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.

OTHER SYSTEMIC DISEASES

Periodontitis and systemic diseases: consensus report of the Joint EFP/AAP Workshop on Periodontitis and Systemic Diseases

Linden GJ, Herzberg MC, working group 4 of the joint EFP/AAP workshop. *J Clin Periodontol* 2013; **40** (Suppl. 14): S20–S23. doi: 10.1111/jcpe.12091

Causation or common risk factors?

The four criteria of Koch's postulates for causation were used to claim that *Streptococcus mutans* was a causal agent of dental caries. In this rigorous consensus statement, the nine Bradford Hill criteria were advocated in order to establish a 'causal relationship between an incidence and a consequence'. This working group concluded it was not possible to infer causal relationships between periodontitis and, among other diseases, COPD, chronic kidney disease, rheumatoid arthritis, cognitive impairment and cancer (other working groups were charged to explore links between periodontitis and adverse pregnancy outcomes, cardiovascular diseases and diabetes). It was reported, however, that 'organisms originating in the oral microbiome can cause lung infections.'

DOI: 10.1038/sj.bdj.2013.605

ADVERSE PREGNANCY OUTCOMES

Periodontitis and adverse pregnancy outcomes

Sanz M, Kornman K, working group 3 of the joint EFP/AAP workshop. *J Clin Periodontol* 2013; **40** (Suppl. 14): S164–S169. doi: 10.1111/jcpe.12083

Only 'modest associations' and no causation, but excuses for no clear message and mechanisms that could underpin any such relationship.

Apart from only 'modest associations' between periodontitis in mothers and pre-eclampsia, low birth weight babies and pre-term birth babies, it was reported that such relationships seem to vary across different populations. Many of the deliberations in this consensus statement did focus on why such relationships have been difficult to elucidate and mechanisms that could support any contended association. For example, there is the perennial problem of classifying periodontal diseases and that none of the purported disease entities 'adequately reflect the infectious/inflammatory burden'. It was conceded, that although adverse pregnancy outcomes could be as a consequence of haematogenous spread, such 'primarily originate from ascending infections from the vagina or cervix'.

DOI: 10.1038/sj.bdj.2013.607

ATHEROSCLEROTIC CARDIOVASCULAR DISEASE (ACVD)

Periodontitis and atherosclerotic cardiovascular disease

Tonetti MS, Van Dyke TE, working group 1 of the joint EFP/AAP workshop. *J Clin Periodontol* 2013; **40** (Suppl. 14): S24–S29. doi: 10.1111/jcpe.12089

Robust intervention studies are required to examine the impact of periodontal treatment on prevention of ACVD.

'...excess risk (of ACVD) could be due to unknown confounders.' This statement is based on recent findings from the ENCODE project. At the heart of this project is the premise that 'there are common genetically determined pathways underpinning various complex inflammatory diseases.' It could be these unknown confounders, and not periodontal diseases, have an aetiological role in ACVD. Notwithstanding this, the consensus from this working group was that longitudinal studies have found that those with periodontitis have significant excess risk of incident cardiovascular events. On the flip side, a recent study is cited that reported, somewhat worryingly, that periodontal treatment 'elicits a transient increase of systemic inflammatory/pro-thrombotic mediators...'

DOI: 10.1038/sj.bdj.2013.606

DIABETES

Diabetes and periodontal diseases

Chapple ILC, Genco R, working group 2 of the joint EFP/AAP workshop. *J Clin Periodontol* 2013; **40** (Suppl. 14): S106–S112. doi: 10.1111/jcpe.12077

Studies have shown that 'Periodontal treatment reduces plasma HbA1C (a measure of glycaemic control)... by levels equivalent to adding a second drug to a pharmacological regime.'

In this rigorous statement, it was affirmed that periodontal diseases and diabetes exhibit a bidirectional relationship: periodontitis adversely affects glycaemic control and concomitant diabetic complications in those with diabetes, and in turn there is 'emerging evidence' that there is an increase of incident diabetes in those with severe periodontitis. Although the consensus was that the 'ultimate goal' was to establish causality between diabetes and periodontal diseases, using the Bradford Hill Criteria, this should not divert present endeavours to confirm unequivocally that 'periodontitis is independently associated with adverse diabetes outcomes' and the 'impact of periodontal interventions on adverse diabetes outcomes'. No-nonsense dental/interface guidelines for physicians and dentists caring for patients with diabetes and for patients with diabetes, are proposed.

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