Review

Dentin Hypersensitivity: Etiology, Diagnosis and Contemporary Therapeutic Approaches—A Review in Literature

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Abstract: The aim of this review was to provide dentists with comprehensive information regarding dentin hypersensitivity. This includes presenting its etiology, outlining the process of diagnosis, discussing clinical management strategies, and exploring technical approaches aimed at alleviating sensitivity. Dentin hypersensitivity is characterized by distinctive short, sharp pain arising from exposed cervical dentin in response to various external stimuli. The etiological factors cause two specific changes in teeth. First, the dentin surface must be exposed and denuded, which requires the loss of enamel or gingival recession combined with the loss of cementum. The second condition is the opening of the dentin tubules to allow the sensory mechanisms in the pulpal area following stimulation of the dentin surface. The accurate diagnosis of dentin hypersensitivity before receiving therapies is critical for successful treatment. The diagnosis of the disease starts through investigating the medical history of the patient and examination. In the examination, some techniques such as air stream and water rinsing are used in order to simulate the stimulating factors and to determine the degree of pain of the patient. Numerous materials and methods have been proposed to reduce or alleviate sensitivity. These materials typically exert their effects by either sealing dentin tubules or disrupting the transmission of nerve impulses. Generally, the primary approach in addressing dentin hypersensitivity involves the utilization of toothpaste containing potassium salts and fluoride. Newly introduced materials and in-office methods for treating dentin hypersensitivity include bioactive glasses, iontophoresis, CPP-ACP, and lasers.

Keywords: bioactive glasses; dentin hypersensitivity; diagnostic methods; fluorine compounds; lasers; in-office treatments; etiological factors

1. Introduction

Dentin hypersensitivity (DH) is characterized by brief and intense pain, occurring in teeth with exposed cervical dentin. This pain is triggered by different external stimuli, including thermal changes, cold air evaporation, tactile pressures (e.g., probing), electrical sensations, osmotic influences, or chemical exposures. Importantly, this pain cannot be attributed to any other dental pathology, defect, or disease [1–4].

A widely accepted theory that explains the sensation of pain resulting from external stimuli is the “hydrodynamic theory”, initially proposed by Brannstrom et al. [5]. This theory suggests that the movement of fluids within dentin tubules is responsible for the sensation of pain [5,6]. To induce DH, two surface changes must occur on the tooth: the exposure and removal of dentin due to receding gums, coupled with the loss of cementum, and the enlargement of dentin tubules [7,8]. Gum recession and dentin exposure can arise from factors such as excessive or incorrect brushing techniques, as well as periodontal disease [7].

Accurately diagnosing DH requires a substantial amount of time, as it involves obtaining a comprehensive dental history and relies on the process of eliminating other
comparable conditions. When symptoms of DH are present, it is imperative to conduct a differential diagnosis in order to distinguish it from other forms of orofacial pain, such as pulp inflammation, periodontal discomfort, tooth cracking, microleakage, and atypical odontalgia, among others [7,8].

Although DH is most frequently observed in premolars and canines, all teeth and surfaces are susceptible to experiencing hypersensitivity. The prevalence of DH has been documented to vary significantly across various studies, even reaching up to 100%. This variability arises from the diverse population groups studied and variations in assessment methodologies [9].

Despite the numerous methods proposed for treating DH, a definitive solution to the issue has yet to be identified. Clinical studies evaluating the efficacy of different treatment approaches yield contradictory results [10–12]. This discrepancy stems from the fact that all these studies assess pain intensity, a parameter that proves notably challenging to evaluate due to its inherently subjective and intricate nature [13].

Therefore, the objective of this literature review was to furnish dental practitioners with comprehensive information concerning DH. This includes outlining its etiology, diagnostic methods, and treatment options, while also addressing the effectiveness of the approaches employed to manage DH.

2. Etiology of Dentin Hypersensitivity

Based on the hydrodynamic theory of dentin sensitivity, thermal, osmotic, tactile (from probe pressure), and drying-induced evaporation stimuli transmit sensations to the pulp nerves through the dentin tubules. These tubules contain fluid, originating from pulp fluids. Dentin hypersensitivity typically occurs when external stimuli are applied to the surface of exposed dentin with open tubules. Different stimuli lead to rapid fluid movement within the tubules, creating pressure changes that activate pressure receptors located close to the pulp. This activation results in immediate acute pain [14]. Unlike other stimuli, heat causes a gentle movement of the dentinal tubule fluid toward the pulp, leading to a milder activation of the pressure receptors and consequently causing less pain [14].

It has been discovered that clinically hypersensitive teeth exhibit an exposed dentin tubule count approximately eight times higher and a dentin tubule diameter around two times larger in comparison to non-sensitive teeth [15,16]. This discrepancy in dentin tubule diameter between hypersensitive and non-sensitive teeth appears to hold clinical significance [14]. Two prerequisites are necessary for DH to occur on a tooth’s surface. Firstly, there must be exposure and removal of dentin, which can result from enamel loss or a combination of receding gums and the loss of root cementum. Secondly, the dentinal tubules must become accessible, allowing external stimuli to activate the sensory mechanism present on the dentin surface [6].

Gum recession and dentin exposure can be attributed to excessive brushing, improper brushing techniques, and periodontal disease, often arising after either surgical or nonsurgical treatment [14]. While regular brushing typically does not lead to substantial enamel loss, the combination of acid erosion from acidic foods and beverages with brushing can result in significant tooth tissue loss across all tooth surfaces, with a particular impact on cervical regions [17]. During the removal of tartar from root surfaces, it is common for scalers to inadvertently eliminate a thin layer of cementum (20–40 μm), giving rise to the creation of a smear layer containing blood and saliva that covers the underlying dentin [18].

Pashley and Tay (2012) [18] demonstrated that dentin tubule fluid gradually permeates the smear layer and moistens the dentin surface. However, the smear layer presents significant resistance (86%) to the outward flow of fluids through the dentin [18,19]. Generally, the presence of the smear layer offers patients protection against dentin sensitivity, as it effectively seals the dentin tubules, even more efficiently than the resin infiltrations formed by bonding agents [20,21]. This smear layer, however, does not persist on the dentin surface for an extended period. Over approximately 7–10 days, microbes accumulate on it, forming a biofilm that produces acids capable of dissolving the smear layer [20]. The loss of the
smear layer leads to increased dentin permeability, resulting in the emergence of dentin sensitivity. In some instances, the level of sensitivity can escalate further, progressing to the state of hypersensitivity from the prior condition of simple dentin sensitivity.

3. Pain Mechanism of Dentin Hypersensitivity

While considerable knowledge has been acquired regarding DH since its initial documentation, there remains a deficiency in clinical evidence-based research, particularly concerning the underlying pain mechanisms, which remain incompletely understood [22]. The theory that continues to hold the most widespread acceptance posits that sensitive dentin arises from the stimulus-triggered fluid flow within the dentin tubules, leading to the subsequent activation of nociceptors at the border between the pulp and dentin [23]. It is believed that intradental myelinated A-β fibers and some A-δ fibers respond to stimuli that displace the fluid within the dentin tubules, culminating in the distinct, brief, and sharp pain experienced in cases of DH [24].

Despite the fact that the inner diameter of dentin tubules measures about 1 µm, their hydrodynamic behavior, influenced by collagen fibers and calcium phosphate salts within them, resembles that of tubules with a functional diameter of less than 0.1 µm [21]. Consequently, while microbial penetration through the dentin tubules into the pulp is unlikely, microbial byproducts, including endotoxins and exotoxins, can readily traverse the tubules and reach the pulp [25,26]. In cases where toxins induce localized inflammation of the pulp, an escalation in pulpal pressure occurs, consequently increasing fluid outflow. This process ultimately leads to increased sensitivity of the pulp nerves beyond normal levels. Furthermore, localized pulp inflammation can prompt the proliferation of nerve endings located close to the dentin tubules, intensifying the overall sensitivity of the pulpo–dental complex [21,27].

The proliferation of nerve endings is triggered by inflammation mediators, such as histamine, bradykinin, prostaglandins, neuropeptides, and others. These mediators induce the proliferation of fibroblasts, aiming to restore the damaged collagen within the pulp’s connective tissue due to inflammation [28–30]. Furthermore, the cell membrane receptors on the newly formed nerve branches identify microbial antigens and inflammatory byproducts. This recognition activates mechanisms for generating new protein factors that subsequently stimulate sodium channels. As a result, these channels are further stimulated, leading to an increased sensitivity of the pulp [31].

It has been noted that DH can, in certain instances, gradually subside without external therapeutic intervention, often following a period of exacerbation [32,33]. Experimental studies conducted in vivo by Pashley et al. [30] offered insight into this phenomenon. When cavities were created in dog teeth and dentin permeability was measured hourly over an 8 h span, a decline of 15% in permeability occurred each hour in living teeth, in contrast to pulpless teeth [33]. The researchers deduced that this decrease in permeability resulted from the release of plasma proteins from the pulp into the fluid of the dentin tubules, leading to a reduction in tubule diameter. The significance of fibrinogen, the largest plasma protein, in reducing dentin permeability after cavity preparation was also experimentally confirmed in dogs [34]. Additionally, other researchers discovered that immunoglobulins, such as IgG, can induce a gradual reduction in dentin permeability over time [35].

Consequently, microbial toxins originating from saliva and plaque have the capability to infiltrate the pulp, prompting pulp irritation, elevating pulp pressure, stimulating new neuron growth, and increasing nerve sensitivity [21]. These toxins are believed to underlie the sustained presence of DH within a subset of patients who initially experienced it. It is plausible that this persistence of hypersensitivity is linked to multiple episodes of localized pulpitis in these patients, which eventually resolves through pulpal eschar formation [21]. The escharated tissue lacks vasculature, potentially hindering the delivery of sizable proteins into the dentin tubules, unlike the norm for a healthy pulp. Furthermore, the reaction of the pulp to microbial toxins might not be uniform across all patients [21].
4. Diagnosis of Dentin Hypersensitivity

4.1. Clinical Examination

The accurate diagnosis of DH demands a substantial amount of time, primarily because it involves obtaining a thorough dental history and relies on the process of eliminating other analogous conditions. In the course of the clinical examination, patients are questioned about discomfort experienced when consuming hot, cold, iced, acidic, or soft foods and beverages. Additionally, patients are inquired about whether symptoms manifest during brushing or emerge subsequent to a dental restoration procedure. In instances of pain, patients are further queried about specific attributes of the pain, encompassing its location, character, intensity, timing, and the particular stimulus that triggers it [36].

Subsequently, the patient’s personal habits (including the consumption of acidic foods and beverages, as well as excessive brushing) and prior dental interventions (such as teeth scaling, other periodontal treatments, bleaching, and fillings) are assessed. Furthermore, the presence of corrosion, receding gums, and the exposure of cervical dentin is examined. As long as symptoms of DH are present, it becomes essential to differentiate and diagnose other forms of orofacial pain, such as pulp inflammation, periodontal discomfort, tooth fractures, microleakage, atypical odontalgia, and more [36]. Following this step, a specific clinical assessment of DH is conducted by gently probing the exposed dentin in the mesial–distal direction. To confirm the diagnostic outcome, a second stimulus is typically employed, often utilizing an air stream from an air syringe [4,37].

Once the diagnosis is confirmed, patients are provided with instructions to mitigate predisposing factors such as acidic foods and drinks, as well as incorrect brushing techniques [38,39]. Treatment then commences with agents that alleviate sensitivity, either at the dentist’s office or through at-home methods [14]. Individuals suffering from DH often recognize a diminished quality of oral health compared to the general population. Employing a questionnaire specifically focused on the impact of DH on patients’ quality of life would significantly enhance the disease diagnosis process within routine dental practice. Regrettably, the two most widely recognized and validated questionnaires employed in various clinical studies focusing on oral health consist of a minimum of 48 questions each [40,41]. Given the extensive nature of these questionnaires and their practical challenges, there have been recommendations to employ shorter versions consisting of around 12 to 14 questions, referred to as the Oral Health Impact Profile – 14 and the General Oral Health Assessment Index [42,43].

4.2. Assessment and Recording of Dentin Hypersensitivity

When addressing DH, it is often necessary to document the severity of symptoms or the effectiveness of implemented treatments. The assessment of DH in clinical studies should always be regarded as subjective and reliant on the individual and distinct responses of each patient to various stimuli [36,44]. The perception of pain stemming from a stimulus on the exposed dentin surface is influenced by a range of different factors unique to each patient, encompassing psychological, cultural, emotional, and socio-economic elements [36].

Patients’ responses tend to be more pronounced when exposed to sudden stimuli in contrast to anticipated ones. Furthermore, it is acknowledged that interactions with healthcare providers can trigger psychological effects in patients, potentially giving rise to phenomena such as the placebo, nocebo, and Hawthorne effects [36,45,46]. In particular, the placebo effect constitutes a multifaceted psychophysiological reaction caused by the administration of a sham treatment. Essentially, patients experience an improvement in their condition after interacting with their dentist and expecting positive outcomes following medical advice contributes to symptom amelioration [45,47]. Conversely, the nocebo effect, which operates through negative expectations, can induce adverse side effects even within placebo treatments, shifting symptoms from positive to negative [48]. Currently, it is believed that, under normal circumstances, individuals are subject to varying degrees of susceptibility to the placebo effect, making it challenging to overlook or categorize those who respond positively.
The Hawthorne effect represents another psychological facet in the patient–dentist interaction, wherein individuals alter or enhance aspects of their behavior in response to the perception of being observed [46]. These factors may provide insight into the scarcity of robust evidence or the presence of contradictory outcomes in clinical studies concerning the efficacy of different treatment options [49]. The potential for quantitatively assessing patients’ sensitivity to external stimuli on the exposed dentin surface could offer a more precise determination of the problem’s extent and assist in evaluating various treatment approaches in a more objective and dependable manner.

Two distinct approaches for evaluating dentin sensitivity have been outlined. Firstly, sensitivity can be assessed based on the strength of the stimulus required to induce pain (stimulus-based assessment), and secondly, a subjective evaluation of the pain provoked by a specified stimulus is conducted (response-based assessment) [3,50]. Response-based methods gauge the immediacy of pain following the application of a consistent, dependable, and repeatable stimulus. Normal stimuli such as probe pressure (tactile stimulus), cold, and evaporative air are frequently employed due to their easy control and reproducibility [51,52]. Given that dentin sensitivity can vary in response to different stimuli, it is recommended to employ a minimum of two distinct stimuli during the examination to validate the outcome [3,53]. Among these stimuli, probe (tactile) stimulation has been observed to be less effective than thermal and drying stimuli. When applying various stimuli, it is advisable to commence with the least intense stimulus initially [44,50].

5. Dentin Hypersensitivity Assessment Methods

5.1. Response-Based Methods

Within response-based methods, the stimulus remains constant while the patient’s response varies. An illustrative example of this approach involves employing an air current for a specific duration. During this method, adjacent teeth are isolated using the examiner’s fingers, and a stream of air with a pressure of 60 ± 5 psi and a temperature of 21 ± 1 °C is directed perpendicularly to the tooth under examination from a distance of 1 cm for a duration of 1 s. Immediately following the stimulus, the patient rates the pain by visually recording it on an analog table (visual analog scale—VAS), which features gradations spanning from no pain to the most intense pain (Figure 1) [54]. Alternatively, another method entails using a pain scale wherein the patient verbally describes the pain intensity [55]. Utilizing VAS for pain recording offers the advantage of being more accurately portrayed the various levels of pain intensity.

![Visual analogue scale (VAS)](image)

**Figure 1.** The visual analog scale that is used for assessment of dentin hypersensitivity.

Schiff’s scale of sensitivity to cold air, frequently introduced, comprises four distinct gradations as follows [51,56–58]:

0—The patient displays no response to the air stimulus.
1—The patient reacts to the air stimulus without requesting it to cease.
2—The patient reacts to the air stimulus and either requests it to stop or moves away from it.
3—The patient responds to the air stimulus, deeming it painful, and explicitly asks for it to stop.
5.2. Stimulus-Based Methods

In stimulus-based investigations of dentin sensitivity, the stimulus is adjusted with varying levels of intensity. One frequently used approach in this category involves utilizing a probe that is adjusted to exert forces on the tooth in increments of 10 g, employing an electronic device for control [51,56,58]. In this method, the patient is directed to indicate the point at which they initially experience pain subsequent to the probe stimulation, and the applied force is recorded. While thermal and electrical devices have been previously used to generate increasing stimulus intensities, concerns have arisen regarding their efficacy [59]. Drawbacks of methods hinging on stimulus intensity include the potential for repeated stimuli to alter sensitivity and the possibility that the expectation of pain linked to the stimulus could influence the degree of response. Additionally, the time-intensive nature of these approaches leads to a reduction in the number of teeth that can be examined within a given session [59].

6. Therapeutic Approaches of Dentin Hypersensitivity

The treatment of patients with DH entails a structured series of steps, comprising six key points [2]:

1. Accurate diagnosis subsequent to recording the patient’s dental history and initial clinical examination.
2. Identification of etiological and predisposing factors, with particular attention to dietary and brushing habits and their potential impact on erosion and abrasion.
3. Differential diagnosis to rule out other conditions presenting similar pain symptoms.
4. Therapeutic intervention for any coexisting conditions manifesting symptoms similar to DH.
5. Removal or mitigation of etiological and predisposing factors, accompanied by guidance on appropriate dietary habits and oral hygiene practices.
6. Dispensing patient instructions or implementing office-based treatments in accordance with the patient’s requirements.

Addressing the potential etiological factors contributing to DH requires comprehensive patient education. The patient should be instructed in proper brushing techniques, advised against using abrasive toothpaste (such as whitening dentifrices) [60] and firm toothbrushes, cautioned against excessive brushing force and advised to abstain from brushing for at least one hour after consuming acidic foods or beverages. Acidic substances, whether from dietary intake or gastric reflux, have been identified as triggers or exacerbators of DH [61]. Following the consumption of acidic foods or beverages, the consumption of alkaline drinks like milk or neutral options such as water is recommended. Practicing proper oral hygiene can also play a substantial role in maintaining healthy periodontal tissues and preventing gum recession, which can lead to root exposure and potentially trigger DH [61].

6.1. At-Home Therapeutic Treatments of Dentin Hypersensitivity

For at-home management of DH, patients receive guidance to utilize specialized toothpastes and mouthwashes designed to effectively alleviate sensitivity [62–64]. These approaches function by either impeding nerve impulse transmission or blocking dentin tubules (Table 1). If DH persists for 2 to 4 weeks after utilizing specialized toothpaste or mouthwashes, patients should consider progressing to the second phase of treatment, which involves professional treatment at the dentist’s office.
Table 1. The at-home therapeutic treatments for dentin hypersensitivity.

<table>
<thead>
<tr>
<th>Active Agents</th>
<th>Form</th>
<th>Mechanism of Action</th>
<th>Duration of Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potassium salts</td>
<td>Toothpaste–mouthwash</td>
<td>Impeding nerve impulse transmission</td>
<td>2 times daily for 2–4 weeks</td>
</tr>
<tr>
<td>Sodium fluoride</td>
<td>Toothpaste–mouthwash</td>
<td>Occlusion of dentin tubules</td>
<td>2 times daily for 2–4 weeks</td>
</tr>
<tr>
<td>Stannous fluoride</td>
<td>Toothpaste–gel</td>
<td>Occlusion of dentin tubules</td>
<td>2 times daily up to 2 weeks</td>
</tr>
<tr>
<td>Arginine</td>
<td>Toothpaste–mouthwash</td>
<td>Occlusion of dentin tubules</td>
<td>2 times daily for 2–4 weeks</td>
</tr>
<tr>
<td>Hydroxyapatite or</td>
<td>Toothpaste–mouthwash</td>
<td>Occlusion of dentin tubules</td>
<td>2 times daily for 2–4 weeks</td>
</tr>
<tr>
<td>nano-hydroxyapatite</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bioactive glass</td>
<td>Toothpaste–mouthwash</td>
<td>Occlusion of dentin tubules</td>
<td>2 times daily for 2–4 weeks</td>
</tr>
<tr>
<td>CPP-ACP</td>
<td>Toothpaste–mousse</td>
<td>Occlusion of dentin tubules</td>
<td>2 times daily for 2–4 weeks</td>
</tr>
<tr>
<td>Strontium chloride</td>
<td>Toothpaste–mouthwash</td>
<td>Occlusion of dentin tubules</td>
<td>2 times daily for 2–4 weeks</td>
</tr>
</tbody>
</table>

6.1.1. Potassium Salts

Many DH-reducing toothpastes contain potassium salts such as potassium chloride (KCl), potassium citrate (K₃C₆H₅O₇), and potassium nitrate (KNO₃) [65]. Through penetration into dentin tubules, these salts numb nerve fibers, consequently diminishing tooth sensitivity. More specifically, they can increase the pain threshold of nerve fibers in response to specific stimuli [66,67]. Toothpastes containing KNO₃ and fluoride have demonstrated positive impacts on DH reduction [66,68–70]. For optimal outcomes, these toothpastes should be used in conjunction with soft toothbrushes and minimal water, two times daily following tooth brushing for at least 14 days [66]. Moreover, mouthwashes containing potassium oxalate (K₂C₂O₄), potassium nitrate, and fluoride are also deployed for daily use, significantly contributing to DH alleviation [71].

6.1.2. Sodium Fluoride

The most common fluorine compound in toothpastes is sodium fluoride (NaF). Fluoride helps to deposit minerals, such as calcium and phosphate, onto the dentin surface, which can help strengthen and harden it. Toothpastes incorporating NaF and calcium phosphate [Ca₃(PO₄)₂], aimed at dentin remineralization, also notably mitigate DH. When fluoride ions come into contact with the tooth’s surface, they can block the exposed dentin tubules [72].

6.1.3. Stannous Fluoride

Stannous fluoride (SnF₂) has shown remarkable effectiveness in sealing dentin tubules, which can be attributed to the strong attraction of Sn ions to the mineral surfaces of tooth hard tissues [73]. It creates a protective layer on the tooth’s surface by establishing bonds with exposed tissues. More specifically, tin ions (Sn²⁺) become part of the near-surface layer of the tooth, penetrating to a depth of around 20 µm and providing extended protection, guarding the underlying surface against further external stimuli. Moreover, the bond between F⁻ and Ca²⁺ resulting from the application of various fluoride salts is less robust compared to the bond established on tooth surfaces treated with SnF₂ [74]. In a previous study [75], it was observed that after 8 weeks of using a toothpaste containing 0.454% SnF₂, the dentin surfaces displayed effective coating, with the open dentin tubules being sealed. In contrast to the control toothpaste, the experimental toothpaste showed a significant
reduction in tooth sensitivity, underscoring its notable efficacy. In addition, in a recent investigation, it was reported that the ability of a SnF$_2$ gel to occlude dentin tubules reached 99.5% [76]. Nonetheless, despite its effectiveness, SnF$_2$ presents some disadvantages, such as a metallic taste, the potential for tooth staining, and the possibility of diminished taste sensation with prolonged use [74].

6.1.4. Arginine

Another category comprises toothpastes containing the amino acid arginine (C$_6$H$_{14}$N$_4$O$_2$), recognized for their substantial DH reduction potential and anticariogenic effects [77–80]. These formulations, containing 8% arginine (arginine bicarbonate), calcium carbonate (CaCO$_3$), and 1450 ppm fluoride, establish an alkaline environment that leads to reduced calcium and phosphorus levels on the dentin surface and within the dentin tubules. The arginine works by interacting with calcium ions to create a calcium-rich layer that can occlude the dentin tubules. One notable benefit of arginine-based toothpaste is its potential for long-lasting relief [81].

6.1.5. Nano-Hydroxyapatite

Currently, toothpastes containing nano-hydroxyapatite (n-HA) have emerged delivering positive effects in DH reduction [82–84]. Nano-hydroxyapatite is a form of calcium phosphate that is structurally similar to the hydroxyapatite found in natural hard tooth tissues. When applied to the tooth surface, n-HA can mimic the natural mineral composition of the tooth structure, which helps to replenish lost mineral content in areas where dentin is exposed. By strengthening and remineralizing the tooth, n-HA can reduce the sensitivity caused by the exposure of dentin tubules [85,86]. Furthermore, mouthwashes containing n-HA, and fluoride are also deployed for daily use, significantly contributing to DH reduction [87,88].

6.1.6. Bioactive Glasses

Additionally, toothpastes containing bioactive glasses in combination with fluoride have been suggested to alleviate the symptoms of DH. Bioactive glasses can effectively occlude or block the dentin tubules. When applied to the tooth surface, bioactive glass particles can penetrate the tubules and form a physical barrier within them, as well as attract tooth structural ions (Ca$^{2+}$ and PO$_4^{-3}$) and promote the formation of hydroxyapatite crystals [76,81,89].

6.1.7. Casein Phosphopeptide-Amorphous Calcium Phosphate (CPP-ACP)

A recently introduced dentin remineralizing agent derived from milk proteins is known as Recaldent™. This product incorporates phosphopeptides and plays a role in retaining and stabilizing amorphous calcium phosphate. CPP-ACP possesses the capability to remineralize initial subsurface enamel lesions [90]. Additionally, it has been asserted that CPP-ACP significantly contributes to both the prevention and treatment of DH. The mechanism by which CPP-ACP operates involves a series of processes that collectively contribute to its beneficial effects on tooth surfaces. Firstly, CPP-ACP enhances the availability of binding sites on the tooth surface for Ca, which results in a reduction of the continuous diffusion of Ca. Moreover, ACP functions as a buffer for free calcium and phosphate ions found in dentin, helping to maintain a state of supersaturation. This state is crucial in preventing demineralization and promoting the remineralization of the tooth’s structure [91]. Another reason for the positive outcomes of CPP-ACP is that its application facilitates the formation of a crystalline layer that covers the openings of dentin tubules. This protective layer acts as a barrier against external stimuli, providing additional protection for the tooth surface [92]. However, this layer is not entirely uniform; it exhibits attached irregularities and appears as clusters of structures that fill the interprismatic spaces while partially covering the tubules [76].
6.1.8. Strontium Chloride

Strontium chloride (SrCl₂), usually at a concentration of 10%, operates in a manner reminiscent of calcium within the human body [93]. It has the potential to act as a substitute for apatite biomineralization, as has been described in previous reports [94]. Within the oral environment, Sr⁺², along with Ca⁺² and other divalent cations sharing a similar charge/size ratio as calcium, can seamlessly integrate into the hydroxyapatite structure [95]. The scientific literature also highlights that this active element stimulates dentinogenesis in human dental pulp stem cells. This stimulation, as it has been demonstrated in previous in vitro studies, may foster their proliferation, differentiation, and mineralization, leading to the formation of tertiary dentin [96].

One of the key attributes of SrCl₂ is its capacity to create intricate strontium phosphate compounds, thus exerting a chemical influence on dentin [93]. A previous report [97] affirmed the formation of a Ca and Sr apatite denoted as Ca₆Sr₄(PO₄)₆(OH)₂, which emerges through the replacement of intracrystalline Ca in apatite with Sr. Furthermore, the affinity of strontium salts toward dentin remains significant due to the increased permeability of dentin and the likelihood of absorption in organic connective tissues as well as odontoblast processes [98]. This propensity leads to protein precipitation and the formation of a sealing film [99], potentially capable of restricting the transmission of external stimuli within the structure through a tubular occlusive mechanism.

6.2. In-Office Therapeutic Treatments of Dentin Hypersensitivity

Numerous products and methods have been suggested for addressing DH in dental practice, and these have been assessed through various studies, yielding divergent outcomes. While in-office treatments can provide immediate relief, long-term management of DH may require additional at-home measures. The agents employed within the dental practice for DH treatment are categorized based on their mode of action into two groups (Table 2):

a. Those that hinder nerve impulse transmission;
b. Those that occlude dentin tubules (Figure 2).

Table 2. The in-office therapeutic treatments for dentin hypersensitivity.

<table>
<thead>
<tr>
<th>Active Agents</th>
<th>Form</th>
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<th>Duration of Therapy</th>
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<tr>
<td>Potassium salts</td>
<td>Gel</td>
<td>Impeding nerve impulse transmission</td>
<td>2–3 times during a two-week period</td>
</tr>
<tr>
<td>Low-level laser irradiation (LLLT)</td>
<td>Irradiation</td>
<td>Impeding nerve impulse transmission</td>
<td>Case-sensitive</td>
</tr>
<tr>
<td>Sodium fluoride</td>
<td>Varnish or gel</td>
<td>Occlusion of dentin tubules</td>
<td>Once</td>
</tr>
<tr>
<td>Silver diamine fluoride (SDF)</td>
<td>Solution</td>
<td>Occlusion of dentin tubules</td>
<td>Once</td>
</tr>
<tr>
<td>Adhesive agents</td>
<td>Solution</td>
<td>Occlusion of dentin tubules</td>
<td>Once</td>
</tr>
<tr>
<td>Bioactive glass powder</td>
<td>Air abrasion</td>
<td>Occlusion of dentin tubules</td>
<td>Once</td>
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<tr>
<td>Portland cement</td>
<td>Paste</td>
<td>Occlusion of dentin tubules</td>
<td>Once</td>
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<tr>
<td>Oxalate salts</td>
<td>Gel or solution</td>
<td>Occlusion of dentin tubules</td>
<td>Once</td>
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<tr>
<td>Laser irradiation</td>
<td>Irradiation</td>
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<tr>
<th>Treatment Type</th>
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<tr>
<td>Sodium fluoride varnish</td>
<td>Varnish or gel</td>
<td>Occlusion of dentin tubules</td>
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<td></td>
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<tr>
<td>Silver diamine fluoride (SDF)</td>
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<tr>
<td>Bioactive glass powder</td>
<td>Air abrasion</td>
<td>Occlusion of dentin tubules</td>
<td>Once</td>
<td></td>
</tr>
<tr>
<td>Portland cement paste</td>
<td>Paste</td>
<td>Occlusion of dentin tubules</td>
<td>Once</td>
<td></td>
</tr>
<tr>
<td>Oxalate salts</td>
<td>Gel or solution</td>
<td>Occlusion of dentin tubules</td>
<td>Once</td>
<td></td>
</tr>
<tr>
<td>Laser irradiation</td>
<td>Irradiation</td>
<td>Occlusion of dentin tubules</td>
<td>Once</td>
<td></td>
</tr>
</tbody>
</table>

Figure 2. Occlusion of the orifices of the dentin tubules after treatments for dentin hypersensitivity. The rate of occlusion is measured by the reduction of the diameter of the tubules (%). (a) 0% reduction, (b) 50% reduction, and (c) 100% reduction of the diameter of the tubules.

a. Therapeutic agents that hinder the transmission of the nerve impulses

Potassium salts (most common KNO₃) and low-level laser therapies (LLLT) belong to this category.

6.2.1. Potassium Salts Gels

Potassium salts-containing products for in-office treatments are marketed mainly in the form of gels. In particular, potassium-based agents facilitate the elevation of potassium ion (K⁺) concentration in nerve endings, thereby diminishing the nerve’s capacity to transmit sensory stimuli and modifying its action potential [2,100]. When these salts are applied to the exposed dentin, they increase the concentration of K⁺ in the tubules and surrounding tissues, influencing the normal depolarization and repolarization processes of nerve cells [101]. More specifically, nerve cells rely on the movement of various ions, including Na⁺ and K⁺, to generate and transmit electrical signals, known as action potentials. When a nerve is stimulated, Na⁺ ions rush into the cell, causing depolarization, which is a change in the electrical potential of the cell membrane. This depolarization triggers the transmission of the nerve impulse along the nerve fiber. Potassium ions play a crucial role in repolarizing the nerve cell after the action potential has passed. They move out of the cell, helping to restore the cell’s resting electrical state. However, when potassium salts are present in high concentrations, as in the case of potassium-based agents used for treating DH, the movement of K⁺ becomes disrupted. The elevated concentration of K⁺ outside the nerve cells interferes with their ability to repolarize efficiently. This results in a dampened or altered response to sensory stimuli. Essentially, the nerve cell becomes less responsive to subsequent stimuli, reducing its capacity to transmit pain signals caused by external triggers like temperature changes or mechanical pressure. This mechanism contributes to the reduction of DH by impeding the transmission of nerve impulses associated with pain sensation [102].
6.2.2. Low-Level Laser Therapy

Low-level laser therapy (LLLT), also known as cold laser therapy or photobiomodulation therapy has been explored as a potential treatment for DH. Various laser wavelengths have been investigated for this purpose, with some of the commonly used wavelengths falling within the red (630–690 nm) and near-infrared (810–980 nm) spectrum. LLLT can engage with the dental pulp, resulting in increased odontoblastic activity, which leads to the production of tertiary dentin and a reduction in the lumen size of dentin tubules. Such lasers can also induce cell activity, trigger anti-inflammatory responses, and generate analgesic effects [103–105]. LLLT is dose-dependent; therefore, it is generally used in sequential appointments with a time interval. There are various clinical protocols that dental practitioners can follow. Most of the studies found that pain levels were significantly reduced 3 months after the therapies [106].

LLLT is believed to reduce pain during DH through various biological mechanisms. While the exact processes are still being studied, here are some proposed ways LLLT might work to alleviate pain in this context [107–109]:

1. Increased blood flow and microcirculation: LLLT has been shown to promote vasodilation and improve microcirculation in the treated area. This enhanced blood flow can facilitate the delivery of nutrients and oxygen to the tissues, aiding in their healing and reducing inflammation. Improved circulation can also help remove waste products and inflammatory mediators that contribute to pain.
2. Stimulation of cellular activity: LLLT is thought to stimulate cellular activity, including the activation of mitochondria, the cell’s energy-producing organelles. This increased cellular energy can accelerate tissue repair and regeneration, promoting healing and reducing pain.
3. Anti-inflammatory effects: LLLT is known to have anti-inflammatory effects by modulating immune responses and reducing the release of inflammatory cytokines. This can lead to a decrease in inflammation, which often accompanies pain in conditions like DH.
4. Neurological modulation: LLLT may influence nerve function and sensitivity. It could modulate nerve conduction and reduce the transmission of pain signals. Additionally, LLLT might affect nerve endings and receptors, altering their response to stimuli.
5. Endorphin release: LLLT might trigger the release of endorphins, which are natural pain-relieving chemicals produced by the body. Endorphins can help block pain signals and promote a sense of well-being.
6. Stimulation of dentin remineralization: LLLT might encourage the remineralization of dentin, potentially sealing or occluding the tubules and reducing their sensitivity.

It is important to note that the effectiveness of LLLT for DH can vary among individuals and might depend on factors such as the specific laser parameters used, the duration and frequency of treatment, and the underlying causes of hypersensitivity.

b. Therapeutic agents that occlude the dentin tubules

6.2.3. Sodium Fluoride

The impact of fluorine compounds on DH reduction has been extensively investigated [110–112]. Fluorine compounds lead to a reduction in calcium fluoride (CaF$_2$) crystal presence within dentin tubules, subsequently decreasing dentin permeability [113]. These crystals are almost insoluble in saliva. The 2% sodium fluoride (NaF) varnish commonly employed in dental practice induces a reduction in calcium salts; however, these are readily eliminated from the dentin surface by saliva and brushing. To address this challenge, an acidified formulation, namely acidulated phosphate fluoride (APF 1.23%, 12,300 ppm F, pH 3.2) is available as solution, gel, or thixotropic gel, and as foam, is employed to prompt the reduction of salts deep within dentin tubules [65]. If fluoroapatite forms after the treatment, it exhibits improved stability against the effects of saliva, brushing, and dietary substances [114]. Fluorine compounds can also be employed in conjunction with iontophoresis, a method that employs an electric current to enhance ion diffusion [4,115].
Products containing fluorine compounds for in-office use are applied to exposed dentin usually in varnish forms.

6.2.4. Silver Diamine Fluoride

Silver diamine fluoride (SDF) is a topical medicament used in dentistry to treat various dental issues, including DH. When SDF comes into contact with the tooth surface, it reacts with the hydroxyapatite in the dentin and enamel, forming silver fluoride (AgF) and silver chloride (AgCl) compounds. These compounds can occlude the tubules present in the dentin [116], preventing the external stimuli from reaching the nerve endings, thus reducing sensitivity [117]. It is important to note that while SDF can be highly effective in managing DH [118], it may cause temporary discoloration of the treated area. The darkening of the tooth surface occurs due to the formation of silver compounds and for this reason, it has been suggested to be accompanied by potassium iodide (KI) [119]. This potential aesthetic concern should be discussed with patients before using SDF (38%) as a treatment option. Consequently, SDF is usually considered for use in cases where conventional treatments for DH, such as desensitizing toothpaste or fluoride varnishes, have not provided adequate relief.

6.2.5. Adhesive Agents

In contrast to other agents that reduce DH, adhesive agents exert a prolonged or even permanent effect. When combined with resin composites, they create an occlusion of the dentin tubules, yielding a hybrid zone [68]. In the past, older adhesive agents formed this hybrid zone through the removal of the smear layer and etching of the dentin surface, allowing for deep resin penetration into the dentin tubules [120]. However, newer bonding agents alter the smear layer and integrate it into the hybrid zone. It is assumed that these modern adhesive agents can effectively address and act preventively against DH. An illustrative instance of an adhesive agent utilized to mitigate DH is Gluma (Heraeus, Hanau, Germany; Kulzer), which comprises hydroxyethyl methacrylate (HEMA, Amsterdam, The Netherlands), glutaraldehyde, and fluoride. Glutaraldehyde prompts protein coagulation within the dentin tubules, while HEMA penetrates the tubules and blocks them. Clinical studies have substantiated Gluma’s significant efficacy in DH reduction [121].

6.2.6. Air Abrasion with Bioactive Glass Particles

Bioactive glasses have demonstrated a significant role in promoting dentin remineralization [110,121]. Their principal constituent, silicon (Si), functions as a nucleation site for calcium and phosphorus ions. Scanning electron microscopy (SEM) studies have revealed that the application of this bioactive substance leads to the formation of an apatite layer, ultimately occluding dentin tubules [122,123].

Bioactive glasses represent specialized biomaterials comprised of glass that are notable for their tendency to react on their surfaces. When these glasses come into contact with the surface of teeth and interact with saliva in acidic conditions, they can dissolve. This dissolution releases calcium and phosphate ions while also raising the pH levels [124]. This series of actions set off a chain of chemical responses that result in the formation of a layer made up of hydroxycarbonate apatite [125]. This apatite layer shares similarities with the mineral hydroxyapatite, which is commonly found in dentin. Both substances can create a chemical bond, promoting a stronger attachment of the glass particles to the dentin surface [126]. It is important to note that the complete development of this apatite layer takes some time, often spanning multiple hours. This gradual formation of reparative dentin, characterized by its low permeability, hinders the diffusion of harmful substances from dentin tubules toward the dental pulp. As a result, it restricts the movement of dentin fluid, offering a long-lasting solution for tooth sensitivity [127].

A commonly used variety of bioactive glass in dentistry is known as Bioglass 45S5, which is a calcium sodium-phospho-silicate compound sold under the trade name NovaMin®. In general, glasses are amorphous solids lacking a defined crystal structure, primarily composed
of silica-based elements with minor additives. However, Bioglass 45S5 diverges from this standard by featuring lower levels of silica and higher concentrations of calcium and phosphorus compared to typical glasses [128]. A previous study demonstrated that using a powder of Bioglass 45S5 for air abrasion can effectively block the dentin tubules (>99%) [76].

6.2.7. Portland Cement

Portland cement, specifically a type called “white Portland cement”, has been explored as a potential treatment for DH. Similarly, to bioactive glasses, Portland cement, primarily comprised of Si, has shown substantial potential in treating DH. Its attributes facilitate the occlusion of dentin tubules through the remineralization of dentin [115]. White Portland cement has fine particles that can infiltrate and occlude these dentin tubules. It contains calcium silicates, which can release calcium and hydroxide ions when they come into contact with moisture, contributing to the remineralization of the dentin, and making the tooth structure stronger and more resilient [129]. It is important to note that while white Portland cement has been investigated as a treatment for DH, it is not a standard or widely accepted treatment in dentistry.

6.2.8. Oxalate Salts

Oxalates have been identified as agents capable of occluding dentin tubules, potentially leading to a reduction in dentinal permeability of up to 98% [130,131]. The application of 28% potassium oxalate can facilitate the formation of calcium oxalate within the dentin tubules. For enhanced efficacy, dentin etching can be employed in conjunction with oxalates [131]. Specifically, oxalate salts, such as potassium oxalate and ferric oxalate [132], are believed to work through a mechanism of precipitation and formation of crystals within the dentin tubules [133]. Oxalate salts have the ability to react with Ca\(^{+2}\) present in the dentin structure. Indeed, when applied to the exposed dentin surface, these salts can react with the Ca\(^{+2}\) in the tubules and surrounding dentin, leading to the formation of insoluble calcium oxalate crystals. The formation of calcium oxalate crystals within the dentin tubules creates physical barriers, which can partially or completely block the dentin tubules [132,133]. It is important for patients to be aware that oxalate treatments might cause tooth discoloration or lead to the formation of insoluble deposits on the teeth. As such, the potential aesthetic concerns associated with these treatments should be discussed with patients before their use.

6.2.9. Laser Irradiation Treatments

According to the results of various studies, the effectiveness of the laser in the treatment of DH varies significantly and ranges between 5–100% [134–136]. Patients often experience immediate relief from DH after laser treatment. The results depend on the wavelength of the laser and the setting parameters, such as application time, mode of irradiation (pulsed or continuous), and average power [137–139].

Various laser wavelengths have been utilized for the reduction of the DH symptoms. More specifically, diodes (810–980 nm) [139–141], neodymium: yttrium–aluminum–garnet—Nd:YAG (1064 nm) [142,143], erbium: yttrium–aluminum–garnet—Er:YAG (2940 nm) [138,144], erbium, chromium: yttrium–scandium–gallium–garnet—Er,Cr:YSGG (2780 nm) [76,145], and carbon dioxide—CO\(_2\) (10,600 nm) [146,147] lasers have been employed against DH.

The mechanism of action of lasers has not been definitively determined, but multiple theories exist regarding the effects of laser exposure on dentin. The most widely accepted one suggests that it involves the occlusion or sealing of dentin tubules through a process of dentin melting and subsequent recrystallization [148]. When addressing tooth sensitivity, it is crucial for the laser beam to avoid ablation of the treated dentin surface and instead induce only structural and chemical transformations. Ablation of dentin can lead to a surface that lacks a smear layer, has open dentin tubules, and exhibits uneven textures, which is an undesirable outcome for the procedure [149]. Furthermore, using laser settings exceeding 0.75 W may result in the formation of charred areas, making such protocols
unsuitable for addressing DH [150]. There have been suggestions that employing lower power settings might initiate the dissipation of dentinal fluid. This, in turn, could lead to a reduction in dentin permeability, ultimately resulting in a decrease in dentinal pain [148]. Representative SEM images of the rate of occlusion after various treatments for DH are presented in Figure 3 [73]. It is important to mention that DH cannot lead to necrosis of the pulp like it could in cases of extensive tooth restorations [151].

Figure 3. Representative SEM photomicrographs showing the rate of occlusion of the dentin tubules after various treatments for dentin hypersensitivity (×3000 magnification). (a). Untreated dentin; (b). SnF$_2$ treatment; (c). CPP-ACP treatment; (d). calcium phospho-fluoro-silicate glass treatment; (e): Bioglass 45S5 treatment; and (f): Er,Cr:YSGG laser treatment [73].

7. Conclusions

Although numerous methods have been suggested for addressing DH, a definitive solution to the problem remains elusive. The outcomes derived from clinical studies assessing the efficacy of diverse treatment approaches for DH often yield conflicting results. One plausible explanation for this disparity is that assessing objective pain intensity proves challenging due to the inherently subjective and intricate nature of pain. Hence, the effective therapeutic management of DH should rest upon accurate diagnosis alongside robust measurement and evaluation. The development of new materials and techniques (at-home and in-office) to treat DH has led to the possibility of alleviating the symptoms in many clinical cases. This literature review focuses on the most important contemporary information on this topic to help dental clinicians stay aware of the latest developments in the treatment of DH.

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