

## Sclerotic or Translucent Dentin: A Review

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### ABSTRACT:

Sclerosis of dentinal tubules may be a response to pathological or physiological insult in the crown & root that starts in the root and gradually extend to the crown of the tooth in case of physiological sclerotic dentin. The exact mechanism of the formation of sclerotic dentin is not known, however various authors have proposed different possible mechanisms, which are not universally accepted but, it is generally agreed that sclerotic areas of root dentin increases in size with age.

**Keywords:** Sclerotic dentin, Dentinal tubules.

### INTRODUCTION

Dentin is a mineralized connective tissue. Similar to all other mineralized tissues, it is laid down as an organic matrix which subsequently mineralizes. The organic matrix of dentin is formed by odontoblasts. The initial and foremost mineralization occurs during dentinogenesis while a gradual mineralization continues with age<sup>1</sup>. Odontoblasts form and maintain dentin and are located in the pulp, sending a long, cellular extension into the dentin, called the odontoblastic process.

The dentinal tubules contain either the odontoblastic processes or their remnants and tissue fluid (also called "dentin liquor"). The odontoblast layer is an integral part of the dental pulp. This intimate relationship between the cells of the dentin and the pulp represents as one functional entity<sup>1</sup>.

On the basis of histologic characteristics and time of development, dentin has been termed into primary, secondary and tertiary dentin. Primary dentin is the major component of the crown and root, which is formed before root completion, the dentin formed after the root completion is the secondary dentin and responsive dentin formed to protect pulp is termed as tertiary dentin<sup>2</sup>

Sclerotic dentin is one of the form of dentin which may be a response to pathological or physiological insult in the crown & root that starts in the root and gradually extend to the crown of the tooth in case of physiological sclerotic dentin.

### SCLEROTIC DENTIN

Aging manifest as cumulative changes in various

tissues including dental hard tissue. One such age related change is the formation of physiologic translucent dentin (sclerotic dentin).

Sclerotic dentin, which forms gradually with aging, begins at the apical end of the root and in the periphery of the dentin, later extending into the coronal dentin. Physiologic sclerotic root dentin, as distinguished from pathologic sclerosis subjacent to caries, appears to form without trauma or caries attack as a natural part of aging.

### MECHANISM OF SCLEROTIC DENTIN FORMATION

Sclerotic dentin is formed either as a reactive process or aging and is seen in the occlusal and apical part, latter being more common.

Stimuli may not only induce additional formation of reparative dentin but also lead to protective changes in the existing dentin. In situation of caries, attrition, abrasion, and cavity preparation, sufficient stimuli are created to cause collagen fibers and apatite crystals to begin appearing in the dentinal tubules. Apatite crystals are initially only irregular in a dentinal tubule but gradually the tubule becomes filled with a fine meshwork of crystals. Thereby the difference in refractive indices between intratubular organic and extratubular inorganic materials will be equalized resulting in increasing translucency of the dentin so affected<sup>3</sup>.

Sclerotic dentin may be related to aging, when associated with the dentinal tubules close to root apex. The root dentin usually appears to become transparent during the third decade that starts at the

tip of the root and advances coronally with age<sup>4</sup>. The alteration is believed to be caused by a reduction of the diameter of the dentinal tubules caused by intratubular calcification.

The exact reason for intratubular calcification is not known but suggested that translucent dentin has been regarded to be caused by changes in the organic substances of the dentin, possibly a fatty degeneration<sup>5</sup>. However the theory was questioned since permeability of dentin was found to be not affected by the removal of fat by solvents<sup>6,7</sup>.

Sclerotic dentin was considered to be due to secondary dentin formation on the sides of the pulp chamber leaving no room for the odontoblasts, which gradually die off, allowing calcification of the tubules<sup>6</sup>. However this theory can also be probed since secondary dentin is found at the incisal end of the pulp chamber, but transparency starts with the most apical tubules.

Another explanation put forward by Jenkins was that the tubules become narrower by deposition of calcium salts upon the peritubular dentine of the walls. This leads eventually to the total occlusion of individual tubules<sup>6</sup>.

Other explanation was based on "Dissolution & Reprecipitation" where a smaller mean intertubular crystal size could be the result of dissolution of mineral from the intertubular matrix, which is then deposited into the tubules. The similar chemical composition of the inter and intra tubular mineral in transparent dentin supports such a "Dissolution – Reprecipitation." It is also possible that the dissolved mineral is re-deposited within the intertubular matrix instead of within the dentin tubules lumens. With age, the intertubular crystal dissolve uniformly, thereby decrease in size, and the mineral is reprecipitated within the tubules as a result of an increased saturation of calcium and phosphate ions within the dentinal tubules<sup>8</sup>.

Chemical analysis suggests that the material occluding the lumina of the dentinal tubules of the sclerotic areas of root dentin is mineralized to about the same extent as cementum<sup>9</sup>. Microradiography shows that the degree of mineralization is more like that of peritubular dentin<sup>10</sup>.

According to Nalbandian et al, occlusion of the dentinal tubules in the translucent zones of root dentin is the result of the centripetal growth of the peritubular dentin<sup>10</sup>. These results conflict with findings of Vasiliadis L et al, who found that the annular space between the peritubular dentin & the occluding material might be the result of difference in structure. The shape of the exposed parts of the

occluding rods indicated that they were not part of the peritubular dentin which had been accidentally fractured at this level, but was a material of different structure<sup>11</sup>

In physiological sclerosis the occluding material presents a relatively smooth surface and there is characteristically a plane of separation or cleft between it and the surrounding dentin, which may be either intertubular or peritubular dentin. Transmission electron microscopy of sclerotic root dentin found to have same electron density as peritubular dentin in its final stages of formation and concluded that the developmental process of the occluding material is different from that of peritubular dentin<sup>12</sup>.

According to present knowledge, the formation of peritubular dentin is governed by the odontoblast and its process. It has been shown that the process of development and formation of peritubular dentin proceeds in an appositional fashion maintaining a clear forming front, whereas the occlusive changes occurs diffusely, with the packing of the occluding minerals increasing with time<sup>13</sup>.

Absolute increase in the amount of mineral in the sclerotic dentin indicates that the occluding mineral is not derived from the adjacent intertubular dentin but originates from another source like the pulp because, if it arose from an external source and passed through the cementum, the process of sclerosis would be self-limiting<sup>11</sup>.

Study focused on differences in the structure and mechanical properties of normal versus transparent dentin, stated that the mineral concentration, as measured by X – ray computed microtomography, was significantly higher in transparent dentin, the elevated concentration being consistent with the closure of the lumens. Crystallite size, as measured by small angle X – ray scattering, was slightly smaller in transparent dentin, unlike normal dentin, exhibiting almost no yielding before failure. In addition, the fracture toughness was lowered by roughly 20% while the fatigue lifetime was deleteriously affected at high stress levels<sup>14</sup>.

When the effect of aging on the mineral phase of dentin, was studied by high resolution TEM and revealed that the intertubular mineral crystallites were smaller in transparent dentin, and that the intratubular mineral (larger crystals deposited within the tubules) was chemically similar to the surrounding intertubular mineral<sup>8</sup>.

The increase in size of apical zone of sclerotic dentin in human teeth has been used in forensic science as a method of age estimation, as one of several

regressive changes related to age 4 or, more commonly, as the sole variable since the finding that the increase in apical translucency was the factor most commonly linked to age<sup>15,16</sup>.

**CONCLUSION:**

Knowledge of the development and distribution of sclerotic dentin which produces translucency in root dentin is still incomplete. The theories proposed to explain the cause and mechanism of sclerotic dentin formation are not universally accepted but, it is generally agreed that sclerotic areas of root dentin increases in size with age.

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