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## **Declining allergic contact dermatitis from tosylamide formaldehyde resin in nail polish**

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Running title: Allergic Contact Dermatitis from TSFR

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Nail polish was first launched in 1919, initially consisting of only collodion mixed with ether and ethanol (1). In 1939, tosylamide formaldehyde resin (TSFR), also known as toluenesulfonamide formaldehyde resin, was introduced into polishes to enhance their brilliance, adhesion and durability (2,3). Subsequently, TSFR was found to be an important trigger for allergic contact dermatitis (ACD) often affecting the eyelids and neck (4). TSFR has been a significant cause of ACD for several decades, resulting in its inclusion in patch test baseline series in several countries (2,5). Nowadays TSFR-free nail polishes, sometimes with the inclusion of different allergens such as acrylates, formaldehyde and polyester resins, are widely used (6,7). We were interested to explore the changing pattern of both contact sensitisation and ACD to TSFR.

## **Methods**

A retrospective review of all patients tested with TSFR over a 16-year period (January 2002 to December 2017) at the Skin and Cancer Foundation, Victoria, Australia was conducted. Patients had attended either occupational or non-occupational contact dermatitis clinics. TSFR (10% in petrolatum) had been included in our baseline series prior to being incorporated into the Australian baseline series, which was implemented in 2013. Patch testing was performed according to ICDRG guidelines. Positive reactions were classified as relevant to the presenting dermatitis, or of old or unknown relevance.

## **Results**

A total of 7408 patients were patch tested for TSFR over the 16-year period. Among these, 83 patients (1.1%) developed reactions, of which 58 (0.78% of overall) were positive. Twenty had a reaction of unknown relevance and 5 had a reaction of old relevance. All positive TSFR reactions were related to nail polish use. There has been a noticeable reduction in the number of reactions to TSFR over the last 16 years ( $P < .001$ ) based on statistical assessment of a linear trend. The highest prevalence of contact sensitisation to TSFR occurred in 2005 and the highest proportion of ACD occurred in 2003, see Table 1.

## **Discussion**

Nail polishes nowadays comprise multiple components including primary film formers, secondary film formers, plasticisers, solvents and colorants (8). Secondary film formers are ingredients that enhance the durability and adhesion of primary film formers, which are polymers that provide a hard and glossy film for nail lacquer (8,9). Secondary film formers are commonly resins, and TSFR used to be one of the main secondary film formers (8,9).

Other examples of secondary film formers include polyester resins (e.g. terpolymer of 2,2,4-trimethyl-1,3-pentadienol, isophthalic acid and trimellitic anhydride) and cellulose acetate butyrate (8,9). In non-TSFR nail polish, also known as “anti-allergic” or “hypoallergenic” products, TSFR is substituted by some of the latter and other secondary film formers (2,8).

TSFR has been traditionally recognised as the cause of nail polish ACD, and a study published in 1993 revealed 6.6% positive reactions to TSFR amongst women with nail polish associated ACD (10). However, our data demonstrates a statistically significant decline in

reactions to TSFR over the last 16 years. This may be attributed to the increased use of non-TSFR nail polish. In addition, other nail polish allergens have also been identified. For example, there has been a rise in ACD to acrylic nail products, such as shellac nails, porcelain nails and gel nails following increasing popularity of these products (7). Other hardening resins, such as glycerophthalic polyester, 4-methylbenzene sulfonamide-epoxy and phthalic polyester have also been reported to cause nail polish associated ACD (2).

In summary, clinicians need to be aware of changes in nail cosmetics, which appear to have caused the decline of ACD to TSFR, at least in our patient population. Although the prevalence of ACD to TSFR is declining, it may still be considered to test TSFR in baseline series given that it has not been completely withdrawn from commercial use.

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Table 1 Patch test results of patients tested with TSFR from 2002 to 2017

<b>Year</b>	<b>Number of patients tested for TSFR</b>	<b>Total Reactions (%)</b>	<b>Relevant Reactions (%)</b>	<b>Reactions of unknown relevance (%)</b>	<b>Reactions of old relevance (%)</b>
2002	514	7 (1.4%)	6 (1.2%)	1 (0.2%)	0 (0%)
2003	520	9 (1.7%)	7 (1.3%)	2 (0.4%)	0 (0%)
2004	505	7 (1.4%)	4 (0.8%)	3 (0.6%)	0 (0%)
2005	527	10 (1.9%)	6 (1.1%)	3 (0.6%)	1 (0.2%)
2006	602	10 (1.7%)	8 (1.3%)	2 (0.3%)	0 (0%)
2007	495	6 (1.2%)	6 (1.2%)	0 (0%)	0 (0%)
2008	457	5 (1.1%)	4 (0.9%)	0 (0%)	1 (0.2%)
2009	485	5 (1%)	2 (0.4%)	3 (0.6%)	0 (0%)
2010	497	2 (0.4%)	2 (0.4%)	0 (0%)	0 (0%)
2011	442	5 (1.1%)	3 (0.7%)	2 (0.5%)	0 (0%)
2012	452	6 (1.3%)	6 (1.3%)	0 (0%)	0 (0%)
2013	370	3 (0.8%)	1 (0.3%)	1 (0.3%)	1 (0.3%)
2014	430	3 (0.7%)	0 (0%)	1 (0.2%)	2 (0.5%)
2015	389	3 (0.8%)	2 (0.5%)	1 (0.3%)	0 (0%)

2016	359	2 (0.6%)	1 (0.3%)	1 (0.3%)	0 (0%)
2017	364	0 (0%)	0 (0%)	0 (0%)	0 (0%)



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