Do you know the word “caries risk”? Caries risk refers to a susceptibility to dental cavities. Individuals with high caries risk are more likely to develop cavities despite frequent brushing, while those with low caries risk are less likely to develop cavities despite relatively infrequent brushing. What causes this difference? We’ll begin by explaining how dental caries begins.

Dental caries affects teeth, and its development involves the three factors of food, bacteria, and time.

Dental caries develops in the following stages:

- C1: Caries localized in the enamel without subjective symptoms such as pain
- C2: Caries reaching the dentine; creating sensitivity to cold
- C3: Caries reaching the dental pulp; associated with sharp pain
- C4: Caries affecting the roots

Caries develops faster with deciduous teeth and newly erupted permanent teeth than with permanent adult teeth. This is because the surface of a new tooth is soft and relatively easily dissolved in acid, increasing susceptibility to caries.
Streptococcus mutans is a well-known agent of caries, but just one of over 300 types of bacteria found in the oral cavity. After a meal, Streptococcus mutans uses the sugars occurring in the food as an energy source to multiply, producing acid as a metabolic byproduct. This acid dissolves the surface of the tooth (decalcification equals caries). The normal pH of the oral cavity is around 7. At a pH of 5.5 or below in an acid environment, calcium and phosphate ions are released from the surface of the tooth. This pH is called the critical pH. Since it takes about three minutes to reach the critical pH after food first enters the mouth, the tooth surface begins to dissolve during the meal.

And Streptococcus mutans produces not just acids, but dextrans, a viscous substance that allows bacteria to adhere to the tooth surface and multiply, creating plaque. Although plaque looks like food residue, it is actually a bacterial mass; 1 mg of plaque contains a few hundred million bacteria.

The adhesion of plaque to the tooth surface prevents acid diffusion, thereby maintaining high concentrations of acids within the plaque, facilitating decalcification and increasing caries risk. Plaque is not water soluble and cannot be eliminated by gargling, but is easily removed by brushing. Brushing after meals is important because it takes around three hours for plaque to form.

As mentioned at the start, individual differences in caries risk are attributable to the quality and quantity of saliva. Sugar intake will lower pH below the critical level of 5.5 and initiate decalcification, but saliva makes teeth to recalcify as well as decalcify. The inorganic component of tooth enamel is hydroxyapatite (chemical formula: Ca₁₀(P-O₄)₆(OH)₂), and the relationship between decalcification and recalcification can be represented by the following chemical formula:

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\text{Ca₁₀(P-O₄)₆(OH)₂} + 8\text{H}^+ \quad \text{Decalcification} \quad \text{Recalcification} \\
10\text{Ca}^{2+} + 6(\text{HPO}_₄)^{2-} + 2\text{H}_₂\text{O}
\]

Saliva naturally contains the calcium and phosphate ions used to build hydroxyapatite. Even when calcium and phosphate ions are released from the tooth surface, the supersaturation maintained by saliva promotes recalcification. This recalcification is facilitated by fluoride: when tooth crystals bind to fluoride, hydroxyapatite changes to fluorohydroxyapatite, which is more acid resistant than ordinary hydroxyapatite. This is why fluoride toothpaste is recommended. Saliva also contains bicarbonate, which neutralizes oral acidity. Since more saliva is produced when the salivary glands are stimulated, chewing food increases bicarbonate levels and improves the quality and quantity of saliva, reducing caries risk.