THE MATURE STOMATOGNATHIC SYSTEM IS A COMPLEX ADAPTIVE SYSTEM

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ABSTRACT

During development, the teeth, jaws and facial structures emerge in a process that has the characteristics of a complex adaptive system (CAS). The outcome is the mature stomatognathic system which is an interactive network of teeth, their occlusion and supporting mechanisms, the upper and lower jaws, the temporomandibular (jaw) joints, the muscles, the blood and nerve supplies, and the salivary glands. The aim of this paper is to explore whether this mature network also demonstrates the characteristics of a CAS. This network is a carefully balanced mechanism with functions required throughout the lifespan of the individual. It is exposed to a series of mechanical and chemical environmental challenges. Tooth wear is an example of the impact of such challenges, resulting in loss of surface enamel and dentine. The response is spread through the network: reparative secondary dentine occurs on the inner pulpal walls of the tooth; secondary cementum is formed and bone remodelling occurs in the suspensory mechanism; and the masticatory pattern of dental occlusion adapt under neuromuscular control. These adaptive changes arise from interactions at molecular and cellular levels. They are evidence of ongoing emergence and robustness in the network as it functions. Considering these changes to be positive responses that enable both maintenance of function and adaptation to age changes in the component parts of the network, represents a paradigm shift. It has major implications for oral health teaching and clinical practice. Attempting to restore teeth to the size and detailed cuspal shape, they showed when they first erupted is not only unnecessary for late adult function but can also place excessive loads on other subnetworks such as on the jaw joints and muscles. Thus, appreciating that both the developmental and mature stomatognathic networks are CAS establishes their value as exemplars for biological systems in general and has important implications for enhancing health care.

Keywords: caries, craniofacial, dental, dentine, enamel, erosion, oral health, phenomics, tooth wear

1 INTRODUCTION

Prenatal and postnatal growth and development of all craniofacial structures represent a complex adaptive system (CAS) where genetic, epigenetic and environmental influences occur on tissues at different levels with varying degrees of intensity and duration [1]. It is an interactive, carefully balanced network of components that remain dynamic throughout life.

Prenatal growth and development has a very strong genetic component that is moderated by epigenetic factors via maternal environmental exposure [1]. These interactions occur at a cellular and molecular level, however, from the commencement of postnatal growth and development and throughout life, there are continual physical environmental challenges that mechanically load the system at a ‘macro level’ influencing the craniofacial structures. The teeth, both individually and collectively as occluding opposing arches act as a ‘portal’ through which physical forces act on the network of underlying tissues. The intensity and the duration of the mechanical load acting via the occlusion is responsible for adaptive changes that can be further explained at cellular and molecular levels within the tissues. Such adaptations are uniquely evolved physiological mechanisms neces-

This paper is part of the Proceedings of the New Forest Conference on Complex Systems 2016 (Complex Systems 2016)
www.witconferences.com

ISSN: 1755-7437 (paper format), ISSN: 1755-7445 (online), http://www.witpress.com/journals
DOI: 10.2495/DNE-V11-N4-670-675
sary for tolerating environmental changes, and that they are necessary for survival. They are an indication to the robustness of the network. They are also a measure of physiological fitness of the individual, the population and indeed the species.

Coupled with these influences, there are also other dietary effects where food of low pH can potentially demineralise tooth surfaces through chemical action. This is naturally prevented by the symbiotic relationship developed between the human and the oral micro-biome [2].

The complexity model effectively explains craniofacial phenotypic variation, observed within and between populations, as always being in a state of flux, reflecting the intensity and duration of environmental stress. The model also emphasises that anatomical structures such as muscles and in particular bone, are physiologically ‘plastic’ and can change in accordance with the directional physical forces. It is when the individual cannot adapt, or is too slow to adapt in response to environmental stress that pathological inflammatory responses occur.

2 AIMS
The aims of this paper are to describe how the mature human stomatognathic system represents a CAS, and then to consider how this concept has major implications for research and teaching in oral health and clinical dental practice.

3 THE OVERALL NETWORK AND ITS INTERACTIONS WITHIN THE STOMATOGNATHIC SYSTEM
Figure 1 summarises the interactions that occur between the various components of the stomatognathic system, which is a part of the overall craniofacial complex. Moss’ functional matrix hypothesis states that function determines the morphology of the craniofacial complex as an adaptive response to mechanotransduction [3]. For example, the influence of air passage through the nasal cavity when breathing can have a direct effect on the plasticity of bone and associated soft tissues to ensure the development of patent airways [4, 5]. In this paper, we discuss various subnetworks in the stomatognathic system involving multiple interactions between environmental factors and teeth that trigger various forms of adaptive responses (Fig. 1). The system is best described in terms of environmental factors, such as tooth wear from abrasive diet and acids as well as dental caries (tooth decay) from

Figure 1: The mature stomatognathic system as a complex adaptive system in which reciprocal, recurrent interactions with environmental factors determine the outcome for health and disease.
dietary sugars and patient factors (i.e. poor oral hygiene and lack of protective biofilms), that interact within subnetworks to produce adaptations upon the already established stomatognathic components. The whole system is dynamic where the various inter-related components adapt in response to the environmental factors. Thus, within the overall network, one subnetwork is that between the biofilm, saliva and tooth surfaces (Fig. 1).

3.1 Environmental influence on the neuromuscular network

3.1.1 Mechanical loading and wear
Once the teeth emerge and an occlusion is established, the diet becomes one of the most predominant postnatal environmental factors in the CAS. The diet helps to establish loading between the occlusal surfaces of opposing (i.e. upper and lower) teeth, establishing the masticatory cycle. The physical forces acting on the teeth stimulate and shape both immediate oral components such as the underlying periodontal structures, as well as other craniofacial structures distant to and not directly associated with the oral cavity [6]. The coarseness of the diet and the intensity of masticatory activity are responsible for both the occlusal and interproximal wear of teeth.

3.1.2 Chemical acid effects
Chemical acid effects on teeth are referred to as dental erosion (Figs 1 and 2). It can be caused by either strong intrinsic acids (e.g. gastric acids from vomiting and regurgitation, commonly known as “gastric reflux”) or extrinsic acids (e.g. soft drinks, citrus fruits and alcoholic beverages) at around pH 3.0 or less [7]. Dental erosion results in immediate surface loss and softening after acid contact [8, 9], and it is a growing concern because of increasing prevalence in recent decades [10]. Repeated exposure to such agents results in the clinically apparent loss of tooth structure. Severe wear that has extended well into dentine has the potential to allow bacterial fluids and toxins from oral environment to communicate with the pulp tissue through open dentinal tubules.

3.1.3 Dietary sugars, oral hygiene and dental caries
Dental caries (decay) is caused by acidic demineralisation of teeth but at a higher acidic environment (around pH 4.0 to 5.0) compared with dental erosion (at around pH 3.0 or less). It is caused by the

![Figure 2: Tooth wear caused by a combination of both tooth grinding and dental erosion (wine tasting). Tooth grinding produces Facetted surfaces (black arrows) from contact during lateral movement of the lower jaw and dental erosion causes loss of surface features (such as surface grooves) and scooping (white arrows). [Courtesy of Dr D Hunt and Dr J McIntyre].](image-url)
presence of fermented sugars (carbohydrates) that are fermented by oral bacterial to produce mild lactic acid. In initial stages, the acid causes sub-surface demineralisation as redeposition of dissolved crystals occurs within the surface layers. As the process continues, this is overcome and cavitation occurs.

Both extensive forms of erosive wear and dental caries can result in exposure of the pulp chamber, allowing free passage of bacteria and their products (toxins) to elicit cellular response (inflammation) in the pulpal tissues. Both abrasive and erosive wear can result in loss of vertical dimension (i.e. bite overclosure), whereas interproximal wear, combined with mesial tooth migration [11], is responsible for a reduction in dental arch length and width.

3.2 Adaptive response to environmental influences

The masticatory cycle can be described as a rhythmic muscular action that is centrally controlled via proprioceptive feedback loops originating from craniofacial structures. Over time, the functional forces acting on teeth result in tooth wear, the degree of which is determined by the consistency of the diet. The changing tooth morphology due to wear does not reduce masticatory efficiency due to differential wear between enamel and dentine [12, 13], and the nature of the masticatory cycle changes in tandem with the wear. The periodontal ligament, the muscle fibres and the temporomandibular joints provide proprioceptive information and are a part of a feedback loop that changes the masticatory cycle via the central ‘masticatory /grinding centre’ in the central nervous system. As the cusps of the teeth are reduced by wear, the masticatory pattern becomes broader. Collectively, the neuromuscular system is designed to perpetually change proportionally to masticatory load, and to perpetually change tooth anatomy.

The adaptive response to mechanical (abrasive) wear includes a cellular response by pulpal cells (i.e. odontonblasts) to lay reparative dentine (i.e. a calcified bridge at the dentine boundary around the pulp chamber) and isolate the pulp from the oral environment. In relation to the loss of vertical dimension of the face (from reduced tooth crown height from severe wear), the occlusal forces acting on the periodontal ligament stimulate cementoblasts on the root surface of the tooth to produce cementum. This causes the teeth to continually erupt to compensate for the occlusal wear. This is another example of how the physical forces, on the one hand, can produce wear and, on the other hand, compensate for tooth wear, thereby allowing the teeth to remain functional.

The degree of masticatory muscle development is also a cellular response to the diet. Masticatory muscle hypertrophy is not uncommon in populations living in environments where vigorous mastication is required to process fibrous, hard food. In tandem with this, vigorous masticatory forces acting on teeth also stimulates alveolar bone growth, aligns trabecular patterns within the bone and therefore is responsible for bi-maxillary protrusion observed in pre-contemporary hunter-gatherer populations. In summary, although the genetic makeup of the individual underlies the direction of general development, it can be argued that the environmental forces acting throughout life alters the genetic phenotype accordingly. Overall, this to some degree, not only explains craniofacial differences observed within populations but explains craniofacial differences between populations.

In a healthy stomatognathic system, a good balance is maintained in this dynamic subnetwork between risk factors (e.g. poor diet and poor oral hygiene for dental caries, and gastric and/or strong dietary acids for dental erosion) and host factors (e.g. protection from saliva and oral biofilm made of commensural bacteria) (Fig. 1). While biofilm is a physical barrier against acids, it also acts as a reservoir of calcium, phosphate and fluoride ions that are necessary for remineralising (repairing) demineralised tooth structure. At the tooth enamel surface, there is a constant demineralisation/
remineralisation interaction, with the healthy balance favouring remineralisation. Chewing stimulates salivary flow and an increase in its buffering capacity.

4 DISCUSSION

The application of the concept of the CAS in craniofacial growth and development highlights the dynamic nature of the craniofacial system throughout life. Static concepts of the past, such as Bonwill’s Triangle, the Curve of Spee, Monson’s Curves etc., are teleological concepts that do not cater for dynamic change and variability [14]. The implications for various types of dental treatment need to be explored. For example:

1. Restoring teeth to their original newly erupted anatomy in a generally worn dentition may reduce functionality, as steep cuspal inclines could lock the bite. Such iatrogenic change may be significant and akin to sudden (unnatural) environmental change where the body is unable to adapt fast enough, thereby resulting in clinical symptoms such as tenderness to biting (i.e. occlusal trauma) or even temporomandibular joint pain.

2. The use of implants in a dynamic system also needs to be explored. Implants by their nature are osseo-integrated without a periodontal ligament. However, the periodontium with its cellular, fibrous and fluid contents allows adaptation to high masticatory forces. Lack of periodontal movement and tooth migration of implants may affect the adaptive responses in the craniofacial system.

3. The physical forces on teeth also have orthodontic implications in association with relapse of the dentition after orthodontic treatment. Understanding how the force vectors change with tooth repositioning could give some insight towards avoiding failures.

4. There is a dynamic subnetwork within the overall system influencing the integrity of the tooth surface. The balances within this determine whether ongoing demineralisation will lead to caries or whether remineralisation and lesion healing will predominate. This subnetwork is incorporated in Fig. 1.

5 CONCLUSIONS

Application of the concepts of CASs to craniofacial growth and development has many positive outcomes, including an appreciation of the overview of the interactions that occur within subnetworks in the mature stomatognathic system. This approach enables appreciation of the nature of the patterns in the functional dentition, with teeth acting as a ‘portal’ through which adaptive changes occur. Such knowledge has implications in dental education and clinical practice in relation to restoration of functionality of teeth. Furthermore, this approach opens new directions for future research in dental phenomics that has the potential to correlate large-scale genomic and phenotypic data and to improve health outcomes [15, 16].

REFERENCES


