

Other journals in brief

A selection of abstracts of clinically relevant papers from other journals.

The abstracts on this page have been chosen and edited by John R. Radford.

Arginine – ‘a genuine prebiotic’

Changes in the oral ecosystem induced by the use of 8% arginine toothpaste
Koopman JE, Hoogenkamp MA *et al.* *Arch Oral Biol* 2016; **73**:79–87

The Colgate toothpastes containing 8% arginine ‘lead(s) to a shift in the salivary microbiome composition towards a healthy ecology’.

Prebiotics foster human health by working with specific microbes. But can arginine be metabolised by oral bacteria such that the pH of the oral cavity is increased? Colgate Sensitive Pro-Relief Toothpaste is one of a range of toothpastes (Extra Sensitive and Repair and Prevent, to name just two) that contain 8% arginine. This group of Colgate toothpastes is not to be confused with Colgate Maximum Cavity Protection with Sugar Acid Neutraliser which contains 1.5% arginine; clinical trials on the efficacy of this latter toothpaste have been the subject of controversy (see The tribulations of toothpaste: Unethical arginine dentifrice research. *Br Dent J* 2015; **219**: 567–569). In this pilot study published in *Arch Oral Biol*, nine subjects used a toothpaste containing 8% arginine for eight weeks. Saliva was collected to determine arginolytic and sucrose metabolic activities. Although the investigators did not recruit a control group and the number of subjects were small, the following observations were made: 1) the arginolytic potential of saliva increased, whereas the sucrose metabolism in saliva decreased; 2) this was reversed during the wash-out period; 3) there was no change in the plaque microbiome, in contrast to the saliva microbiome, where there was an increase in the composition of the genus *Veillonella*.

DOI: 10.1038/sj.bdj.2016.946

Inferior dental block

An unusual ocular complication following dental anaesthesia: case report
Kempster C, Ghabriel M *et al.* *Aust Dent J* 2016; **61**: 374–380

A sympathetic emotional response or an idiosyncratic reaction, but possibly the spread of anaesthetic agent through anastomoses among terminal branches of the external carotid artery.

The discussion, which comprises some four pages, gives a comprehensive overview of complications associated with the administration of local anaesthetics, particularly after giving an inferior alveolar nerve block. It is full of horror stories; for example, the ‘...unusual onset of symptoms 24 hours after the delivery of artic(i)ane...’ that only subsided after 8 weeks, and the much vaunted but very alarming complication of transient visual loss following intra-arterial injection leading to spasm of the central retinal artery. This paper reports a case of an unusual contralateral involuntary closure and opening of the eyelids until complete closure, following administration of an inferior alveolar nerve block.

DOI: 10.1038/sj.bdj.2016.948

Football injuries

Facial fractures in football: incidence, site, and mechanism of injury
Kim SY, Chan CL *et al.* *Br J Oral Maxillofac Surg* 2016; **54**: 936–940

‘...the headgear is for the protection of the opposing player...’ and not necessarily for the player wearing it.

Although the authors do concede that any legal requirement to wear soft headgear may change fundamentally the playing of association soccer. This paper reports a surgical audit of maxilla-facial injuries incurred when players participated in several Australian ‘football codes’. The authors state 1) rugby league had the highest incidence of facial fractures, followed by rugby union, ‘Australian rules’, and then soccer; 2) clash of heads was the most common cause of fractures, although injuries were sustained from impacts with shoulders, and forearms and fists particularly in rugby league; and 3) two thirds of such injuries required surgical intervention. Some of the observations reported in this paper reflect merely those who participate in these sports.

DOI: 10.1038/sj.bdj.2016.947

Dysbiosis

Gut microbiota dysbiosis motor deficits and neuroinflammation in a model of Parkinson’s Disease

Sampson TR, Debelius JW *et al.* *Cell* 2016; **167**: 1469–1480

‘Signals from gut microbes are required for the neuroinflammatory responses...in Parkinson’s disease.’

A recent article (*Br Dent J* 2016; **221**: 657–666) focused on dysbiosis; this is when the ‘normal microbiome population structure is disturbed, often through external burdens such as disease states or medications’. Dysbiosis in the oral cavity could be fundamental in the aetiology of both dental caries and periodontal diseases. This paper published in *Cell* comprises a succinct graphical Abstract and Highlights, an overview of the elegant experiments that explore Braak’s hypothesis and details of the methods used in these experiments. As background, it is hypothesised that Parkinson’s disease is because of dysbiosis in the gut; ‘Braak’s hypothesis posits that aberrant aSyn (alpha-synuclein) accumulation initiates in the gut and propagates via nerve to the brain in a prion-like fashion’. One of the experiments published in *Cell* describes germ-free mice that had been genetically modified to overproduce alpha-synuclein maintain motor skills, but similar mice with a gut microbiome showed brain damage. In another experiment, mice developed enhanced motor dysfunction when samples of gut bacteria from humans with Parkinson’s disease were transplanted into germ-free mice that overexpressed alpha-synuclein, but other mice who were inoculated with gut bacteria from humans without Parkinson’s disease did not develop disease.

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