Researchers have reported the first successful use of gene therapy to limit salivary gland inflammation and preserve saliva flow in a mouse model of Sjogren's syndrome. It was found that interleukin-10 (IL-10) gene transfer inhibits salivary gland inflammation and preserves saliva flow. The scientists found that transferring the gene for human IL-10 via an adeno-associated virus into the animals' salivary glands reduced salivary gland inflammation and preserved saliva flow. The treatment worked whether it was started before or after the onset of salivary dysfunction. The scientists describe their findings in the November 20 issue of Human Gene Therapy. Clinical application of this finding would greatly improve the quality of life and health of those afflicted with this autoimmune disease. The journal has prepublished the paper online [1].

The National Institute of Dental and Craniofacial Research (NIDCR) now has a web site on Health Disparities. The purpose of this web site is to provide a reference point for those interested in programmes and research aimed at reducing Health Disparities among all people. They have included documents and linkages that will provide access to broadly based information on this topic, including information that is relevant to our current initiative, 'Centers for Research to Reduce Oral Health Disparities'. For further information, visit the site [2].

Scientists have theorised that people with chronic periodontal disease may be predisposed to heart disease and stroke. However, supporting this hypothesis has been difficult, in part, because researchers have yet to identify a molecule or some other revealing biological marker that is in some way associated with these conditions. Recently, scientists reported that they may have found a probable indicator. As published online in the journal *Stroke*, the researchers found in a large, racially mixed group of adults that the more teeth a person has lost, the more likely he or she is to have both advanced periodontal infections and potentially clogging plaques in the carotid artery, the vessel that feeds the brain [3].

Should the theory prove to be accurate, it may be possible to help prevent or control the development of vascular disease in some individuals by treating their periodontal disease. Over the past decade, however, researchers have yielded mixed results to support the theory. According to some, these variable results do not necessarily disprove the hypothesis. Rather, they show how difficult research on complex biological problems can be without the needed specificity of biomarkers and other necessary research tools to simplify the process. The study is titled, 'Relationship between periodontal disease, tooth loss, and carotid artery plaque', and it was published online in the journal *Stroke*: Journal of the American Heart Association on Thursday, July 31, 2003. The authors are: Moise Desvarieux, Ryan T. Demmer, Tatjana Rundek, Bernadette Boden-Albala, David R. Jacobs, Panos Papapanou and Ralph L. Sacco [3].

A remarkable new treatment, ApoA-I Milano, pledges to unblock arteries, even in people with advanced heart disease. The original molecule was found in the blood of an Italian family with unusually healthy arteries, despite high levels of fats in their blood and low levels of protective 'good' high density lipoprotein (HDL) cholesterol. Now a genetically engineered version of this 'good' HDL cholesterol protein has been tested in a small human trial. The results, published in the November 5 issue of the *Journal of the American Medical Association*, astounded even the doctors who performed the trial [4]. Animal studies over the last 8 years have shown plaque regression in 5 weeks, with positive changes within 48 h.

It works in the following way: low density lipoprotein (LDL), or 'bad' cholesterol, clogs the walls of arteries when there is a surplus of LDL. HDL, the 'good' cholesterol, removes excess cholesterol from the body and takes it to the liver for elimination. Both LDL and HDL are packages in which cholesterol is linked to a molecule called apolipoprotein or Apo. HDL particles carry a form of Apo called ApoA-I. It is the key to cholesterol removal. A mutant form of ApoA-I, ApoA-I Milano, was discovered in Italy. Those with this form had little heart disease and tended to live long lives. Further study showed that the Milano version of ApoA-I has a special chemical property that seems to help it bind cholesterol in plaque very quickly. Esperion's ETC-216 (experimental heart drug) is a genetically engineered version of ApoA-I Milano [4].

A study by Nissen [5], shows that ApoA-I Milano shrinks plaque deposits in humans. It is a small study with 55 patients who got the drug, and only 36 completed the study. Nissen's study did not look at whether the patients' health improved; it only used a sophisticated imaging device, intravascular ultrasound (IVUS), to measure plaque reduction. The researchers have no indication whether changes in IVUS make a difference in heart attack risk. If plaque changes in human are seen, it may be an indicator of a smaller amount of heart attack risk. However, until clinical changes are observed, no conclusions can be reached. Another issue discussed is the *degree* of change seen in patients' plaque. In 5 weeks, patients treated with ApoA-I Milano had about a 4% decrease in plaque volume, that is, 10 times greater reduction than ever seen before, but in the overall

picture, 4% is not a huge number. However, this 4% reduction was observed in only 5 weeks. Larger clinical trials are needed. Clinical application is the unblocking of clogged vessels without the need for risky vascular surgery. The proposal being investigated is called reverse-cholesterol transport.

Also regarding cardiovascular disease, there is growing evidence that lifestyle factors influence heart disease risk as early as adolescence and even childhood. Two new studies suggest a link between early obesity, high cholesterol, high blood pressure and an elevated risk of cardiovascular disease later in life [6]. Research from Finland on this issue is published in the November 5 issue of *The Journal of the American Medical Association*. Although the young adults in the studies had no outward signs of heart disease, cardiovascular risk factors measured during childhood such as obesity and 'bad' LDL cholesterol were associated with increased carotid artery wall thickness. Carotid arteries on either side of the neck carry oxygen-rich blood away from the heart to the head and body. A thickening of the walls of these arteries because of fat and cholesterol deposits, atherosclerosis, is a marker for heart disease.

The researchers also found that adult measures of obesity, higher than normal LDL cholesterol levels, and systolic blood pressure were independent risk factors for carotid artery wall thickening. The men in the study had a higher overall risk than did the women, and blacks were at higher risk than whites. A second study found that risk factors measured between the ages of 12 and 18 were linked to carotid artery wall thickness before age 40 [7]. These risk factors included obesity, high levels of LDL cholesterol, high systolic blood pressure and smoking, and they remained significant after adjustment for other potential risks. These same risk factors, measured during adulthood, also predicted thickening of the carotid artery wall. The clinical implications are that prevention programmes aimed at kids and teens could have a dramatic impact on future public health.

Also related to cardiovascular disease, a new study showed that a person's risk of developing a potentially fatal blood clot, caused by sitting in an airline seat too long, can be four times higher within 2 weeks after taking a long international flight [8]. The likelihood of developing deep vein thrombosis, a condition also known as 'economy class syndrome', drops significantly 2 weeks after a long-haul flight, which is shorter than the current 2- to 4-week postflight 'hazard period' suggested by previous studies. Blood clots can travel through the bloodstream and lodge in the lungs, blocking blood flow and leading to severe organ damage or even death. The study also showed that the risk of deep vein thrombosis rises by 12% each year if a long-haul flight is taken annually [8].

Individual risk of a blood clot might be higher if certain medical conditions, such as varicose veins, cancer, a history of leg clots or recent leg or pelvic surgery or a leg injury, are present. Also, smokers, pregnant women, overweight individuals, the elderly and tall people may have a higher risk of flight-related deep vein thrombosis. Researchers state that although the average risk of deep vein thrombosis is small, airlines and health authorities should continue to advise passengers on ways they can minimise their risk, including: walking around the plane during long flights, stretching the legs and ankles when seated, avoiding excessive alcohol intake, and drinking plenty of water in flight.

As dental hygienists, we can pass this information to our clients, and possibly be instrumental in saving lives and increasing quality of life.

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