## Effect of cold atmospheric plasma treatment on dental pulp in rat molars

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Cold atmospheric plasma treatment of hard tooth substances has shown in previous investigations to disinfect by killing of adherent bacteria [1], to efficiently clean Ti surfaces from biofilms [2] and to improve tooth-filling interaction by surface modification [3].

The aim of this study was the investigation of the influence of cold plasma treatment on the rat dental pulp in combination with adhesive filling therapy.

Occlusal cavities were prepared in first upper molars of 20 Sprague-Dawley rats (1 x 1.5 x 0.5 mm, remaining dentin: 0-0.3 mm, diamond bur ISO 008, 6.000 U/min, air/water spray cooling). In a split mouth design, one prepared rat molar and the adjacent unprepared molar were treated with cold atmospheric plasma (pulsed microwave 2.45 GHz, mean power 3 W, plasma jet with Gaussian profile: 8 mm length 1.5 mm FWHM, 2.0 slm He, treatment time 5 s per tooth, surface temperature: maximum 40 °C). The prepared cavities were filled with a self-conditioning adhesive and flow composite material. After 24 h and 28 d ten rats each were sacrificed and the upper molar segments were dissected. Teeth were demineralized by 10 % EDTA for 4 weeks and embedded in paraffin. Histological sections (6  $\mu$ m) were prepared and stained with haematoxylin-eosin (HE) and chlorazetatesterase (CAE). The odontoblast layer, appearance of inflammatory cells, necrosis, pre- and secondary dentin formation were assessed in the histological sections.

Distinct inflammation was detected in pulps of teeth treated with plasma and filling as well as in pulps of teeth which were only filled after 24 h. After 28 d in both groups secondary dentin formation and reduced inflammation were observed. Pulps of teeth treated with cold plasma but not filled did not show any increased inflammation compared to untreated controls neither after 24 h nor after 28 d.

In this animal experiment the treatment of vital teeth with cold atmospheric plasma did not result in an increased inflammation of pulp.

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## References

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