Clinical Implications: These findings confirm the value of ATP bioluminescence chairside testing to determine the caries risk based on bacterial numbers and biofilm load.

Researchers at Oregon Health and Sciences University in Portland, Oregon, compared laboratory methods of measuring bacteria to a chairside version. Laboratory cultures of plaque samples were grown to determine numbers and specific oral bacteria present. A laboratory assay and CariScreen, the chairside test, both measure ATP production by the bacteria using bioluminescence. This approach measures release of visible light by the bacteria. Measuring the energy potential of bacteria in the biofilm is reflective of actual cell numbers.

A total of 33 children ages seven to 12 years participated in the study. Plaque and caries were measured and a saliva sample was also collected. Plaque biofilm samples were taken from one tooth surface in each quadrant and parallel testing was done using the laboratory and chairside techniques. The chairside technique uses a swab to collect plaque biofilm. The swab is returned to its sheath and a bulb is opened on the opposite end releasing extraction components that drain over the biofilm swab. The closed sheath is then inserted into a handheld device for reading.

Both the laboratory and the chairside bioluminescence readings were comparable. Culture counts of bacteria reflected similar readings to the chairside test. Clinical indications of active caries also correlated highly with the chairside test scores.
Plaque pH Drops When Exposed to Sugar

This classic study, published in 1944, is referenced by many subsequent researchers. Prior to this study, Dr. W.D. Miller postulated in 1890 that decalcification of enamel was due to acids produced by bacteria metabolizing carbohydrates – something we still believe today. However, the acid-producing bacteria were found in the mouths of those with and without caries, leading to the concept of caries-susceptible and caries-immune people. Evidence was offered showing that the pH of a carious lesion was acidic by placing litmus paper on the open carious lesion, but resting saliva was not found to be in the dangerous acid range.

This study was undertaken to determine if plaque pH changes after a sugar rinse were the same in those with and those without caries. Five groups were tested: caries free, caries inactive, slight caries activity, marked caries activity and extreme caries activity. They were instructed to refrain from oral hygiene for three to four days before the test. Plaque pH was measured on the facial surfaces of maxillary and mandibular anterior teeth. Additional pH measurements were made on the gingival tissues, cheeks near parotid ducts, floor of the mouth near sub-maxillary ducts and the dorsum of the tongue. After baseline readings, subjects swished with a 10 percent glucose solution for two minutes. The pH reading was repeated after the rinse and every 10 minutes for an hour.

The drop in plaque pH and duration was greater in those with the most caries activity compare to those without caries activity. The plaque pH dropped below five only in those with caries activity. Interproximal surfaces might have lower pH and for longer times, due to the inaccessibility of saliva to flush the area, thus trapping carbohydrate food particles there longer.

Clinical Implications: These findings are referred to as the Stephan Curve, describing the impact of sucrose on the pH of bacterial plaque.

Saliva protects the teeth through antimicrobial functions, mechanically clearing bacteria from the mouth and buffering the acids, thus elevating the pH. Saliva is the primary host defense system against the bacteria and acids associated with caries. Saliva provides the balance between demineralization and remineralization.

The most important functions of saliva regarding caries are flushing and neutralizing. The higher the salivary flow rate, the better the oral clearance capacity. In general, those with reduced saliva often have a high caries incidence.

The buffering action of saliva is due to three buffering systems: bicarbonate, phosphate and protein. Reduced flow rate and reduced buffering capacity mean poor resistance to an acid attack. This is especially true among the elderly with xerostomia. Hormones, metabolic changes in the body and general health also influence the buffering capacity of saliva. Interestingly, as the flow rate decreases with malnutrition, the buffering capacity increases.

Proteins in saliva can either help or hinder the situation. Proteins are important in the formation of pellicle on tooth surfaces, providing protection from acids. However, some proteins assist bacteria in adhering to the pellicle-covered tooth surfaces. In the protective mode, the proteins cause oral bacteria to stick together and be flushed from the mouth.

The mucin protein, MG1, is higher in those susceptible to caries while MG2 is higher in those resistant to caries. One study shows MG2 to be four-times higher in caries-resistant people.

Immunoglobulins also influence the incidence of caries – some helping prevent caries while others hinder the preventive process. Differences in saliva between caries-susceptible and caries-resistant people suggest a host derived genetic influence.

Clinical Implications: Saliva is an amazing, multifactorial substance that can encourage the caries process in some and prevent it in others, depending on many factors. The more you know about a person’s saliva, the more effective your preventive plan will be.