruppe mit Muskelschmerzen signifikant. Nur ein kleiner Teil der Veränderungen konnte erklärt werden: 6%–8% der Diskusluxationen mit Reduktion; 10%–14% der Diskusluxationen ohne Reduktion; 11%–20% der Arthrosen, die einer Diskusluxation folgten; 17%–38% der primären Arthrosen; und 4%–10% der Muskelschmerzen. Nicht duch Verkehrsunfälle verursachte Traumata waren der häufigste Grund für intrakapsuläre Beschwerden; Verkehrsunfällle können nur einen kleinen Teil der Muskelschmerzpatienten erklären. Folgen und Vorschläge für zukünftige Forschungsarbeiten werden diskutiert.