hydrofluoric acid (HF) the decontamination of skin should focus on the inactivation of free fluoride ions. The present *ex-vivo* study investigated the effects of exposure duration and different antidotes on the potential systemic uptake of fluoride.

Methods The transdermal penetration of HF (c=30%) through excised human skin was investigated by using static diffusion cells. After dermal application of the acid (100 µl/0.64 cm²) for 1 min the excess was removed using one dry cotton swab. Subsequently, the skin was cleaned with water, calcium gluconate (CaGI), polyethylene glycol (PEG) 400 or hexafluorine[®] using a standardised protocol. In a further study, the application time was extended to 3 min to assess the effect of exposure duration. Chemical analyses of fluoride were carried out by GC-MS or via a fluoride-sensitive electrode.

Result Extension of the exposure time from 1 to 3 min led to an enhancement in the transdermal penetration of fluoride, however with similar penetration kinetics. At the end of experiments (6 hour) a 7-fold higher fluoride amount was detected in the receptor fluid (16 vs 114 µg). In all test series maximum flux was achieved within the first hour past exposure. Decontamination of the skin reduced the cumulative penetrated amount of fluoride by 28% (PEG 400), 49% (water) and 64% (CaGI/hexafluorine[®]) compared to control.

Discussion The results indicate that the systemic uptake of fluoride ions and therefore possible systemic intoxication after exposure to hydrofluoric acid can be diminished by shortening the exposure duration. Reduction was further increased by decontamination of skin – most by substances which are known to supply the complexation of fluoride ions.

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DERMAL ABSORPTION OF FLUORIDE AND HYDROGEN IONS FOLLOWING TOPICAL EXPOSURE TO HYDROFLUORIC ACID

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Introduction Despite being an extremely hazardous liquid, hydrofluoric acid (HF) is commonly used in industry due to its unique chemical properties. Chemically HF is defined as a 'weak' acid but fluoride ions can induce serious systemic toxic effects. Upon contact with low concentrated HF, symptoms such as pain or local lesions may be delayed.

The aim of this study was to characterise changes in intradermal pH and dermal fluoride penetration following HF application.

Methods A static diffusion cell model was used to study dermal fluoride penetration for 6–72 hour following application of varying amounts of HF (c=5%–50%, 100–160 µl/0.64cm², 1–10 min.) on human skin (thickness 0.9 or 2.5 mm). Intra- and transdermal amounts of fluoride and intradermal pH were determined.

Result Transdermal penetration of fluoride increased exponentially with increasing HF concentration. In addition, penetration increased four-times by extending the exposure time from 1 to 3 min. No further increase was seen with longer HF application (5 and 10 min.). The increased amount of HF penetrated through 0.9 mm compared to 2.5 mm skin within one hour was levelled out at later time points. Intradermal

accumulation of fluoride increased dose-dependently but to a lower degree. Intradermal pH dropped with increasing HF concentration and exposure time. Additionally, the lag time between HF application and onset of pH changes decreased with increasing HF concentration and application time.

Discussion The results of the present study show that following 3 min. HF application maximal amounts of fluorides seem to have penetrated the skin. The longer lag time in pH drop with lower concentrated HF might explain the delay between HF contact and onset of pain.

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TOXICOLOGICAL EFFECTS OF REPETITIVE EXPOSURE TO MIG-WELDING FUME PARTICLES ON RAT PRECISION-CUT LUNG SLICES

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Introduction The proinflammatory effects of metal inert gas brazing (MIG) welding fumes containing zinc and copper have been demonstrated in humans. However, little is known about the specific effects on the lung. Here we examined the effects of different concentrations of welding fumes for repetitive exposure in rat precision-cut lung slices (PCLS).

Methods PCLS were prepared from agarose-filled lungs of male rats. To mimic a five day 'work week', PCLS were incubated in welding fume containing media with 1 and 0.1 µg/ml in a repetitive exposure model for 6 hours on 5 consecutive days. For the remaining 18 hours PCLS received incubation in standard incubation medium. For each day cytotoxicity was determined via WST-1 and LDH assay. To determine the maximal LDH release possible, PCLS were treated with Triton X-100 as a positive control.

Result Over all days of consecutive treatment no significant reductions of mitochondrial activity determined via WST-1 could be found in comparison to untreated controls. LDH levels in supernatants increased up to 15% of levels of positive controls treated with Triton X-100, indicating no relevant toxicity.

Discussion This is the first time repetitive toxicological effects of welding fumes on the lung have been examined in isolated lung tissue with intact microanatomy. We demonstrate that a repeated exposure for up to five days has no relevant toxic effects on lung tissue in doses comparable to a realistic occupational exposure. Lung tissue slices could be a promising model to study toxicity of welding particles and need to be investigated further.

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MINERAL OIL IS A RISK FOR AUTOANTIBODIES INDUCTION

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Introduction Mineral oil(hydrocarbon)is also used in many factories, and workers are exposed to a lot kind of mineral oil. It was reported that one element mineral oil named pristane induced inflammatory arthritis in rats and also induced lupus-