Environmental Conditions as Epigenetic Modulator of Craniofacial Growth and Development Phenotypes a Mini Review

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Abstract

Environmental variation is translated into phenotypic variation based on how the same genotype can produce a variety of phenotypes across a range of environmental circumstances. Traits showing greater phenotypic variation are either under less direct genetic control and/or mature less rapidly. As expected, the vertical aspects of mandibular growth, which are the least mature in the craniofacial complex, showed the most pronounced effects, developing three types of phenotypes, where normodivergent and hypodivergent have a good facial, but hyperdivergents are the real problem, because they decrease their growth, they are increasing the incidence and prevalence, and with them, are increasing bad functional and esthetics on the population. The environmental conditions that are associated to hyperdivergent phenotype are masticatory loading during early facial growth and airway obstructive problems.

Introduction

It seems reasonable to expect that complex, highly intrincated objects will function less if they are modified, because even small alterations are likely to have potential deleterious consequences. If we compare primates in general with human linage in particular, heads appear to be no less morphologically conservative than the rest of the body, and possible even more evolvable, despite its complexity [1]. Comparison of genetic and craniofacial variation among human population suggest that most changes in craniofacial from among recent humans occurred from random, unselected genetic changes rather than from natural selection [2, 3]. Has been seen during the process of growth and development, that regulation of local growth (of different craniofacial modules) occurs through interactions between genes (that cause skeletogenic cells to synthesize, resorb, or otherwise modify skeletal tissue), and stimuli from other genes or cells. Such interactions between cells and their environment (which includes other cells) are generally categorized as epigenetic interactions, of which are several types that regulate most bone growth. Then, we have secondary epigenetic interactions between bone and neighboring tissues. Such interactions preserve functioning during complex growth, even when the growth involves many different tissues that grow all at different rates under different mechanisms

of control [4]. These interactions also permit morphological variations that range from minor differences between members of the same species to novel phenotypes [2,3].

Craniofacial Phenotypes

Most craniofacial, dentoalveolar, and occlusal traits show a quantitative, often normal, distributions of phenotypes. Traits showing such distributions are polygenetic, due to the actions and interactions of multiple genes [5]. It follows that variation in such traits must be due to genetic, epigenetic, and environmental influences. The relative contribution of genes to phenotypic expression varies depending on the environments in which they are expressed. The way in which environmental variation is translated into phenotypic variation is based on the norm of reaction [6], which states that the same genotype can produce a variety of phenotypes across a range of environmental circumstances. Traits showing greater phenotypic variation are either under less direct genetic control and/or mature less rapidly than traits showing less phenotypic variation. For example, modern day Finns exhibit substantially larger gonial and mandibular plane angles, with a 6% decrease in mandible length, despite overall skull size increases, compared to Finnish

samples from the 15th and 16th centuries [7]. Since the time span was insufficient for genetic changes to have occurred, the same genotypes must have been adapting to different environmental factors. As expected, the vertical aspects of mandibular growth, which are the least mature in the craniofacial complex, showed the most pronounced effects [8].

This is how we stablished different phenotypes for traits that are genetical controlled in craniofacial structures, in which vertical aspect of mandible is the latest trait in mature. That suppose then three types of phenotypes, where normodivergent and hypodivergent are forward rotators, in other words with normal facial growth, and both phenotypes have a harmonic development [9,10]. Hyperdivergents are the real problem, because we have seen that over time, they are increasing the incidence and prevalence, and with them, are increasing bad functional and esthetics consequences, with significant decreases of the posterior aspect of manbible [8,11,12].

Hyperdivergent Etiology

The etiology of hyperdivergent phenotype appears to be environmental, due to postural adjustments related with compromised airways and weak masticatory musculature. Changes in diet and food processing technology post industrial revolution, have contribute to some proportion of variations in facial size and shape [2]. Human diets since the Middle Paleolithic have changed substantially in content [13-15] and in how they are processed through cooking, soaking, leaching, and grinding [16-18]. Food processing improves digestibility, but also makes food softer and smaller in particle size, requiring less occlusal force per chew and fewer chewing cycles per unit of food [1,19, 20]. In turn, softer and more processed foods are widely hypothesized to lead to less facial growth, especially in the lower face and the alveolar crests, because of the potential effects of force-generated strain [21,22]. While one might expect nutritional improvements since the Middle Paleolithic to contribute to increases in overall cranial size [23], the evidence points to a trend towards smaller facial size, with the most dramatic decreases occurring after the Neolithic. Comparisons of Nubian populations prior to and after the introduction of agriculture show significant reductions in many mid- and lower facial dimensions including infraorbital height (7.8%), masseter origin length (26.3%), mandibular corpus length (22%), and mandibular symphysis thickness (15.3%)-despite concurrent increases in brain size [24,25].

Environmental conditions that can modulate craniofacial phenotypes

No human studies have examined the effects of masticatory loading during early facial growth, when such effects are likely to be greatest, and none have quantified strains or site-specific growth rates. Most experimental data on facial growth responses to masticatory loading come from studies on non-human anthropoids and other mammalian models. One primate study [22,26,27] compared 19 adult squirrel monkeys (Saimiri sciureus) raised on soft food diets with 24 controls raised on hard food diets; another study [22, 23] compared 16 adult baboons (Papio cynocephalus) who were raised on a hard diet for two years with 24 baboons raised on a soft diet. In both studies, animals who chewed harder food had significantly wider and taller faces, thicker mandibular corpora, and taller palates. However, primates raised on softer food often had serious malocclusions from narrowed maxillary arches, rotated, and displaced teeth, crowded premolars, and palatal arching, suggesting abnormal growth patterns. A related study on macaques obtained similar results, but also showed that Haversian remodeling rates were higher in monkeys fed hard food [28]. Experiments on non-primates reveal a similar picture [1,2,29].

On the other hand, airway problems have been associated with hyperdivergent phenotypes. On this pathway, there is some statistical support for an association between craniofacial disharmony and pediatric sleep-disordered breathing, however, in children with obstructive sleep apnea and primary snoring, compared with the controls, could be regarded as having marginal clinical significance (e.g., an increased ANB angle of less than 2º) [30]. Evidence for a direct causal relationship between craniofacial structure and pediatric sleep-disordered breathing is unsupported by Katyal´s (2013) meta-analysis. There is strong support for reduced upper airway sagittal width in children with obstructive sleep apnea (representing adenoid hypertrophy) [3,31]. Besides these results, there are some promising studies that have statistical results not only on the sagittal plane, but also on the vertical plane, that is what we expose in this new way of understanding craniofacial growth, were vertical plane affects directly sagittal plane. The investigation group of the author have preliminar results were the saggital of the upper airway is associated to hyperdivergent pediatric population.

Conclusion

Environmental conditions such as obstructive airway and weak muscles can modulate craniofacial phenotypes. When these variables are present, the face turn to limit it´s growth on the vertical posterior aspect of mandible due to be the latest craniofacial trait on mature and generate an hyperdivergent phenotype.

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