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Evaluation of emergency skin decontamination protocols in response to an acid attack (vitreolage)

H. Matar, P.R. Vuddanda, R.P. Chilcott*

Research Centre for Topical Drug Delivery and Toxicology, University of Hertfordshire, Hatfield AL10 9AB, UK.

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ABSTRACT

The incidence of "acid attacks" (vitreolage) is a global concern, with those affected often receiving lifelong medical care due to physical and psychological damage. The purpose of this study was to evaluate the effectiveness of several emergency skin decontamination approaches against concentrated (> 99 %) sulphuric acid and to identify the effective window of opportunity for decontamination. The effects of four decontamination methods (dry, wet, combined dry & wet and cotton cloth) were assessed using an in vitro diffusion cell system containing dermatomed porcine skin. Sulphuric acid (H_2SO_4) was applied to the skin with decontamination protocols performed at 10 s, 30 s, 8 min, and 30 min post exposure. Skin damage was quantified by tritiated water (^3H_2O) penetration, receptor fluid pH and photometric stereo imaging (PSI), with quantification of residual sulphur (by SEM-EDS) to determine overall decontamination efficiency. Skin translucency (quantified by PSI) demonstrated a time-dependent loss of dermal tissue integrity from 10 s. Quantification of dermal sulphur content confirmed the rapid (exponential) decrease in decontamination efficiency with time. The pH of the water effluent indicated complete neutralisation of acid from the skin surface after 90 s of irrigation. Wet decontamination (either alone or immediately following dry decontamination) was the most effective intervention evaluated, although no decontamination technique was statistically effective after 30 s exposure to the acid. These data demonstrate the time-critical consequences of dermal exposure to concentrated sulphuric acid: we find no practical window of opportunity for acid decontamination, as physical damage is virtually instantaneous.

1. Introduction

The term "acid attack" (or 'vitreolage') is used to describe attacks with corrosive chemicals (including alkalis). Over the last 10 years, the prevalence of such attacks in the UK has increased [12,19,8]. The consequences of using corrosive substances may leave victims with lifelong disfigurement and significant impacts on both physical and mental well-being [3,12]. Following exposure to alkaline chemicals such as sodium hydroxide will typically result in liquefactive necrosis due to the saponification of fatty acids by hydroxyl ions [23]. This damage tends to manifest deeper into tissues compared to acid exposures. In contrast, acids result in the precipitation of proteins due to hydrogen ions [H+] leading to coagulative necrosis [23]. Several factors influence the severity of injuries such as concentration of the substance,

* Correspondence to: Toxicology Research Group, University of Hertfordshire, Hatfield AL10 9AB, UK.
E-mail address: tox.publications@herts.ac.uk (H. Matar).

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route and duration of exposure [22]. Corrosive materials indiscriminately damage most biological tissues and are widely used in both domestic and occupational environments; therefore, accidents are generally the leading cause of corrosive exposures [4,14].

It is evident from a clinical perspective that persons in acid attacks will receive ongoing medical care and in severe cases, multiple skin grafts [1,15,26]. The medical treatment of acid attack victims is typically irrigation with water in the first instance. Irrigation is typically performed to protocols based on perceived best practice [2,15]. Typically, patients are washed, not bathed as to limit the spread of damage. Patients are rapidly administered potent analgesics to manage the pain [13,16,21] and the burns managed by continual irrigation and in some instances, neutralisers such as diphoterine [11]. However, the extent of damage may potentially be mitigated by first responders by performing immediate decontamination.

Recently, the UK has published guidance to the public on how to respond to acid and chemical burns [20]. This involves three key steps, 1) report: contact emergency services and request urgent help, 2) carefully remove the chemical and affected clothing and 3) rinse continuously with clean water taking care not to rub or wipe the area. This approach is simple and promotes early decontamination which can subsequently reduce the extent of tissue damage. However, this campaign is not common knowledge and more should be done to promote its message as burn care begins on scene [25]. In contrast, some view this message as outdated following the availability of specific decontamination solutions such as Diphoterine® [12]. As with all specialist decontamination products, availability is a limiting factor, particularly during the initial stages of exposure.

Whilst specific decontamination solutions for vitriolage are carried by some first responders in the UK [25], they are unlikely to be available immediately following exposure due to the finite delay in responding. Conversely, within an occupational setting, access to such countermeasures is likely to be more readily available. It is widely reported in the literature that the use of water and other commercially available neutralising products can reduce the extent of damage. However, we note a dearth of studies involving dry decontamination of corrosives, such as the use of absorbent paper towels or clean, uncontaminated clothing.

The aims of this study were to (1) evaluate different emergency skin decontamination methods (likely to be immediately accessible to victims) against concentrated sulphuric acid, (2) identify an effective decontamination window and (3) assess a range of different aqueous-based materials to ascertain their potential exothermic effect on skin temperature.

2. Materials and methods

2.1. Chemicals and decontamination materials

Sulphuric acid (99.9 %) was purchased from Sigma Aldrich (St. Louis, USA). Sodium chloride (99.5 +%) was purchased from Fisher Scientific (Leicestershire, UK). Ultra-pure water (> 18.2 MΩ) for receptor fluid media was obtained by ultrafiltration of the municipal supply via a MilliQ Integral 3 system (Millipore, MA, USA). Soluene®– 350, Ultima gold and tritiated water (^3H_2O; 3.7 MBq mL^{-1}) were purchased from Perkin Elmer (Cambridgeshire, UK). Tritiated water was diluted to a nominal activity of 3.7 kBq μL^{-1} by the addition of an appropriate volume of non-radioactive water. “Blue Roll” absorbent paper (Wypall™, Kimberley Clark, UK) was purchased from Fisher Scientific (Loughborough, UK). Swatches of clothing were taken from black cotton t-shirts (100 % cotton, 190 g cm^{-2}, average thickness 810 µm, Vend Fabrics Ltd, Leicester, UK). Sodium bicarbonate solution (1 M) was purchased from Fisher Scientific (Leicestershire, UK). Pasteurised, semi-skimmed milk was purchased fresh from a local grocery store.

2.2. Skin samples

Full-thickness skin was obtained *post-mortem* from four female pigs (*Sus scrofa*, large white strain, weight range 15–25 kg) purchased from the Royal Veterinary College following approval by the local Animal Welfare and Ethical Review Board. The skin was close clipped and excised from the dorsal aspect from each animal. The skin was then wrapped in aluminium foil and stored flat at – 20 °C for 4 months before use. Prior to the start of each experiment, a skin sample from a single donor was removed from cold storage and thawed in a refrigerator (4 °C) for approximately 24 h. The skin was then dermatomed to a nominal depth of 500 µm using a Humeca Model D80, (Eurosurgical Ltd., Guildford, UK) and the thickness of the resulting skin section confirmed using a digital micrometer gauge (Tooled-Up, Middlesex, UK). Once dermatomed, the skin was then cut into squares (approx. 3 × 3 cm) in preparation for mounting on to diffusion cells.

2.3. Diffusion cells

Static, horizontal, jacketed (Franz-type) skin diffusion cells [7] were purchased from PermeGear (Chicago, Illinois, USA). Each diffusion cell comprises an upper (donor) and lower (receptor) chamber, with an area available for diffusion of 1.77 cm^2. Dermatomed skin sections were placed between the two chambers, with the epidermal surface facing the donor chamber and the ensemble securely clamped. The receptor chamber was filled with ~14 mL 0.9 % saline. A barrier integrity test of each excised skin sample was performed using an inductance capacitance and resistivity meter (LCR model 821, ISO-TECH, UK). This involved placing 1 mL of 0.9 % saline on the skin surface (donor chamber). Resistivity was measured across the skin with the LCR electrodes. Skin with a resistivity < 2x that of skin intentionally damaged (with a single needle puncture) was excluded from the study. Saline in the donor was then gently removed using a cotton swab and the diffusion cell connected to a manifold. Each diffusion cell was placed in a Perspex™ holder above a magnetic stirrer which constantly mixed the receptor fluid via (12 × 6 mm) Teflon™-coated iron bar placed within the receptor chamber. The receptor chambers were jacketed to enable the flow of warm (35 °C) water from a circulating water heater (Model TC120), Grant instruments, Cambridge, UK) via the manifold

Table 1 – Summary of treatment groups. A total of n = 6 diffusion cells were used per treatment group per exposure duration (10 s, 30 s, 8 min or 30 min).

Treatment Group		Parameters
NC	Negative control	No acid contamination, no decontamination.
PC	Positive control	Contaminated with acid, no decontamination.
DD	Dry decontamination	Acid followed by decontamination using weighted 3-ply absorbent paper ('blue roll') applied for a total of 10 s
WD	Wet decontamination	Acid followed by 90 second showering with water (21° C; 7.2 mL min^{-1}).
DWD	Dry + wet decontamination	Acid followed by DD performed as described above immediately followed by WD.
CLD	Clothing layer decontamination	Acid followed by decontamination with a layer of 100 % cotton t-shirt material according to DD protocol.

to ensure a constant skin temperature of 32 °C (confirmed by infrared thermography; FLIR Model P620 camera, Cambridge, UK). Once assembled, the diffusion cells were left in situ for an equilibration period of up to 24 h following which a baseline receptor fluid sample was withdrawn (250 µL).

2.4. *Treatment groups*

A total of four experiments were performed: one per acid exposure duration (see below). Each experiment used thirty-six diffusion cells divided into six treatment groups (n = 6 per group) as outlined in Table 1. Each experiment was initiated by the addition of a 20 µL droplet of concentrated sulphuric acid to the skin surface of each diffusion cell (except negative controls).

2.5. *Decontamination*

Where applicable, decontamination was conducted at 10 s, 30 s, 8 min (480 s) or 30 min (1800 s) post exposure.

Dry decontamination (DD) was performed by applying a Section (2 × 2 cm^2) of blue roll to the skin surface with a 14.23 g metal weight (~8 g cm^{-2}) placed on top. After five seconds, the weight was removed, and the blue roll turned over. An aluminium foil disc (1.72 cm^2) was placed on top of the blue roll to prevent contamination of the weight which was reapplied for a further 5 s before the ensemble was removed, giving a total decontamination duration of 10 s

The protocol for wet decontamination (WD) involved showering the skin surfaces with water at a temperature of 21 °C delivered via a peristaltic pump (Watson-Marlow 520S0, at a flow rate 7.2 mL min^{-1} for 90 s using a bespoke (3D-printed) shower adaptor clipped onto the donor chamber of each diffusion cell. Shower effluent was collected in 2 mL glass vials every 15 s for subsequent pH analysis.

A combined dry and wet decontamination process (DWD) was performed as described above, with wet decontamination being performed immediately after dry decontamination.

Clothing layer decontamination (CLD) was performed as described for DD but using a layer of 100 % cotton t-shirt material (185 gsm) cut to 2 × 2 cm.

2.6. *Measurement of skin barrier function*

Two minutes after decontamination, all skin surfaces were gently rubbed with a cotton swab for 15 s prior to the addition of 100 µL ^3H_2O. After 20 min, the unabsorbed water was removed using a cotton swab and the receptor fluid decanted into a pre-weighed 20 mL vial. The skin was removed and placed in a small polystyrene petri dish for subsequent photometric stereo imaging after which it was dissolved by immersion in glass vials containing 10 mL soluene-350.

The radioactivity within the receptor fluid, skin and cotton swabs was quantified using a PerkinElmer Tri-Carb liquid scintillation counter (Model 2810 TR) employing an analysis runtime of 2 min per samples and a pre-set quench curve specific to the brand of LSC fluid (Ultima Gold™, PerkinElmer, UK). The amounts of radioactivity in each sample were converted to quantities of ^3H_2O by comparison to standards (measured simultaneously). The standards were prepared on the day of each experiment by the addition of a known amount of ^3H_2O to cotton wool swabs in 10 mL water or 10 mL of solubilised skin tissue. A standard receptor fluid solution was also prepared by the addition of 10 µL of ^3H_2O to 990 µL of fresh receptor fluid (0.9 % saline), from which a range of triplicate samples (25, 50, 75 and 100 µL) were placed into vials containing 5 mL of LSC fluid to produce a standard (calibration) curve. Aliquots (250 µL) of each sample (receptor fluid, cotton wool swab and skin) were taken and placed into vials containing 5 mL of LSC fluid for liquid scintillation counting.

2.7. *Image analysis*

Skin samples were prepared by excising the area available for absorption from the excess tissue by careful excision with a scalpel. The prepared sections of skin were imaged using a device capable of photometric stereo imaging and CIELAB colour quantification L*a*b* measurements (C-Cube, Pixience, France). However, as this was non-viable tissue, the skin was mounted on a bespoke 3D-printed tissue mount produced with red polylactic acid thermoplastic in order to interpret the a* parameter (typically used for measuring erythema) as a surrogate for skin damage based upon translucency. Each skin sample was imaged using 2D and 3D imaging to evaluate the overall integrity of each skin section over a range of parameters (elevation, surface roughness wrinkles and the bespoke surrogate for skin translucency). The resulting digital images were analysed using ImageJ (National Institute of Health, v1.52a) to quantify the area of skin damage. Each image was converted to a calibrated binary 8-bit image and an area of interest specified corresponding to the area of the skin to determine the area of viable skin in the image. This was then subtracted from the average area of viable skin from the non-exposed controls.

2.8. Liquid decontamination temperature studies

Sections of dermatomed (500 µm) porcine skin (3 × 3 cm) were placed onto a plastic weigh boat (to limit reflections which may interfere with infrared thermography). Infrared images were captured every 15 s using a FLIR P620 camera. A total of five treatment groups were assessed (each comprising n = 3). Groups consisted of a non-exposed (negative control), exposed (positive control, contaminated but not decontaminated) and decontaminated groups comprising either water, 1 M bicarbonate solution or milk. Each skin surface was baselined over 30 s before application of a 20 µL droplet of concentrated sulphuric acid (except for the negative control). At the appropriate time point (30 s) post exposure, 200 µL of each decontamination solution was applied to the acid droplet and the temperature captured for a further 2.5 min. Each image was analysed by FLIR tools software (version 5).

2.9. Scanning electron microscopy with energy dispersive spectroscopy (SEM-EDS)

The sulphur content within skin sections was quantified using a SEM (Hitachi TM4000Plus Mk II, UK) coupled with an EDS (Aztec One Xplore EDS; Oxford Instruments, UK). Sections of dermatomed porcine skin (3 ×3 cm; 500 µm thick) were sandwiched between chambers of static diffusion cells. The skin samples were subjected to either NE, E, DD, WD, DD +WD and C at either 10 s, 30 s, 480 s or 1800 s post exposure to a 20 µL droplet of sulphuric acid. The decontamination approach was similar to the main study. However, each treatment group comprised a total of $n = 3$ replicates. Immediately following the decontamination (where applicable), the post-experiment skin samples were sectioned into approximately 2 (width) x 10 mm (length) and placed onto a SEM specimen holder (with the cross section facing upwards). Each cross-section sample was observed using an accelerating voltage of 15 kV using a Back-Scattered Electron (BSE) detector under medium vacuum conditions. All samples were observed at 100 x magnification. The sulphur content in each sample was mapped using the EDS to detect relative levels of sulphur content on the image (atomic %). Decontamination efficiency, based on sulphur content, was calculated using Eq. 1.

$$\%E = 100 - [((Q_d - Q_{ne})/(Q_e - Q_{ne})) \times 100]... \quad (1)$$

Where %E is the percentage efficiency and Q is the atomic percentage of sulphur measured in skin which has been decontaminated (d), non-exposed (ne) or exposed (e).

2.10. Statistical analysis

Statistical analysis was performed using GraphPad Prism 7.03. Normality tests (Shapiro-Wilk) were performed on all data (where possible). Comparisons of % area damaged was performed by a two-way ANOVA with Dunnett's multiple comparisons test which compared each treatment group to the positive control group.

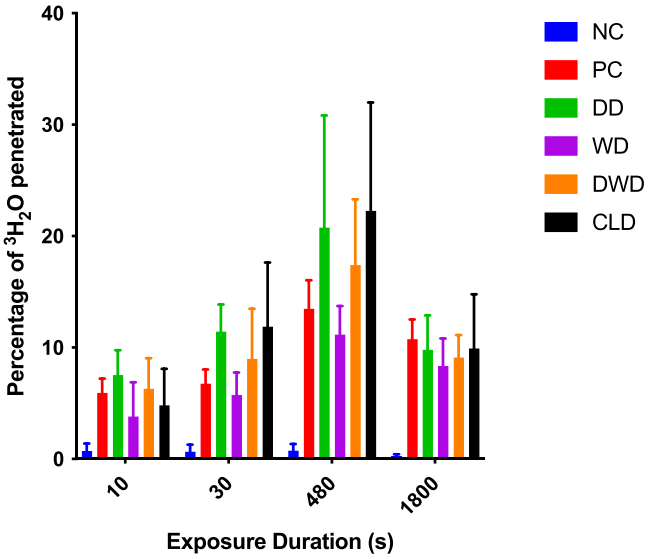

Fig. 1 – Percentage of applied ^3H_2O recovered from receptor fluid of control and treated diffusion cells, expressed as a function of acid exposure duration. A total of six treatment groups (Table 1) were evaluated; negative controls (NC), positive controls (PC), or following dry decontamination (DD), wet decontamination (WD), combined dry and wet decontamination (DWD), or clothing layer decontamination (CLD). All points are mean ± standard deviation of n = 6 diffusion cells.

3. Results

3.1. Tritiated water penetration

Exposure to acid in the absence of treatments (positive control; PC) caused a statistically significant ($p < 0.05$) increase in permeability to tritiated water when compared to unexposed skin (negative control; NC) at all exposure durations (Fig. 1). Skin permeability following all decontamination treatments (DD, WD, WDW and CLD) was not statistically different to the PC group at any exposure time (Fig. 1), indicating that damage to skin barrier function was present from 10 s and was not influenced by any of the decontamination methods.

3.2. pH measurements

The receptor fluid pH of non-exposed (NC) and exposed (PC) control groups were significantly different ($p < 0.05$) at all exposure times, being 6.8 ± 0.5 and 2.43 ± 0.2, respectively; Fig. 2). Wet (WD), dry and wet (DWD) and cloth decontamination (CLD) resulted in a significant ($p < 0.05$) improvement in receptor chamber pH when performed 10 s post exposure. However, delaying decontamination from 30 s onwards negated this effect (Fig. 2).

The pH of water effluent collected from wet (WD) and combined dry and wet (DWD) decontamination demonstrated a time-dependent increase which appeared to plateau

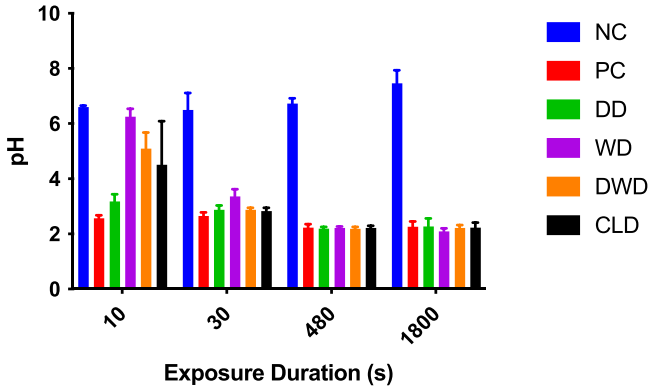

Fig. 2 – Average pH of receptor fluid following a 20 µL droplet of concentrated sulphuric acid (99.9 % H_2SO_4) applied to the skin. Decontamination was performed at 10, 30, 480 or 1800 s post exposure. A total of six treatment groups (Table 1) were evaluated; negative controls (NC), positive controls (PC), or following dry decontamination (DD), wet decontamination (WD), combined dry and wet decontamination (DWD), or clothing layer decontamination (CLD). All points are mean ± standard deviation of n = 6 diffusion cells.

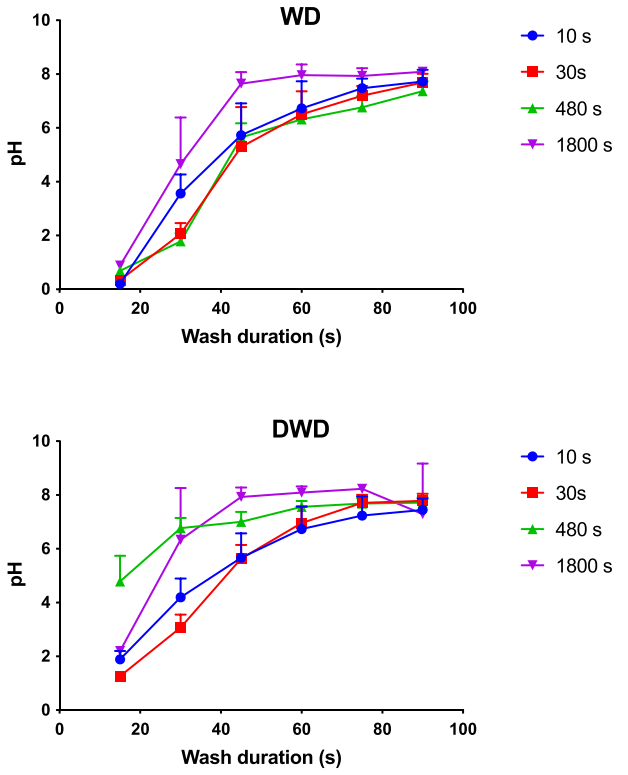

Fig. 3 – Average pH of shower effluent (expressed as duration of washing) collected from wet decontamination only (WD) and combined dry and wet decontamination group (DWD). Wet decontamination was performed using a water shower delivered at a temperature of 21 °C and a flow rate of 7.2 mL min cm^{-2}. Decontamination was performed 10, 30, 480 or 1800 s post exposure with a total shower duration of 90 s. Shower effluent was collected every 15 s. All points are mean ± standard deviation of n = 6 diffusion cells.

by 90 s and was largely independent of the exposure duration (Fig. 3). The only discernible difference between WD and DWD treatments was the initial pH of the effluent, which was generally higher when preceded by dry decontamination.

3.3. *Imaging*

We found no statistically significant differences in the extent of skin surface damage between decontaminated and positive control groups (Fig. 4). Interestingly, quantification of skin translucency using the "erythema" (CIELAB; a* parameter) demonstrated a time-dependent effect for all treatment groups exposed to the acid (Fig. 5), with translucency increasing with longer durations of acid exposure.

3.4. *Exothermic reaction*

The greatest increase in skin surface temperature was observed for the exposed only (PC) group, which reached 40 °C at 90 s (Fig. 6). Each of the test solutions significantly ($p < 0.05$) reduced the rise in temperate from 90 s. We found no statistically significant differences between the test solutions.

3.5. *SEM-EDS analysis*

The atomic percentage of sulphur detected in the skin of negative controls (unexposed, non-decontaminated) was consistently ~ 0.6 ± 0.3 %. In contrast, the sulphur content of positive controls (exposed, non-decontaminated) was roughly an order of magnitude greater (~ 6.1 ± 1.1 %). In terms of decontamination efficiency (%E; Eq. 1), we found a time-dependent decrease in performance for DD, WD and D+W, but not the cloth treatment groups (Fig. 7). Wet (WD) and combined dry and wet (D+W) decontamination were consistently the most effective treatments. In contrast, dry and cloth decontamination were approximately half as effective in reducing sulphur content and were both relatively ineffectual after an exposure duration of 8 min or longer (Fig. 7).

4. Discussion

This study investigated the effects of concentrated sulphuric acid on excised porcine skin. The purpose of this study was to evaluate different decontamination procedures to alleviate these corrosive effects. Therefore, this study focussed on practical measures a contaminated individual could take to reduce damage prior to the arrival of medical staff. A series of exposure durations were explored to assess decontamination efficacy with time. Additionally, this study determined the minimal washing duration to remove surface contamination.

The data obtained in this study clearly illustrates that damage inflicted by exposure to concentrated H_2SO_4 is virtually instantaneous. All decontamination protocols that involved purely dry decontamination did not reduce the

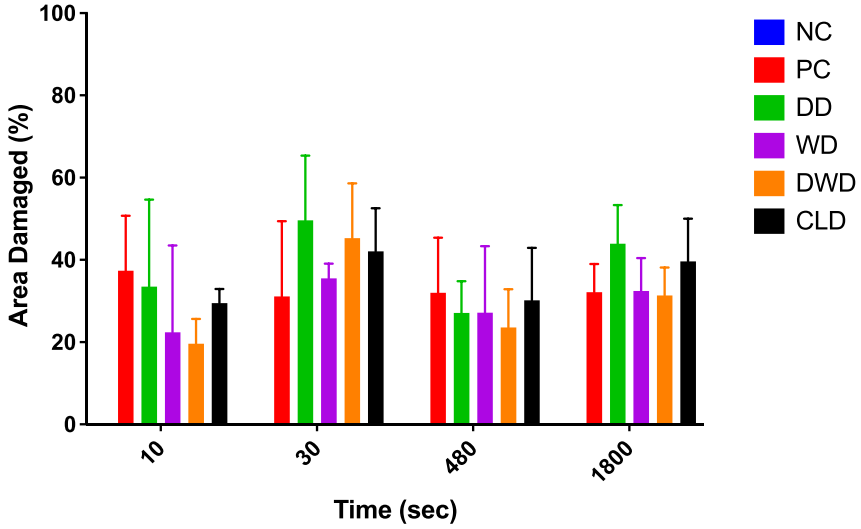

Fig. 4 – Average area of damaged skin expressed as a percentage of the total area available (1.77 cm^2) for each of the respective treatment groups and exposure periods. A 20 µL droplet of concentrated sulphuric acid (99.9 % H_2SO_4) was applied to the skin. Decontamination was performed at 10, 30, 480- and 1800-seconds post exposure. A total of six treatment groups (Table 1) were evaluated; negative controls (NC), positive controls (PC), or following dry decontamination (DD), wet decontamination (WD), combined dry and wet decontamination (DWD), or clothing layer decontamination (CLD). All points are mean ± standard deviation of up to n = 6 diffusion cells.

amount of ^3H_2O penetrating the skin, nor the pH of the receptor fluid compared to the exposed control. This is likely attributable to the rapid destruction of the stratum corneum on contact with concentrated sulphuric acid [6].

Our data suggest that decontamination procedures which include aqueous irrigation may be slightly more effective than those based on dry decontamination. However, frank damage to skin barrier function (measured directly by tritiated water penetration and indirectly through translucency measurements) confirmed that aqueous decontamination was also relatively ineffective after an exposure period of 30 s or more. The only parameter which clearly demonstrated the superiority of aqueous-based decontamination was in the reduction in skin sulphur content (SEM-EDS).

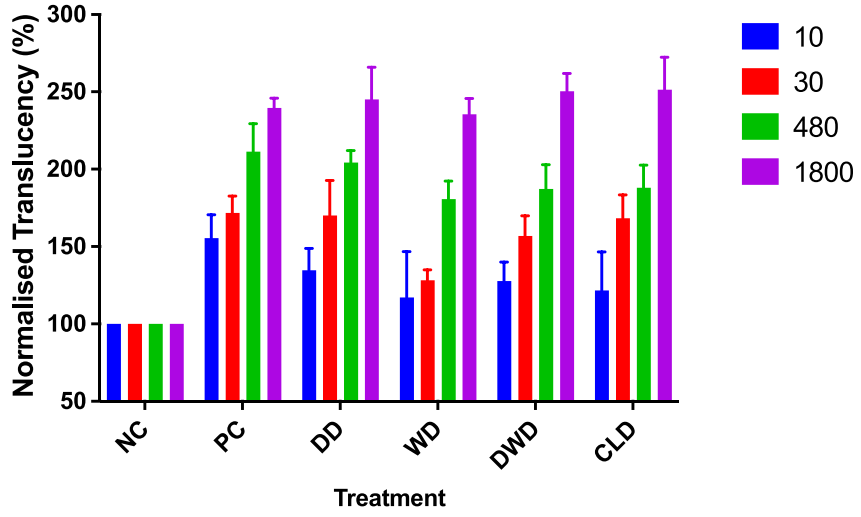

Fig. 5 – Normalised translucency of skin, quantified by photometric stereo imaging (PSI; expressed as percentage of negative controls) following exposure to concentrated sulphuric acid. Decontamination was performed at 10, 30, 480- and 1800-seconds post exposure. A total of six treatment groups (Table 1) were evaluated; negative controls (NC), positive controls (PC), or following dry decontamination (DD), wet decontamination (WD), combined dry and wet decontamination (DWD), or clothing layer decontamination (CLD). All points are mean ± standard deviation of n = 6 diffusion cells.

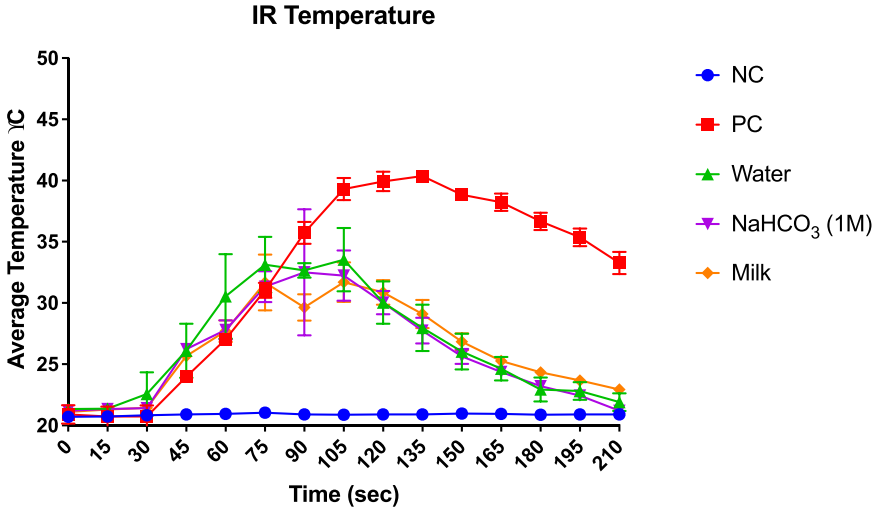

Fig. 6 – Average surface temperature of skin as measured by infrared thermography for negative control (NC), positive control (PC) or skin decontaminated with water, $NaHCO_3$ or semi-skimmed milk. Each point represents mean ± SD of n = 3 replicates.

The time-dependent effectiveness of skin decontamination in this study agrees with previous case reports and epidemiological evidence which reported that the sooner water irrigation was performed the less severe the resulting chemical burn [10,13,20].

In terms of skin irrigation, the minimum wash duration was found to be between 45–60 s to restore pH of the effluent. This was slightly faster when dry decontamination was performed prior to wet decontamination. Complete removal of the acid was achieved by irrigation under all experimental conditions by 90 s. This is most likely due to less contaminant being available at the start of irrigation (as it has been removed at the dry decontamination stage). This observation applies specifically to the dose of acid and flow rate of water

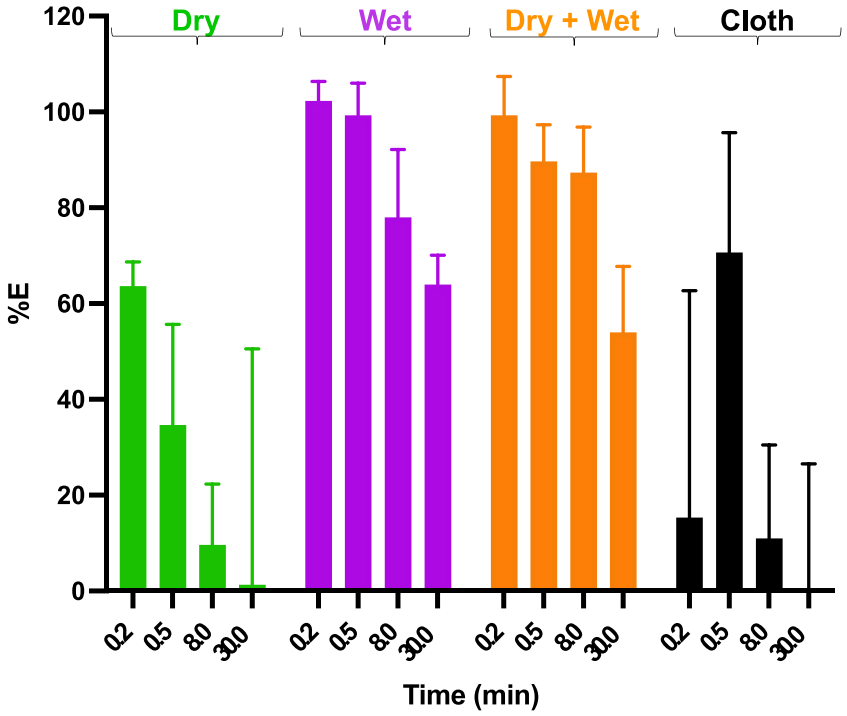

Fig. 7 – Percentage decontamination efficiency (%E; Eq. 1) of dry, wet, dry followed by wet, or clothing layer treatments (calculated from SEM-XDS sulphur content). All values are average ± standard deviation of n = 3 replicates.

used in this study. Further studies are indicated to determine if this duration of irrigation can be extrapolated to different contamination densities.

An additional facet of this study was the quantification of skin temperature following the addition of different decontamination solutions to the skin surface. This was performed to quantify the exothermic reaction of these solutions with the acid and thus identify any potential to exacerbate a chemical burn. All test solutions (water, milk and bicarbonate) resulted in a significant decrease in skin surface temperature. The addition of water to a strong acid typically results in an exothermic reaction and so care has been advised when irrigating contaminated skin [9]. It is conceivable that the excess volume of the test solutions (in combination with removal of the acid) used in this study was sufficient to supress this effect.

The use of translucency measurements in this study represents a novel approach to quantifying dermal tissue damage following acid exposure: a clear dose-response was observed in that translucency increased in proportional to the duration of acid exposure (Fig. 5). A meta-analysis indicated that translucency most closely correlated with receptor fluid pH (r^2 = 0.9468; Fig. 8). Thus, optical changes in the dermal tissue were strongly associated with the extent to which the acid penetrated the skin.

We note several limitations that should be considered when interpreting these experimental results. Firstly, as these studies were performed using excised porcine skin in vitro, tissue damage associated with subsequent inflammation or other pathological sequelae will be absent in this model. Furthermore, measures of healing or "clinical" benefit of decontamination cannot be reproduced or ascertained in this model. The use of porcine skin, whilst morphologically similar to human skin, may also react differently to human skin as it is slightly less vascularised compared to human skin [17,24]. This lower vascularity may be due to the fact that the papillary plexus is deeper in pigs than humans [18]. The area of porcine skin (dorsal aspect) used in this study has been reported to being the closest with regards to thickness, hair follicle density and composition to human skin [27]. In addition, this model does not address any systemic manifestations such as metabolic acidosis [5]. Another potential limitation is that this study used only one contamination density (~ 6 mg cm^{-2}) and so extrapolation of these data to other exposure scenarios indicates further investigation. Taking these limitations into account, our data still support the principle of rapid irrigation with copious amounts of water as a first aid measure either alone, or immediately following dry decontamination.

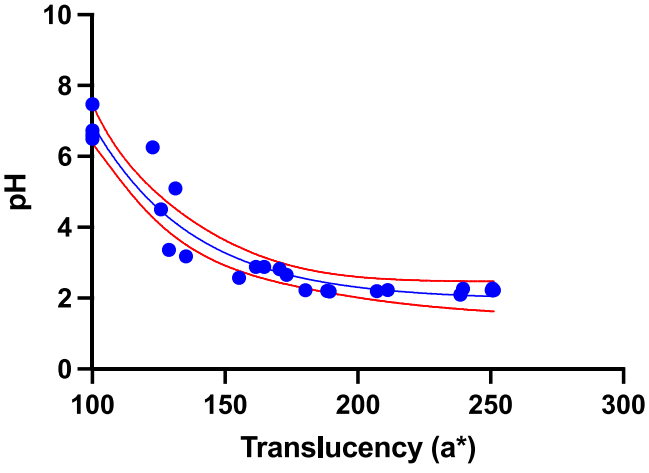

Fig. 8 – Skin translucency (quantified as the CIELAB a* parameter) as a function of receptor fluid pH. Dotted line indicates 95 % confidence intervals of non-linear regression analysis (based on one phase exponential decay). Data collated from all treatment groups (n = 24).

5. Conclusion

This study demonstrates the difficulty in mitigating skin damage following exposure to concentrated sulphuric acid. We found practically no window of opportunity for acid decontamination as damage is virtually instantaneous. Dry decontamination methods which involve rubbing or wiping the skin appears to be contraindicated for acid exposures. This study can also conclude that the minimum duration to limit skin damage is 45 s. However, this may also be subject to amount of acid in contact with the skin.

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CRediT authorship contribution statement

Matar H: conceptualization, methodology, formal analysis, investigation, data curation, writing – original draft, visualization. **Vuddanda PR**: methodology, formal analysis, investigation, data curation, writing – original draft. **Chilcott RP:** conceptualization, methodology, formal analysis, investigation, data curation, writing – review & editing, visualization.

Declaration of Competing Interest

The authors declare no conflicts of interest.

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