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Evidence and Mechanisms of Embodiment in the Developing Dentition

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Abstract

Existing research supports both embodiment of stress through epigenetics and epigenetic impacts on dental development. Two models for embodiment of stress in dental development are proposed. Stress-delay proposes that increased stress during development will produce delays in dental development. Inflammation-acceleration proposes that increased stress will produce faster dental development through inflammatory pathways. These models were tested on dental development scores from the New Mexico Decedent Image Database by using non-accidental manner of death (homicide, suicide, and natural) as a proxy for elevated stress exposure during life and cadaver BMI percentile as a proxy for living BMI. Sex was treated as a positive control because females typically show more rapid dental development than males. Non-accidental manner of death and male sex were both associated with slower dental development; however, manner of death was only significant for 7/32 teeth. Mean BMI percentile was highest for natural manner of death and lowest for homicide. These findings support the stress-delay model while also according with existing studies that found limited evidence for embodied effects on dental development being sufficiently large to affect estimates of age.

Introduction

The dentition is unique among bodily tissues in that enamel and dentine do not turn over during life. As a result, the teeth preserve a record of development and early life experience. The dentition responds to lived experience through tooth size, form, pathology, and developmental timing (Koh et al., 2016; McDermott et al., 2021). This paper will primarily focus on biological embodiment through dental development, assessed via crown and root maturation. Dental development is less affected than skeletal development by internal and external factors, but dental development remains correlated to socioeconomic status and childhood disease (Cardoso,

2007; Garn et al., 1965a, 1965b). I examine potential mechanisms and outcomes for the biological embodiment of chronic physiological and psychosocial stress through nutritional status, trauma, and systemic disease in dental development.

Theoretical context

Embodiment is used here to mean biological embodiment, the manifestation of life experience through individual biology. Hereafter, "embodiment" will refer to biological embodiment. This definition was initially proposed in epidemiology (Krieger, 2005) as well as adopted in critical race studies (Gravlee, 2009). This definition is directly relevant to the study of human variation in biological anthropology. An embodiment perspective connects lived experiences and biological variation. This perspective provides one avenue for addressing questions about broad societal trends (e.g. war, poverty, sanitation, globalization) on a biological level. For example, Clarkin (2019) presents armed conflict as an environment with conditions that affect development of noncombatants, such as malnutrition, stress, and disease. Effects of war on growth and development are documented throughout history, such as decreased height attainment in many countries following WWII (Clarkin, 2019).

It is worth noting that while negative embodiment is often emphasized, positive embodiment is also possible. Negative embodiment refers to the biological manifestation of negative experiences, while positive embodiment is the opposite, biological manifestation of positive experiences. Improved sanitation correlates with increased height attainment (Mulmi et al., 2016; Vyas et al., 2016). Vaccination positively correlates with height, weight, and cognitive function (Anekwe and Kumar, 2012; Bloom et al., 2012). Children who are fostered or adopted exhibit catch-up growth across multiple developmental axes relative to those who stay in institutional care (Johnson, 2002; Johnson et al., 2010).

Both negative and positive embodiment can occur through phenotypic plasticity, differing phenotypes from the same genotype under different environmental conditions. Embodiment is distinguished from plasticity in that embodiment places emphasis on the environmental change, whether it was adverse or favorable to the individual, and what the biological outcome was. Plasticity focuses on connecting genotype by environment interaction to corresponding phenotypic outcomes but is less concerned with individual experiences.

The deciduous teeth develop primarily in utero and finish developing at around three

years of age. The permanent incisors and first molar may begin developing in late gestation. With the exception of the third molar, the permanent teeth complete development by around 15 years. The third molar may finish development as late as the early twenties (Moorrees et al., 1963; Šešelj et al., 2019). The dentition forms a continuous developmental record from the fetal period until at least the upper teens. Exfoliation of the deciduous dentition during childhood means that most of the uterine record is not retained into adulthood.

The cooperative genetic interaction theory (CGI) of dental development proposes that the initiation and timing of dental development is mediated through a combination of factors. These include neural crest cell migration to the developing jaw, local molecular signaling gradients in the oral epithelium, and homeobox gene patterning in neural crest cells. Activation of homeobox genes in neural crest cells affects the responses of these cells to local signaling gradients (Mitsiadis and Smith, 2006). CGI is supported through experimental findings in crown metrics, crown morphology, tooth eruption, and developmental stages (Brook et al., 2014, 2009; Moorrees and Reed, 1964; Parner et al., 2002; Sgheiza, 2024; Stojanowski et al., 2019, 2018, 2017).

The primary signal transduction pathways involved in dental development are bone morphogenic proteins (Bmp), fibroblast growth factors (Fgf), sonic hedgehog (Shh), and wingless-related (Wnt). These four families of signaling pathways operate in multiple positive and negative feedback loops throughout development. Feedback loops initiate tooth formation, control tooth morphology, and determine the number and patterning of teeth within each quadrant (Lan et al., 2014).

A CGI framework suggests multiple potential avenues for environmental disruption of dental development. Smad4 regulates the BMP/TGF-beta > Shh > Nfic signal transduction pathway for the development of root dentin. If Smad4 signaling is disrupted, Shh does not signal expression of Nfic and root dentin does not form correctly (Huang et al., 2010). Smad4 also regulates Runx2, a transcription factor critical in the development of bones and teeth, downstream from both Fgf and Bmp (Camilleri and McDonald, 2006; Huang et al., 2010). Smad4 (and TGF-beta) expression can be affected by micro-RNAs, DNA methylation, and histone modification (Pakravan et al., 2022). Runx2 is also a potential site for DNA methylation, as the related gene, Runx3, shows effects from hypermethylation (Camilleri and McDonald, 2006).

Epigenetic modifications are a normal and essential part of cell differentiation. Histone acetylation is a critical step in dentin formation and the differentiation of odontoblasts (Tao et al., 2020). Activation of the Wnt pathway in adult dental pulp stem cells causes downstream epigenetic modifications in the cell lineage of DNA methylation and histone acetylation (Uribe-Etxebarria et al., 2020). These epigenetic modifications can have massive downstream effects. For example, changes in epigenetics and post-transcription regulation of Runx2 may explain cranial chape differences between modern humans and Neanderthals (Di Pietro et al., 2021). DNA methylation has been shown to differ significantly between individuals with hypodontia and a control (Wang et al., 2016). Histone methylation changes have also shown visible effects on development (Zheng et al., 2014).

Exposure to stress during development can have major effects on epigenetic modifications. Environmental factors tend to have a larger effect when they occur early in life or in-utero because this is when epigenetic patterning for the rest of an individual's life is being set up. While studies that directly tie epigenetic changes due to stress to differences in dental development are few, the effects of stress on epigenetic modification are documented. Exposure to violence has been shown to produce higher epigenetic ages in children, primarily through methylation (Jovanovic et al., 2017). Prenatal stress, lead exposure, frequent infections, and malnutrition have all shown the potential to cause epigenetic changes (Fatima et al., 2017; Koh et al., 2016).

These stressors and others impact development, which may include dental development. Autistic boys showed a higher frequency of linear enamel hypoplasias relative to neurotypical boys, although rate of enamel formation was not significantly different (Kurek et al., 2020). In a population with high environmental lead exposure, poor nutrition, and a high burden of childhood infectious disease, overall dental variation and hypodontia rates were higher than in a comparative modern population (Koh et al., 2016).

Dental response to external factors depends on both the type of external factor and the timing of the insult because the number of teeth developing crowns and roots (and therefore able to show a response) varies throughout the developmental period. There are critical periods during dental development that are more susceptible to external modification. This is part of why the same insult therefore will not produce the same response between people or between teeth in the same person (Brook, 2009). Chronic nutritional stress and disease have both been shown to

reduce overall crown size and increase incidence of agenesis (Koh et al., 2016). Lower socioeconomic status, which likely represents a variable combination of nutritional stress, disease burden, and psychosocial stress, was found to produce significant delays in dental development (Cardoso, 2007).

Paradoxically, some stressors can accelerate dental eruption, development, or both. Advanced dental mineralization and eruption have been associated with obesity, diabetes, and gingivitis, suggesting a potential inflammatory mechanism for dental acceleration (Garn et al., 1965b; Nicholas et al., 2018). Low SES is associated with poor nutrition, but poor nutrition is an umbrella of conditions that includes, both undernutrition, and malnutrition without caloric insufficiency, and overnutrition. In the United States, childhood obesity has shown consistent negative associations with household income (Powell, 2022; Singh et al., 2010). Both low SES and adverse childhood experiences have been correlated with earlier eruption of the permanent first molars (McDermott et al., 2021). Sumner et al. (2019) differentiate between threat exposure (e.g. violence and abuse) and deprivation (e.g. food insecurity or neglect), finding that the former stressor category is associated with accelerated aging markers of pubertal timing and telomere aging in children while the latter is not.

Research questions and hypotheses

Many of the stressors discussed above produce increased inflammation. If dental advancement can occur through an inflammatory pathway, then it is possible that there are two opposing processes at work. First, those that delay dental development through nutritional insufficiency. These would include undernutrition as well as infectious diseases that reduce nutritional availability, such as gastroenteritis. Second are those processes that accelerate dental development through inflammation. Chronic stress increases inflammation through activation of the hypothalamus-pituitary axis (Baumeister et al., 2016; Silverman and Sternberg, 2012). Obesity is also associated with higher inflammation markers in children (Chang et al., 2015). If caloric insufficiency becomes less common but malnutrition persists, we may see more mixed effects on development. A model in which increased childhood stress leads to delayed development is not the only possibility (stress-delay model A). As discussed above, negative embodiment of stress through chronic stress, childhood obesity, or other sources of inflammation may accelerate development in some instances (inflammation-acceleration model B). These

models and their associated logical statements are given in table 1.

{~?~IM: insert Table 1 here.}

Table 1. Logical statements for stress-delay and inflammation-acceleration models. "!" means "not."

Examining these models through the lens of dental development means detecting Q and Q' to imply P and P'. What is important to note, however, is that dental developmental timing is relative. That is, Q is functionally equivalent to !Q' and Q' is functionally equivalent to !Q. This means that the converse of A cannot be readily distinguished from the contrapositive of B and vice versa. What appears to be model A might in fact be the inverse of B, and model B, the inverse of A. Note that statements A and B are statements of negative embodiment and their respective inverses are statements of positive embodiment.

Biologically speaking, a relative delay may imply either the presence of stress or the absence of inflammation compared to other individuals in the sample. Relative advancement may imply the presence of inflammation or the absence of stress compared to other individuals. Separating the two possibilities then would require a means other than dental development for detecting either stress or inflammation, such as a measure of body fat. This would distinguish P from !P' when Q or !Q' is detected and P' from !P when Q' or !Q is detected, clarifying the roles of negative and positive embodiment.

Existing research supports the embodiment of childhood life experience in dental developmental timing (Sumner et al., 2019). Embodiment theory predicts an association between dental development and manner of death if manner of death is treated as a proxy for stress experienced during life. Suicide, for example, suggests a high degree of psychosocial stress. Homicide correlates with exposure to violence during life. Natural deaths in children may be the result of chronic disease. Accidental deaths should capture a group of individuals more representative of the living population. Association between dental development and manner of death is tested here using dental development scores of individuals in the New Mexico Decedent Image Database (NMDID), a database of micro CT scans taken at autopsies (Edgar et al., 2020). This will serve as a test of the stress-delay model in which negative embodiment causes delays in dental development. whether further research is warranted into distinguishing between the stress-delay and inflammation-acceleration models.

Materials and methods

Dental scores were obtained from the publicly available Subadult Virtual Anthropology Database (SVAD) (Stull and Corron, 2022). This dataset consists of AlQahtani et al. (2010) scores of dental development from NMDID micro CT scans. The NMDID began in 2011 and annually represents about 90% of the caseload of the New Mexico Office of the Medical Investigator, which serves the entire state of New Mexico excluding reservation and military deaths (Edgar et al., 2020). This database is therefore a representative sample of the medicolegal decedent population of New Mexico. Individuals in SVAD were matched to manners of death reported in NMDID via common deidentified record numbers. Reported manners of death were then coded as "homicide," "suicide," "natural," "accidental," and "undetermined" using keywords. If the listed manner of death was ambiguous, it was coded as "undetermined." Individuals with undetermined manner of death (n = 75) were removed from the sample. The final sample size for analysis was 590 individuals (425 accidental, 90 homicide, 60 natural, 15 suicide). The sample age distribution by manner of death is provided in Figure 1.

{~?~IM: insert Figure 1 here.}

Figure 1. Sample age distribution by manner of death.

Univariate logistic regression models were fit to each tooth with age on a log scale, regressing stage on age, sex, and manner of death using the *polr* function in R (R Core Team, 2019; Ripley et al., 2019). Logit was chosen over probit regression because logit is more forgiving to small category counts. Regressing stage on age rather than age on stage reduces the influence of the age distribution of the sample. This was an important consideration here due to the uneven age distribution.

Sex and manner of death were included as covariates. Sex served as a positive control. Differential dental development due to sex is consistently documented (Liversidge, 2011; Moorrees et al., 1963), so a detectable effect from sex demonstrates that the analysis is adequately sensitive to detect a true positive. Model fitting was successful for all but the maxillary second premolars and the right mandibular second premolar, for which some category counts were too low to successfully fit the model. Model fitting was performed both with the sex and manner of death covariates included for all models and using stepwise AIC with manner of death collapsed into accidental and non-accidental to examine whether the covariates contributed

to improving model error.

The purpose of logistic regression was to partition the effect of age from the effects of covariates on dental development by including age, sex, and manner of death in the same model. Regressing dental development on all three independent variables is a more robust analysis than dividing the sample into covariate categories and comparing dental development between them because the latter strategy would be heavily influenced by age structure differences between categories. Including age as one of several independent variables makes it possible to examine covariate trends across the age range of the sample.

The coefficients of categorical covariates in a regression model are interpreted as modifiers to the regression equation that are only applied when the covariate is equal to a particular value. For binary categorical covariates one covariate state (i.e. female sex) is randomly assigned as the model null condition with a value of zero and the other state is assigned a value of one. Therefore, for individuals in the covariate null state, the coefficient is multiplied by zero. When a covariate has multiple mutually exclusive categories, each category beyond the null state is treated as a separate binary variable with a value of one if the individual is in that category state and zero otherwise. Here the model null states were female sex and accidental manner of death. The covariate coefficients for sex and manner of death are then interpreted as the effect of male sex (relative to female sex) and homicide, suicide, or natural manner of death (relative to accidental manner of death) on dental development. Positive coefficient values mean that the secondary covariate state advances developmental rate relative to the null state, while negative values mean the opposite.

Cadaver BMI was calculated from cadaver weight and cadaver length as a proxy for living BMI. BMI values were converted into percentile scores for age using the CDC reference for the *sds* function from the *childsds* package (Vogel, 2022). Percentile scores were used because BMI naturally shifts throughout development. BMI percentiles were compared by MOD using a Kruskal-Wallis test with a post-hoc Dunn test and Holm adjustment for number of comparisons due to failure of the ANOVA normality assumption. Proportions of percentiles above 0.85 (overweight) and 0.95 (obese) were also compared by MOD using chi-square tests. Suicide MOD was excluded from these tests due to the small number of individuals with this MOD and their unusual age range.

Results and Discussion

As shown in Figure 2, male sex typically produced a delay effect on dental development, with the exception of the third molar. This is consistent with previous findings that males tend to reach equivalent stages at later ages than females (Moorrees et al., 1963). Differences in third molar development may be less pronounced or reversed in some instances (Liversidge, 2011; Liversidge et al., 2017). For most teeth, an accidental manner of death showed a positive effect on dental development. Homicide and natural MOD showed delay effects more frequently than acceleration effects while suicide MOD showed an acceleration effect for nearly all teeth. This is more consistent with the expectation that individuals who die of homicide or natural causes are more likely to have experienced stressors during life that slowed their dental development than with either the inflammation-acceleration model P'→Q' or the inverse of the stress-delay model !P→!Q.

{~?~IM: insert Figure 2 here.}

Figure 2. Logistic regression coefficients for sex vs. manner of death against the model null conditions that an individual is female with an accidental manner of death.

Natural MOD showed larger coefficients (more positive) for maxillary teeth than mandibular teeth. Homicide MOD showed delays in the post canine dentition and acceleration in the anterior teeth. Suicide MOD showed pronounced acceleration for all but the second and third molars (Figure 2). This suggests an age distribution effect since these are the latest developing teeth and therefore would be the best represented in the limited sample.

According to stepwise AIC results, sex was a meaningful covariate for all teeth but the incisors, while manner of death was a meaningful covariate for all second molars, the mandibular first molars, and one maxillary incisor (Table 2). Consistent with figure 2, the sign was reversed for the incisor. It is not surprising that sex effects were more consistently observed than manner of death effects given that individuals experience both positive and negative embodiment to different degrees, and both the stress-delay and inflammation-acceleration models were likely at work simultaneously.

{~?~IM: insert Table 2 here.}

Table 2. Stepwise AIC results from model fitting. Default sex is male and default MOD is accidental. Positive coefficients indicate an acceleration effect while negative coefficients indicate delay effect. "NA" indicates that stepwise AIC did not retain the variable in the model.

Mean BMI percentiles were highest for natural MOD (0.73) and lowest for homicide MOD (0.58), while accidental was intermediate at 0.69 (Table 3). A Kruskal-Wallis test for difference in means was significant (p=0.0375). A post-hoc Dunn test showed that homicide MOD was significantly different from both accidental (p=0.0328) and natural (p=0.0305) MOD. Proportion of overweight individuals was significantly different by MOD (p=0.0273), but proportion of obese individuals was not. These results support the stress-delay model P→Q for natural MOD because high relative BMI percentiles in this group are contrary to the inverse of expectations for the inflammation acceleration model: !P'→!Q'. It is also possible, however, that individuals in this group experienced less peri- and postmortem tissue loss, resulting in higher BMI values.

{~?~IM: insert Table 3 here.}

Table 3. BMI mean percentiles and proportion overweight and obese by manner of death.

Homicide MOD results are more ambiguous as relatively low BMI percentile results lend support to both the stress-delay and inflammation-acceleration models. Trend reversal in the anterior dentition relative to the posterior dentition in this group is more consistent with an anatomical effect than a timing effect since the maxillary second incisors (the most accelerated teeth by MOD) and the mandibular first molars (the most delayed) develop at similar times. It is also possible that stress-related inflammatory effects not driven by BMI are occurring in this group.

Previous skeletal and dental studies provide mixed evidence of embodiment effects on development. Spake et al. (2021) found no significant mortality bias in dental development when comparing natural and accidental manners of death to approximate an archaeological population, although they did see some non-significant trends of delayed development in the natural death group. In a companion study on long bone growth, they found bias over three years and under six months (Spake et al., 2022). Spake and Cardoso (2018) also found that children who are victims of homicide were shorter than their counterparts, especially in the United States, and Cardoso et al. (2019) found evidence of growth delays in African enslaved children in Portugal. In contrast, Stull et al. (2021) found no significant differences in dental or skeletal development between living and deceased children, or between deceased children by manner of death (accident, natural, homicide, and unknown) for ages 0–15 years.

There are a few key differences between this study and the two dental studies cited above

that also used data from NMDID. Both cited studies used different MOD combinations than what was employed here. Spake et al. (2021) further considered only natural causes of death that would have been present in an archaeological population and included data from a South African sample in these comparisons. Second, both trend analysis and significance testing in this study were performed on individual teeth but not individual stages. Both Stull et al. (2021) and Spake et al. (2021) examined trends and significance at the stage level for individual teeth. A less refined analysis in this instance may have allowed for the elucidation of broader trends by reducing sample fragmentation. Even so, it is worth noting that including MOD only significantly improved model performance for seven teeth as measured by AIC in this study and trend reversals for some teeth were noted in both homicide and natural MOD.

Different ancestry demographic compositions within manner of death categories is also a potential source of unaccounted-for variation in dental development. Effects of genetic ancestry on dental development have not been consistently detectable when model factors and extrinsic sample characteristics are controlled (Braga et al., 2005; Kiran et al., 2015; Liversidge, 2011; Thevissen et al., 2010). Apparent population specificity of dental development may be attributable to methodological factors such as age mimicry and small sample effects (Sgheiza and Liversidge, 2023a, 2023b). Regardless, these results are from a single U.S. sample and may not be fully generalizable to other regions due to extrinsic factors such as SES.

Sample size was the primary limiting factor in this analysis as this limited both the number of covariates that could be included and the number of categories that could be used. Individuals would have experienced different degrees of negative and positive embodiment both within and between categories of manner of death. A major caveat to this analysis is that manner of death frequencies were also not uniform with respect to age, but an interaction effect between age and manner of death could not modeled due to low counts. It is possible that some of the observed effect from manner of death is due to collinearity with age. This possibility was mitigated by regressing stage on age to limit distribution effects on model parameters. Nevertheless, the results for suicide in particular should be interpreted with extreme caution given the small number of individuals with this manner of death (n=15) and their relatively old age range (14–20 years inclusive).

This analysis is additionally limited by the use of cadaver BMI percentile as a proxy for obesity. BMI is a population-level measure that is not sensitive to individual body composition.

Cadaver BMI is based on cadaver weight and length, which are not equivalent to living weight and height due to differences in measurement and factors such as fluid and tissue loss during the peri- and postmortem intervals. Here, cadaver BMI percentile is employed as a *relative* measure for comparing individuals within the dataset. It is likely that there are differences in fluid and tissue loss by manner of death.

Nevertheless, these results demonstrate the potential for embodiment through effects on dental development is detectable. The findings are complicated by the fact that low SES is linked to higher all-cause mortality, including accidental deaths, as well as increased rates of obesity (Rogers et al., 2017; Singh et al., 2010). While manner of death is used here as a proxy for stress variation, it is not a proxy for SES or stress in general. Further research into effects of obesity on dental development would clarify the theoretical mechanisms at work.

Epigenetic mechanisms provide a link between embodiment of childhood experiences and effects on dental development. Embodiment of adverse early life experiences does not exclusively produce delayed development, however. In the example of manner of death in the NMDID sample, there are detectable associations between non-accidental manner of death and slower dental developmental timing. NMDID results for homicide and natural MOD are more consistent with the stress-delay model, rather than the inflammation-acceleration model, but no sample is a monolith. These results are complicated by additional variables such as SES and obesity rate. The developmental state at death of each individual within NMDID was influenced by both negative and positive embodiment including stress-delay and inflammation-acceleration processes. Further examination of intraindividual variation and relevant covariates would provide a more nuanced picture of embodiment in these individuals.

Conclusions

While embodiment as measured by proxy of MOD showed consistent developmental trends, these trends only reached significance in terms of age estimation model performance for seven out of 32 teeth. Further investigation is warranted, but these findings are consistent with previous studies in indicating that prior concern for drastic difference in age estimation performance due to embodied effects may be outsized (Spake et al., 2021; Stull et al., 2021). An additional avenue for addressing the potential for age estimation bias due to embodiment in dental developmental timing is using larger reference samples. Increased sample sizes will capture a larger degree of

population variation in dental development, meaning that a decedent's developmental profile is more likely to be represented in the dataset.

Tooth size and morphology are both promising avenues of investigation for embodiment. The stress-delay model predicts larger, less variable teeth in lower-stress individuals, while the inflammation acceleration model does not. This would differentiate the converse of B (Q'→P'): acceleration → inflammation, from the contrapositive of A (!Q→!P): no delay → no stress, a distinction that was not possible in this study.

Biological embodiment has the potential to provide new insight into current and past populations. One mechanism for this is the differences in environmental buffering between the dentition and the skeleton. Existing research shows that environmental effects on dental development are typically relatively less than those on the skeleton and skeletal development tends to be more variable overall (Cardoso, 2007; Rautman and Edgar, 2020). As a result, differences in dental and skeletal age have the potential to be used as a stress indicator even when chronological age is unknown. Larger relative differences could be used to infer indicate increased stress during life at the individual level. Such an age-gap indicator could also be applied at the population-level using correlations between dental and skeletal age.by studying dental and skeletal age differences at the population level to establish the range of normal variation between these systems throughout the developmental period.

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Table 1. Logical statements for stress-delay and inflammation-acceleration models. "!" means "not."

Statement A	$P = stress \rightarrow Q = delay$
Statement B	$P' = inflammation \rightarrow Q' = acceleration$
Inverse A	$!P = no\ stress \rightarrow !Q = no\ delay$
Inverse B	$!P' = no\ inflammation \rightarrow !Q' = no\ acceleration$
Converse A	$Q = delay \rightarrow P = stress$

Converse B	$Q' = acceleration \rightarrow P' = inflammation$
Contrapositive A	$!\,Q = no\ delay \rightarrow !\,P = no\ stress$
Contrapositive B	$!\,Q' = no\ acceleration \rightarrow !\,P' = no\ inflammation$

Table 2. Stepwise AIC results from model fitting. Default sex is male and default MOD is accidental. Positive coefficients indicate an acceleration effect while negative coefficients indicate delay effect. “NA” indicates that stepwise AIC did not retain the variable in the model.

	Maxilla			Mandible		
Tooth	**Age**	**Sex**	**MOD**	**Age**	**Sex**	**MOD**
Left I1	10.1234	NA	NA	10.8727	NA	NA
Right I1	10.1225	NA	NA	10.8831	NA	NA
Left I2	10.9939	NA	-0.6049	12.1516	NA	NA
Right I2	11.2688	NA	NA	12.0878	NA	NA
Left C	13.1308	-0.8959	NA	12.7421	-1.2309	NA
Right C	12.7526	-1.0009	NA	12.2971	-1.0595	NA
Left PM1	15.5626	-0.7732	NA	17.0367	-1.0704	NA
Right PM1	15.9978	-0.8462	NA	17.2834	-1.0478	NA
Left PM2	-	-	-	17.6417	-1.2407	NA
Left M1	11.5200	-0.5553	NA	12.2353	-0.7482	0.7892
Right M1	11.5342	-0.6214	NA	12.1063	-0.6798	0.6845
Left M2	16.7398	-0.6891	0.4654	16.2768	-0.7343	0.4458
Right M2	16.5987	-0.7564	0.4823	16.5120	-0.7201	0.4423
Left M3	13.8053	0.7675	NA	14.9935	0.6989	NA
Right M3	14.7018	0.9612	NA	15.1861	0.6657	NA

Table 3. BMI mean percentiles and proportion overweight and obese by manner of death.

MOD	Mean BMI percentile	Prop > 0.85 (overweight)	Prop > 0.95 (obese)
Accidental	0.6907	0.4543	0.2774
Homicide	0.5788	0.3521	0.2535
Natural	0.7350	0.5714	0.3571