8 Englische Zusammenfassung

The penetration and tightness of various adhesives into initial enamel lesions in vitro.

The aim of the present study was to evaluate the penetration depth (PD), the thickness of the oxygen inhibition layer (OIL) and the tightness of a fissure sealant (Helioseal®, Vivadent) and various adhesives (Heliobond®, Excite®, Vivadent; Resulcin®, Merz; Solobond M®, Voco; Prompt L-Pop®; 3M-Espe) applied to enamel lesions in vitro. From 27 bovine teeth 54 enamel specimens were prepared and covered with nail varnish (control), thus obtaining three windows for treatment. After demineralisation (pH 5.0, 14 d) two of the windows were etched with phosphoric acid (20%, 5 s), whereas the third area served as control. The specimens were divided randomly into six groups (n=9) and the respective adhesive was applied (90 s) either once or twice. Light-curing followed after each application. Half of the area of each specimen window was then covered with nail varnish and the samples were stored in the demineralising solution again (pH 5.0, 14 d). The specimens were cut perpendicular to the surface. Enamel slabs were studied after infiltration with a fluorescent low viscous resin using confocal microscopy (CLSM). The image of the lesion was divided into two areas with different grey values. Lesion depths were calculated (ImageJ) from the surface to that point in the lesion where the grey value clearly changed to a darker grey value. The zone with the darker grey values marked the front of demineralisation. Mean lesion depths (SD) after demineralisation (14 d) were measured at 105 (21) µm. After single application, Resulcin® [89 (22) %] and Helioseal® [98 (6) %] had almost completely penetrated the lesion. Heliobond® [126 (33) %] and Excite® [184 (40) %] penetrated even deeper than the defined lesion. For Excite® double application decreased the OIL significantly (p=0.03; adjusted paired t-test).

After the second demineralisation the untreated lesions showed a mean progression of 52 (31) %. Helioseal, Heliobond, Resulcin Monobond and Excite were able to inhibit lesion progression completely (p<0.05; t-test). These materials were capable to prevent from a further demineralisation when applied once and there was no difference between the lesion depths after one or two applications (p<0.05; t-test) (fig. 5). In contrast, the adhesives Adper Prompt L-Pop and Solobond M were not able to inhibit further lesion progression when applied once (p>0.05; t-test). After twice application of Solobond M a significantly decreased lesion progression (p<0.05; t-test) could be observed. The other materials proved to be significantly better in hampering further lesion progression (p<0.05; ANOVA, post hoc Bonferroni).
The development of such treatment strategies could offer potential noninvasive means of treating early enamel lesions. Adhesives are capable to penetrate artificial initial enamel lesions completely.