obesity (BMI ≥25 kg/m²) contributed to 5·5% of total deaths, whereas underweight contributed to 0·7%, in their UK-based study population. The estimate for excess bodyweight with mortality is similar to that of the 2015 Global Burden of Disease Study,[3] which estimated that, globally, overweight and obesity contributed to approximately 7·1% (95% uncertainty interval 4·9–9·6) of total deaths. PAF estimates are expected to vary across study populations according to the time period of data collection, socioeconomic development, and the ranking of common causes of mortality. Estimates of PAF for excess bodyweight with mortality will also continue to be a moving target as the population distribution of BMI shifts. Regardless, this metric provides a useful tool for health-care providers and stakeholders to appreciate the importance of overweight and obesity as an important driver of excess mortality.

This study adds to the overwhelming evidence about the public health importance of the obesity epidemic to overall and cause-specific mortality. It also has important clinical and public health implications for obesity prevention, especially for the prevention of further increase in bodyweight and waist size among moderately overweight individuals. Although the debate might persist as to the precise point at which the association between continuous BMI with excess mortality becomes statistically significant, it is important to note that most people will gain weight throughout midlife, which is associated with increased subsequent risk of chronic diseases and mortality.[5] Many individuals with a BMI in the range of overweight (25·0–29·9 kg/m²) are already on a trajectory of gaining more weight that will transition them into the BMI range of obesity (≥30·0 kg/m²). Therefore, although the excess mortality associated with overweight is relatively small, it is important for overweight individuals to prevent further weight gain. Even among older populations, a plateau or decline in bodyweight often masks a trajectory of fat mass gain, offset by losses in lean body mass (ie, decreases in muscle tissue and bone density).[6] Therefore, it is important for older individuals to prevent an increase in waist size, a marker of abdominal obesity, while minimising loss of muscle mass. Additional studies might shed more light on the role of body fat distributions and different fat depots in chronic disease morbidity and mortality. Meanwhile, current efforts need to be intensified to identify more effective and impactful strategies for prevention of weight gain and obesity-related comorbidities.

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Oral health: a neglected aspect of diabetes care

Periodontitis and diabetes are well known to have a bidirectional association.[1] Patients with uncontrolled diabetes are prone to periodontitis, probably due to their hyper-responsiveness to the oral dysbiosis and weakened healing processes, collectively causing periodontal breakdown.[2] Meanwhile, periodontitis associates with poor glycaemic control, leading to microvascular complications.[2] Furthermore, periodontitis can also contribute to macrovascular complications through systemic inflammation and its associated dyslipidaemia;

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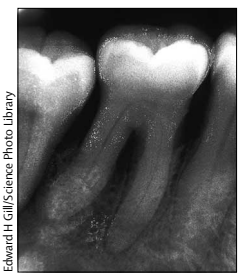
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notably, periodontitis is a strong predictor of cardiorenal mortality in patients with diabetes.[3]

In *The Lancet Diabetes & Endocrinology*, Francesco D'Aiuto and colleagues report the effects of periodontal treatment on glycaemic control of patients with type 2 diabetes in a carefully designed and performed randomised controlled trial.[4] All patients had moderate-to-severe periodontitis with signs of active disease; after initial extraction of unsalvageable teeth, patients were randomly assigned to receive intensive (IPT) or control (CPT) periodontal treatment. In addition to the exemplary periodontal outcomes achieved with the IPT, HbA_{1c} (the primary outcome) and concentrations of glucose, C-reactive protein, and creatinine were significantly reduced compared with CPT at 12 months. Notably, in the primary adjusted analysis, HbA_{1c} was reduced by 0·6 percentage points (95% CI 0·3 to 0·9; p<0·0001) with IPT compared with CPT, a decrease roughly equal to that of an additional glucose-lowering drug. Additionally, United Kingdom Prospective Diabetes Study 10 year cardiovascular disease risk and quality of life were both improved significantly in the IPT group compared with the CPT group. The overall findings suggest improved metabolic control and inflammation status, as well as improved endothelial and kidney function, following the intensive intervention.

Periodontitis is associated with HbA_{1c} levels not only in people with diabetes, but also in people without,[5] suggesting that the presence of periodontal disease might increase the risk of incident type 2 diabetes. In a 2017 study,[6] men with moderate-to-severe periodontitis had a hazard ratio of 1·69 (95% CI 1·06–2·69) for incident diabetes compared with men with no or mild periodontitis. An open question for future research is whether periodontal therapy could delay the onset of type 2 diabetes.

Regarding the systemic effects of oral health, the role of another common infection has gained less attention than periodontitis. Apical periodontitis, commonly referred to as endodontic lesions, affects up to 61% of the European population, and approximately 5% of all teeth might display these pathological changes, which are easily detectable in radiographic images.[7] An endodontic lesion is an inflammatory reaction at the apex of a tooth formed to capsulate the infection in the root canal. Although the inflammatory area is smaller than that found in periodontitis, endodontic lesions can also contribute to systemic inflammatory and immunological burden, and thereby cardiometabolic disorders,[8] including diabetes.[7] However, the scientific evidence on the role of endodontic lesions in general health is not yet conclusive.

Periodontitis and endodontic lesions are the two main reasons for tooth loss in adults. Importantly, retaining teeth and maintaining good oral health is valuable in itself. This benefit was suggested in the present study,[4] which showed an improved quality of life due to the beneficial effects of IPT on working life, self-confidence, and living conditions. Functioning teeth might be expected to help people to be able to enjoy a healthy diet, but the evidence on the effect of missing teeth on the quality of diet are conflicting.[9] Crucially, however, missing teeth are also a valuable indicator of systemic health. In a population-based Finnish cohort who were followed up for 13 years,[9] number of teeth was associated with several risk factors and increased hazards for incident diabetes, cardiovascular events, and all-cause mortality. The risk increased with the number of lost teeth and it was especially evident for advanced tooth loss (ie, nine or more missing teeth at baseline). Moreover, adding information on missing teeth to the list of established risk factors provided a significantly improved clinical prediction profile for diabetes and cardiovascular disease. In general health practice, missing teeth could be a useful indicator of oral health and suggest that a more detailed dental and medical evaluation is warranted.

The results for IPT in the D'Aiuto and colleagues' study[4] are impressive in view of the reported local clinical measures and systemic inflammation biomarkers. Simple cleaning and polishing of the teeth is not sufficient for the proper treatment of patients with periodontitis, which requires more aggressive and repeated removal of the microbial biofilm, including the plaque and calculus. Chronic periodontitis is treated mainly by mechanical means, and might require surgery.[10] Most importantly, oral infections are always diagnosed and treated by a dentist (a specialised dentist if required) and the prescription of systemic antibiotics for bleeding gums by a physician is not appropriate. Naturally, the prevention of gingivitis and its conversion into periodontitis (and early diagnosis when periodontitis does occur) are primary goals. In susceptible individuals, such as people

with diabetes, a professional, biannual removal of biofilm would be an optimum choice.[10]

The treatment results achieved by D'Aiuto and collleagues[4] could suggest protective effects from both microvascular and macrovascular complications, which are important causes of morbidity and mortality in people with diabetes. Hopefully follow-up of these patients will continue—or other longer-term studies will be done—in order to confirm the beneficial effects of intensive treatment of periodontitis in people with diabetes.

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Psoriasis: a novel risk factor for type 2 diabetes

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What do psoriasis, a common chronic skin disease, and type 2 diabetes, a disorder of insulin resistance, have in common? On the surface, not much it would appear. However, advances in medical informatics, immunology, and genetics have challenged old paradigms, breaking down artificial boundaries between the two diseases. Previously, psoriasis was classified as a disease limited to epidermal hyperproliferation.[1] In 1979, the observation that cyclosporin was effective for psoriasis in a patient who required the drug for organ transplantation challenged the long-held dogma that psoriasis was merely a disease of a dysregulated epidermis.[2] Psoriasis is now believed to be a T-helper-17 autoinflammatory disease, characterised by a genetic predisposition, with over 60 genes identified (HLA-Cw6 being the most commonly implicated allele), and by chronic upregulation of innate and adaptive immune responses, antigen presentation (putative antigens being the keratinocyte-derived LL-37 and melanocyte derived ADAMTSL5), and increases in a variety of proinflammatory cytokines.[3]

Although psoriasis and type 2 diabetes are phenotypically distinct, they share several common pathophysiological pathways.[4] Genetically, *CDKAL1* has been linked to both type 2 diabetes and psoriasis.[5] More recently, novel susceptibility genes (*PTPN22*, *ST6GAL1*, *JAZF1*) were identified for psoriasis and type 2 diabetes in a Chinese population.[6] Moreover, the inflammatory cytokines that are upregulated in psoriasis are increased in the skin, adipose tissue, and blood of patients in a manner that positively correlates with the severity of skin disease. For example, tumour necrosis factor α and interleukin 6 are known to promote insulin resistance. Other shared pathways include leptin, adiponectin, insulin-like growth factor-II, and vascular endothelial growth factor.[7] Additional emerging pathways that are implicated include those of the glucagon-like peptide-1 receptor and the incretin effect.[8,9]

Long before the genetics and pathophysiology of psoriasis and type 2 diabetes were defined, the co-occurrence of these diseases was first reported in the 1890s.[10] Modern epidemiological approaches have been applied to explore the relationship between psoriasis and type 2 diabetes. In 2009, the Nurses' Health Study[11] demonstrated an increased incidence of type 2 diabetes in female nurses with psoriasis (n=1813), independent of risk factors such as BMI, smoking status, alcohol intake, and level of physical activity.[12] A pooled analysis of the Nurses' Health Study (women) and the Health Professionals' Follow-Up Study (men) showed that