INDUSTRIAL DERMATITIS

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INDUSTRIAL dermatitis is officially described by the Ministry of National Insurance as P.D. 42 "a non-infective dermatitis of external origin, including chrome ulceration of the skin but excluding dermatitis due to ionization particles of electromagnetic radiations other than radiant heat." This has replaced "24b," dermatitis produced by dust, liquid or vapour.

It could also be described as the eczematous reaction of the skin to a hurtful agent or environmental factor which irritates it or to which it is sensitive (allergic).

Accurate information is not easy to obtain but in the years 1953-1956 in Great Britain industrial dermatitis accounted for 0.3% of 1.3% total lost working days for all skin diseases in a working population of 20,500,000 (Thompson, 1958); Bourne (1960) gives similar figures, 0.32% of 1.6% skin diseases.

Eczematous reactions accepted under P.D. 42 are, however, not solely the contact result (dermatitis) of an external irritant. Many are really eczema determined, often initially by external irritants, but predisposed thereto by a constitution inferiority of the skin and inherited or acquired immuno-chemical relationships. These will both influence the onset and the course of eczema, on a personal basis.

Behind these considerations lie most of the difficulties relative to diagnosis of occupational dermatitis.

Basically the development of industrial dermatitis represents a struggle between an external aggressor (mechanical, chemical or biological) at work, and the defending epidermal defence with its varying anatomical, architectural, molecular or physiological integrity.

Rapid overwhelming of the defence leads to acute contact dermatitis whilst more insidious undermining predisposes to eczema.

Patterns of Dermatitis (P.D. 42).

Eczematous reactions which may be accepted under P.D. 42 or considered for aggravation by work may be designated —

- (a) Contact dermatitis (contact pattern).
- (b) Exogenous "industrial" eczema (eczematous dermatitis), (patchy eczema pattern).

(c) Constitutional eczema (temporarily aggravated by occupation) (constitutional patterns).

Fungal infections of the feet, epidermophytosis, in colliers may be accepted as an "industrial accident" if there is evidence of pithead baths being used.

In true contact dermatitis all exposed areas are affected usually within hours or 2-3 days of contact and recovery should occur in weeks to a few months when such contact ceases.

Patchy "industrial" eczema (eczematous dermatitis) usually presents in a patchy or discoid pattern on the work-exposed skin but usually only after months or years of exposure, its course being chronic for months, years or sometimes permanently. Much patchy eczema is also initiated, however, by other exogenous factors (home or hobbies) acting alone or in concert with those at work.

Allergic sensitisation can occur in both patterns (contact and patchy eczema) but is preceded first by some irritant or traumatic damage to the epidermal protective structures allowing percutaneous penetration of allergens.

The constitutional patterns of eczema which may be temporarily aggravated by work or environment include dysidrotic (pompholyx), atopic (eczema-asthma-hayfever syndrome), seborrhoeic eczemas and neurodermatitis. Such skins are themselves constitutionally unstable and often susceptible to exogenic injury.

Although theoretically a true contact dermatitis and also an "industrial" eczema should recover, soon or eventually, when away from contact, many cases of accepted "P.D.42" become chronic; contact dermatitis developing later into a patchy eczema, and patchy eczema becoming chronic or progressing into constitutional patterns. Factors which determine this include constitutional predisposition itself, continued exposure to irritants or sensitisers at work or in the home, secondary pyogenic infection, adverse effects of topical or systemic treatment, and persisting functional disturbances such as vascular instability and permeability, sweating and keratinisation as sequels of the dermatitis even when the convalescent skin appears clinically well. Emotional factors including neuroses (neurodermatitis), compensation, litigation, resettlement problems or frank malingerers (dermatitis artifacta) will also contribute.

Epidermal Defence

Percutaneous chemical penetration, with irritant action on deeper viable epidermal cells, or allergic-induced sensitisation through the dermal reticulo-endothelial system, is normally prevented by the intact keratinised stratum corneum. An ionic layer (water barrier) of cells, described by Szakall, (1955) below the stratum being electrically charged, repels anions and reattracts cations, and is also repellent to water and water soluble electrolytes. Fat-soluble chemicals may pass this barrier once it is damaged or may pass through the appendageal sebaceous glands. Entrance through sweat ducts is minimal although silver can enter thus to cause argyria (Buckley, 1963).

Later penetration through viable malpighian and basal cells is trans or intercellular in method.

In addition, there exists other "built-in" defences as the neutralising buffer capacity of amphoteric horny keratin, and the "acid mantle" of shed stratum corneum horn cells, acidic sweat and fatty acids. Sebum also lessens water evaporation protecting keratin.

As the stratified horn layer constitutes the main defence, this in turn relates to an intact gross architecture (cell-to-cell adhesion and dermosome bonding including that of other epidermal and epidemo-dermal junction levels) but also at a cellular level based upon the molecular structure of keratin.

Integrity of Keratin in Stratum Corneum

The fibrous protein keratin filling the firmly packed denucleated cells of the stratum corneum has been progressively synthesised by addition of amino-acid polymerisation to polypeptide chains in the viable basal cells and intermediate keratinocytes.

A final cross linkage of adjacent polypeptide grids by strong disulphide ($S = S$) bonds of cystine, or weaker salt (acid or basic) and electrically charged hydrogen atoms of amino acids converts the grids into the rigid horny keratin and hardens dermosome cell adhesion. The keratin molecules themselves add mechanical strength being twisted in helical form like ropes.

Water content

The 10 - 20% of water in the stratum corneum, replaced by diffusion from below, both plumps the individual keratin molecules and acts as a lubricant between the polymer chains, similar to batching emulsions used in spinning textiles, ropes or glass fibre. This reduces fibre to fibre friction, imparting suppleness and abrasion proofing.

Dessication by climatic, environmental or chemical agencies at work causes dryness and fissuring (chapping) and increased liability to mechanical stress and encourages chemical penetration.

Under conditions of work therefore the stratum corneum and deeper layers may be damaged primarily by physical wounding or mechanical stress with individual cell separation or protein damage, by reduction of water content below 10% and at a cellular and molecular level, by chemical damage to protoplasm, to keratin and its cross links.

Occupational insult may also chemically damage enzymes responsible for cellular metabolism and protein synthesis; bullous eruptions may thus develop. For instance, sulphhydryl (-SH) containing enzymes are susceptible to chemicals as methyl bromide, organic mercurials (seed dressings), arsenicals (Lewisite) or nitrogen mustards producing contact vesicant dermatitis whilst other vesicants act by blocking conversion and interfering with cellular respiratory metabolism relative to adenosine triphosphate (ATP) (Burbach, 1964).

During day-to-day exposures constant repair of minor injuries or chemical damage occurs in the stratum corneum unless the result be at once overwhelming. While contact dermatitis is mainly determined by the concentration or allergenicity of the chemical or the degree of exposure, "eczematous dermatitis" occurs mostly in those with abnormally keratinised or non-intact skins in which normal repair less easily occurs.

Among these constitutional inferior skins featuring dyskeratoses, ichthyosis, xerosis, atopic persons, the seborrhoeic state and those with poor water-holding capacity (subject to winter chapping) or buffering capacity. Thinning of the stratum corneum, inherited or acquired by primary collagen degeneration in senile or aging skins also operates. Over use of topical steroids in treatment may itself lead to collagenous degeneration with thinning and transparency and lessened resistance.

Aggressive Industrial Agents

Injurious agents at work (or in the home) may be physical, environmental, chemical or biological.

Chemicals are conveniently classified as irritants or sensitisers, their potential for causing contact dermatitis or eczema is determined by chemical reactivity, concentration, degree and conditions of exposure, and by the constitutional resistance of the exposed skin.

Physical agents

Puncture wounds and lacerations with secondary infection may lead to local traumatic dermatitis or initiate eczema. Topical treatment is also a common cause. These cases are seen particularly in heavy industry, for instance 35% of "P.D.42" in the steel industry is so accountable (Vickers, 1958).

An occupational traumatic dermatitis should commence at the site of injury before complete

healing has occurred. The skin, in the vicinity of recently healed abrasion, is however temporarily more susceptible (non-specific hypersensitivity) to other accepted irritants during the convalescent phase until hardening occurs.

Continuous friction may increase the risk of penetration by smoothing and thinning the horny layer as in pottery workers, paper mills and metal polishing, as can also constant handling of rough materials as rusty metal concrete or bricks.

Keratin chains are stressed or the actual architectural cementing loosened as when friction blisters form from distortion of cells and acantholysis (Naylor, 1955).

Combinations of friction and lacerative injury with infection however are most often eczematogenic. For instance, clothing or footwear dirtied with abrasive materials (coal miners or builders), gloves stiffened by tar, or when synthetic glues cause the fingers to adhere together. Abrasive hard metal dusts as tantalum or zinc, metallic engineering swarf in wipe rags adherent through oil, angular particles of sugar or crumbs in confectionery works, or harsh chrysotile asbestos fibres used for insulation (amphiloie fibres for textiles are soft and flexible) are other examples. Glass fibre causes pruritic, erythematous or follicular traumatic dermatitis and the risk may extend through contaminated clothing to those at home.

Hardening of synthetic glues on the skin causes stripping of keratin with irritant effect and predisposes to entrance of some sensitisers as formaldehyde (woodworkers) and epoxy resins.

Persistent friction however also often causes a callus with localised thickening and hardening of the horny layer; this gives considerable added mechanical protection to injury at work. If there is a disturbance of normal cell shedding with hardening and compression of the keratin lamellae (Rubin, 1949) calluses occur to cause occupational stigmata; they may also indicate diagnostically areas of skin most exposed to irritation at work.

Friction dermatitis will present with an initial redness, scaling, pigmentation or bullous formation at the site affected. Later a patchy dry or moist eczema may develop which on the forearms, thighs and legs may simulate a constitutional discord or nummular eczema. Such industrial eczemas may affect the stokers, boilermen, dustmen, metal workers, labourers, colliers and others in hot or dusty work. Secondary lichenification may occur from continued friction or from habit scratching (neurodermatitis) but however can give some protection and thus in time lessen incapacity.

Inferior skins intolerant of friction include those prone to winter chapping, dry or atopic skins with spicules of projecting keratin, hirsute persons and those with pilomotor instability, the hairs becoming

more prominent through contraction of erector pili muscles.

Environmental factors and sweat disturbances

Keratin dehydration with chapping occurs from variations in barometric pressure and the dew point or from desiccation agents.

Outdoor workers in agriculture and fishing, those working in cold as ice cream or refrigeration men, or those in contact with solvents or alkali are particularly liable to eczematogenic desiccation.

Asteatotic winter chapping may often precede occupational eczema, and inadequate washing facilities or "paid ablution time" and wet towels or hand-cleansing solvents can contribute to this tendency.

Constant climatic exposure in outdoor workers, clayey soil drying on the skin of agricultural workers, heat in furnace workers and those in metal production, smelting foundries and glass work also dries the skin as can such hygroscopic chemicals as salt (fishermen) sugar (confectionery), and lime or cement dusts in the building trade.

On the other hand a constant exposure to water and wet materials, electrolytes and emulsions may soften the keratin and, by maceration, increase penetration of larger chemical molecules; thus develop the initial patchy eczemas, with later allergic sensitisations, of housewives, cleaners, textile scourers (aggravated by alkaline keratin degradation), catering and confectionery workers, tanneries, paper pulp workers, and those in medical and allied trades.

The added degree of keratin damage related to minor irritants at home in all these cases always presents considerable diagnostic difficulties.

Disturbances of sweating. Sweat pore obstruction may occur from hydration oedema as in maceration from sweating or wet work, or by poral hyperkeratosis from micro-frictional trauma. Thus develop sweat obstruction syndromes of the body (miliaria) or hands (dyssidrotic pompholyx eczema.)

Miliaria may precede eczema in body crevices or on areas exposed to the friction of clothing. This occurs in furnace men and soldiers in tropical climates where heat exhaustion and thermogenic anhidrosis may also develop with disturbed temperature regulation. Miliarial eczemas also occur in colliers (papular eczemas of groins, axillae, cap band, belt and lamp battery areas), glass workers (sweat rags on neck), steel and other metal smelting, and in those working in overwarm or humid atmospheres required by a particular chemical process or where ventilation is inadequate, and when impervious protective clothing must be continuously worn. Obesity and alcoholism will contribute in many instances.

Excessive sweating can localise frictional or contact dermatitis from noxious dusts in body creases, increase skin staining from dyes and woods, or cause metal rusting ("rusters"), and by keratin softening increase sensitisation.

Constitutional eczema (dysidrosis) may be initiated or aggravated in those with hyperhidrosis where the handling of toxic chemicals requires long wearing of impervious plastic (P.V.C.) or rubber gloves and footwear. Some convalescent from dermatitis may also find protective gloves difficult to wear.

Sweat obstruction caused by imperfect keratinisation and poral closure in convalescent dermatitis or eczema subjects is a common cause of relapse on their return to work.

Chemical irritants and sensitisers

Chemical irritants are graded as strong to moderate or weak in strength and action on the skin.

Strong to moderate chemicals will normally cause lesions varying from contact chemical burns with erythema, vesication, bullae or ulcers to a classical contact dermatitis developing within hours or a few days of the exposure.

Weak chemicals usually predispose to patchy eczema by slow disturbance of stratum corneum repair or its molecular protein structure.

Secondary allergic sensitisation may complicate either.

Most industrial dermatitis is initiated by chemical irritants or trauma (80%), allergic sensitisation only being responsible for some 20% of cases.

Strong irritants and vesicants. Injury from strong chemicals is only likely to occur as an accident through ignorance, or if normal safety precautions and protective clothing be neglected. Processmen, fitters or other maintenance workers can however have such accidental exposure from pipe breaks.

Burns or dermatitis might occur from contact on unprotected skin with such materials as liquid oxygen or hydrogen peroxide, concentrated inorganic acids and alkalis, chromic acid and chromates (electroplating), hydrofluoric acid and fluorides (glass etching, synthetic resin polymerisation and uranium manufacture), silico-fluorides, organic acids as phenols or cresols (manufacture of phenolic resins), selenious acid (electrical industry), acetic acid and anhydrides (cellulose acetate manufacture and rocket propellant fuels), silanes and chloro silanes (silicone resin manufacture), the chlorinated benzenes (chemical intermediates) and catechols of cashew nut resins, shoe adhesives, copy paper, shellac and vegetation.

Some however through abraded skin cause ulcerations and leave scars which may act as distinctive marks of occupation. Among these are

chromic acid and chromates and cyanides (electroplating), mercuric nitrate and metallic sodium, ammonium nitrate and mercury fulminate (explosives), zinc chloride "Bakers fluid" (engineering fluxes), unslaked lime (building trade). Ulceration of the nasal septum may be similarly caused.

Among other common vesicants are acrylonitrile (vinyl cyanide) for acrylonitrile polymers and synthetic rubber, alkyl mustard compounds (synthetic resins or flavours), chloroacrylates (acrylic resins), dinitrophenols (chemical industry), iodoacetic acid (reagent for biochemical tests) and the alkyl organic tin (plasticisers for vinyl resins) and organic mercury compounds (disinfectant agents and agricultural seed dressings).

Vesicants very often induce sensitisation.

Moderate irritants. These may cause contact dermatitis on unprotected skin or after unusual exposure. They include miscellaneous polyamines, as ethylene amines which are often alkaline (used in chemical synthesis or as hardeners for synthetic resin adhesives), hydrazines (engineering fluxes), fluoacetic acid and acetates (agricultural herbage control), methyl salicylate (oil of wintergreen) and methylene chloride (paint remover).

Weak irritants. Transient erythema, or even contact dermatitis patterns may be produced by this group in constitutionally inferior skins but most often the adverse effects are dehydration with harsh dry scaling and fissuring or persisting erythemas as in "housewives hands," grading over into patchy eczema with probable added allergic sensitisation.

These weak eczematogenic irritants are found in a large variety of occupations. They all predispose to irritation of viable cells or to sensitisation by desiccating, damaging the keratin architecture or cellular protein, or by loosening molecular links and by increasing percutaneous penetration. For instance, alkalis damage protein by reducing the disulphide link ($S = S$) freeing sulphhydryl groups ($-SH$), weak acids dissociate salt links and may dehydrate; oxidisers as perchlorates and perborates affect hydrogen bonds and chlorine compounds as chlorinated lime (bleaching powder) or bisulphites, as reducers, also affect the strong disulphide link. The over use of these latter as stain removers may be eczematogenic. Textile, ink and dye workers are mainly at risk.

Solvent or alkaline agents which remove sebum expose the keratin to dehydration and bacterial infections by removal of the "acid mantle."

Among other weak irritants which usually cause patchy eczema are engineering cutting oils especially "suds" emulsion oils. There are assessed as causative of 15-20% of cases of industrial dermatitis. Synthetic anionic detergents and alkaline "built" washing agents are accepted as eczematogenic. Although occasional acute irritant effects are seen

from some concentrated anionic detergents as moderate irritants, the bad reputation of detergents may be unjustified for as Suskind and Whitehouse (1963) have shown by immersion tests, that housewives eczema, allegedly due to detergents, can still support daily immersion in weak solutions without ill effect.

Solvents vary in their action, most aggressive being petroleum hydrocarbons (petrol, naphtha, white spirit, kerosene), coal tar hydrocarbons (benzene and toluene), chlorinated hydrocarbons (carbon tetrachloride for rubber, paint, dry cleaning and fire extinguishers), and trichlorethylene for metal degreasing. True turpentine is both irritant and sensitising.

Ketones are usually eczematogenic after long exposure and may attack hydrogen bonds; methyl ethyl ketone used in synthetic resin adhesives has caused eczema when used to remove adherent epoxy or polyester glues from the skin. Alcohols are less irritant causing usually a temporary whitening of the skin; the volatile esters and ethers provoking mainly mucous membrane irritation.

The use of petroleum solvents, white spirit or "turps" substitute (decalin and tetralin) by painters and printers for cleaning machinery or stained hands is often a cause of eczema.

Harsh abrasives or alkaline "built" soaps and over use of strong solvent-alkali containing hand or waterless cleansers and over-washing of the hands by those with obsessional traits may be similarly provocative.

Biological irritants

Banal infections with monilia are relatively common in industry, particularly in the catering trade where maceration of the skin and sugary solutions predispose to *candida albicans* growth. Dentists may develop similar infections of the fingers from patients' mouths; occasionally these may become eczematized.

Less obvious however are miscellaneous zoonotic mite infections which may cause urticarial, papulo-vesicular and sometimes frank dermatitis. Dock workers (prunes, figs), millers and grocers (cereal mites), tobacco workers, poultry men including railway personnel (pigeons) are so prone.

The Allergic Sensitisation Process

Allergic sensitisation is of the delayed eczematous type induced by contact and percutaneous penetration. The minimal contact time for sensitisation is some 6-8 hours, and the incubation time before clinical evidence 6-10 days or shorter. Contact with the antigen should cause the delayed eczematous response in 12-48 hours or within a few days. Allergic sensitisation to industrial chemicals is usually specific to one agent but polyvalent sensitisa-

tion may occur although this is often postulated on the grounds of multiple reactions which may be irritant effects of patch testing. Group- or cross-sensitisation is where substances of apparently dissimilar chemistry have a common immune chemical group or molecular nucleus determining group reactions. This was first described by Mayer (1928) in respect of the "para group." Many other examples have been since described including:—

List of Common Group Sensitisers

Antibiotics (penicillin, tetracyclines, "mycins")
 Arsenicals (inorganic, organic)
 Bisphenols (epoxy resins and phenols)
 Catechols (copying inks, shoes and resins, Anacardiaceae trees)
 Chloro-cresols and xylenols (preservatives)
 Essential oils (essences, balsams)
 Esters — Para-aminobenzoic acid esters ("Parabens") (ointment and cosmetic preservatives)
 Paraphenylamine diamine.
 "Para" group — Aniline compounds (dye stuffs), amino-azo dyes, nitrophenols (picric and trinitrotoluene explosives), sulphamides, sapamine (brilliantines Paschoud 1963).
 Hydrazines (engineering fluxes, rocket fuels, pharmaceuticals)
 Hydroxyquinolines (topical dermatological therapy)
 Phenothiazines (pharmaceutical)
 Thioglycollic acid and salts (cold wave hairdressers)
 Vegetation (tars, terpenes, essential oils).

Other examples are continually reported indicating that allergic sensitisation may begin at home or in pursuits outside of work. Iatrogenic sensitisation established by drugs may for instance cause cross sensitisation reactions in industry. Apresoline (hydrazinophthazine), phenyl hydrazine and isonicotinic acid hydrazine (INAH) may pave the way for hydrazine salts used as metal soldering fluxes to cause sensitisation dermatitis (Frost and Hjorth 1959).

Industrial chemicals which induce photosensitive dermatitis include the germicides tetrachlorosalicyl anilides (which provoked an epidemic of contact dermatitis in a toilet soap), bithionol, and hexachlorophene used in cosmetics and soaps, or in pharmaceutical products.

Other iatrogenic or pharmaceutical exposures under poor factory hygiene conditions are the phenothiazines (antihistamines, tranquillisers), oral antidiabetic drugs (Tolbutamide), diuretics (chlorothiazide) and griseofulvin.

Irritant phototoxicity caused by altered light absorption is caused also industrially by petroleum oils, creosote, tar and pitch, perfumes (oil of Bergamot) and furocoumarins from the Umbelli-

ferae plant family; photo-dermatitis in celery or parsnip pickers occurs.

Common trade allergic sensitisations

Sensitisers only account for some 20% of industrial dermatitis, physical and irritant agents being much more important.

Space does not permit of a full list but some are important in a wide range of occupations or handling consumer products.

The sensitisation may however be also often acquired outside industry or on a group basis causing difficulty in incriminating the work. Among important common sensitisers may be listed:—

Arsenic. Both the inorganic and organic arsenic compounds are sensitising. Dermatitis has occurred in metal production from copper ore (including those in villages around), glass manufacture, sheep dips, hide preservation, vine growers, wood preservation and pharmaceuticals, or as agricultural insecticides.

Chromates. Hexavalent chromate is definitely antigenic and so is the trivalent form (Fregert and Rorsman 1964). Trivalent chromate may also be oxidised in tissues, as in tattoo marks, to hexavalent form. Industrial substances and occupations at risk are paint primers, chromated animal glue as in match heads, boot and shoe tanning, building industry (cement), electroplating, metallurgy (alloys), ink and lithography and photoengraving printing, engineering oils (contaminated with chromate), refractory foundry cores, photography, wood and fuels (chromate preserved wood ash), anti-rust radiation coolants for railways engines and dye mordants for furs and textiles.

The metallic chromium or chrome plating is not antigenic.

Cobalt and nickel. At least 86.5% (Marcussen, 1960) of nickel sensitisation occurs from non-industrial sources but 4% from industrial processes as electroplating, metal alloys and fat and margarine manufacture. Cobalt is a sensitiser and, as the element may be associated with nickel, it can be difficult to produce a completely pure metal. Metallic alloys and pigments used in paints, enamels and ceramics are the main exposures.

Formaldehyde. This is a commonly used preservative in medical and allied trades, and for leather, textiles and for coagulation of rubber latex. Combined with phenols or cresols (phenolic) or urea and melamine (aminoplast) it is condensed to make formaldehyde resins. Plywood and the wood adhesives and anti-crease textile resins have been sources of formaldehyde sensitisation.

Amines. These have widespread use in industry and as aniline type intermediates in dye stuff manufacture and as rubber accelerators (toluidine,

piperidine, hexamethylene amine). Ethylene amines which are often alkaline and irritant used as hardening agents with epoxy resin glues have been responsible for dermatitis.

Antibiotics and other topical agents. Sensitisation may occur during manufacture in pharmaceutical workers but mainly contact dermatitis occurs from topical treatments of minor injuries with sensitisers as commercial neomycin (B.C. and neamine neomycin A), penicillin and the tetracyclines (chlor-tetracycline and oxytetracycline). Neomycin is becoming more recognised as a source of contact sensitisation, and a cross sensitiser with framycetin, soframycin, bacitracin, kanamycin and streptomycin.

Other topical agents occasionally causing dermatitis when used in treatment of industrial injuries or skin diseases are the hydroxy quinolines, acriflavine, quaternary ammonium salts and lanoline or other emulsifiers and germicides as "Parabens" used in ointment bases. Necrotising ulcerations are reported from the quaternary ammonium dequalinium chloride.

Dyestuffs. Dyes used are mainly synthetic and derived from coal tar or petroleum chemicals. Soluble dyes are more likely to cause contact dermatitis than insoluble dyes and pigments. Most risk comes not from the fully developed dye, often firmly anchored chemically or mechanically in the fabric or material, but from residual dye intermediates left in the final product or improperly washed textile. Azo dyes are used in colouring of petrol, textiles, food, paint and ink colours. Contact sensitisation has occurred from ball-point pen inks and hosiery dyes, reds and yellows. Amino azo dyes used as food colourings are not thought to induce primary epidermal sensitisation by ingestion but they may, on a cross sensitisation basis, sometimes evoke cross reactions in persons already sensitised by the "para group" (Baer and Leider, 1949).

Other azo dyes liable to sensitise and photosensitise are flavine (azo-acridine), eosin used in lipsticks (azo-xanthene) and optical blanchophores (azo-stilbene) used in textiles and detergents.

Azo pigments may occasionally leach out of synthetic resins and solvents during use.

Other sensitisers include the triphenyl methanes (auramine, brilliant green, crystal and methyl violet), vat indigoid, alizarin and amine dyes as paraphenylene diamine used for hair or textile dyes which has an azo cross sensitisation. Paraphenylene diamine may cause a contact dermatitis or possibly, via toxic enzymal damage also a lichenoid dermatosis quite indistinguishable clinically from lichen planus. This has occurred in photographic workers using substituted paraphenylene diamine colour developers.

Essential oils of vegetation

Trees and timber. The essential oils and oleo-resins and other secondary products formed during the photosynthetic metabolism of trees and plants may act as irritants or sensitisers to those handling them.

Chemically these secondary products include organic acids, phenols, resorcinols, catechols, quinones, tars, terpenes, alkaloids etc.; cross sensitisations may occur. Foreign woods are more likely to cause contact dermatitis in wood workers than native. This occurs from naphthoquinones (lepacchols) in teak, Greenheart, Peroba do Campos and Jacaranda, and catechols in the Anacardiacae trees as Poison Ivy and Sumac, Japanese lacquer, Indian ink trees and Cashew nut used for synthetic resins.

The Conifer family (Pinaceae) of all the native woods are most likely to cause alpha-pinene sensitisation in furniture factories or house building. Sensitivity to alga-fungal tree lichens of trees and woods may cause contact dermatitis; usnic acid is the sensitiser (Mitchell, 1965); clothes may be affected by dried dusts. Preservation of timber with creosote, arsenical chromate or chlorinated organic chemicals, often in solvents, can also be sources of dermatitis.

Plants. Horticultural workers, florists and house workers are prone to irritant acidic juices from a large variety of vegetation or to traumatic injuries from stiff hairs, spines or bristles.

Only a few families however cause dermatitis. They include daffodils and narcissi (Amaryllidaceae), daisies, chrysanthemums (Compositae), lilies, tulips and hyacinths (Liliaceae), Primula obconica (Primulaceae) and buttercups and anemones (Ranunculaceae).

Among classic examples of contact dermatitis are "tulip finger" in florists especially from Rose Copeland and Preludin tulips (Rook, 1961), and hyacinth scabies with papulo-vesicular or follicular eruptions. Onions and garlic bulbs may irritate handlers.

Catering trade

Essential oils as a source of sensitisation include limonene of citrus fruits (lemons, grapefruit, oranges, tangerines), cinnamon and vanilla (cross sensitisers with Balsam of Peru (Hjorth, 1961), and synthetic essences as bitter almond (eugenol), almond oil (benzaldehyde oil). Many vegetables also carry an irritant risk but an unusual adverse effect arises from proteolytic enzymes in pineapples (bromelin) or pancreatic juices. Constant exposure to fruit or vegetable juices and wet conditions being also eczematogenic.

Synthetic resins and rubber chemicals

The technology here is based upon addition or condensation polymer formation of resins with later

cross linkage for final use or consumer products. Miscellaneous catalysts, solvents, plasticisers, fillers, pigments and special additives also being included in the formulation. Plasticisers are often productive of contact dermatitis in the consumer article.

As the manufacture is mainly enclosed, contact dermatitis at that stage is uncommon but initiators such as organic peroxides or metallic halides may cause accidental skin burns. Anti-oxidant amines or phenols, used to stop the polymer at the required length, may be irritant or sensitising as can hydroquinone used later to stabilise the polymer. Raw material condensates or intermediates are only potential hazards till fully polymerised and in the case of the former when cured to harden as with adhesives. The principle is accepted that a fully polymerised or hardened polymer is inert.

Personnel exposed to more risk are those processing the partly polymerised resin as in adhesives, laminates or mouldings to a final product, or occasionally when handling the consumer article.

Possibility of contact with reactive chemicals under such conditions include persistence of unreacted monomers, intermediates or catalysts, leaching of plasticisers to which may be added 25-50% of the whole especially from addition polymers, machining of cured plastics and thermal decomposition. Resins being completely polymerised in synthetic fibres (with the exception of some nylon), emulsion and solvent paints, and such addition polymers as polyethylene, polypropylene are without hazard.

Synthetic resins have widespread industrial and consumer use.

Some reported examples of contact dermatitis and the responsible agents are:—

Addition polymers

Celluloses — dermatitis from plasticisers in spectacle frames.

Vinyls — dermatitis from plasticisers in polyvinylchloride watch straps, rainwear, floor tiles, babies' drawers, artificial leather moulded domestic articles. Triphenyl (TPP) and tricresyl phosphate (TCP) have both caused reactions in the vinyls and cellulose articles.

Acrylics — dermatitis from moulding powders used for dentures, car accessories and consumer goods. The offending agents here are partially polymerised poly methyl methacrylate catalysts as amines, mercaptans, hydrazines in the moulding powder and hydroquinone, phenols and pyrogallol inhibitors, cobalt accelerators and peroxide catalysts of the hardening liquid.

Condensation polymers. The hazards here relate mainly to the incompletely polymerised resin and to catalyst hardening agents until the final product is set. Their final use is mainly for cast resins, adhesives and laminating agents and in paints.

Polyamides. Although pure nylon polymers are considered inert contact reactions have rarely been described to Nylon 6 epsilon Caprolactam polymers (Morris, 1960). Other textile fibres and other nylons, celluloses, polyvinylidene chloride (Saran), isocyanates (Perlon), acrylonitrile (Orlon), linear polyester (terylene) and regenerated protein are not productive of dermatitis although various textile coning oils, sizes and finishes may be.

Formaldehyde resins. Mainly used are phenol formaldehyde (phenolic) and urea or melamine condensates (aminoplast).

Sensitising agents are formaldehyde, intermediates, phenylol methanes or methylol ureas and hardening agents as hexamethylene tetramine. Dermatitis has occurred from those resins used for moulding powders, copying papers (para-tertiary butyl catechol), shoe adhesives (para-tertiary butyl phenol) and anti-crease amino-plast "non-crush" textile resins.

Epoxy resins. These have caused contact dermatitis not only from the epoxy resin itself but also from the polyamines used as cross linkage catalysts. Until adequate protective measures were employed, the incidence was considerable.

Exposure occurs in encapsulating "potting" electrical insulation, non-drip and resistant surface coatings and industrial paints, and as adhesives for metals, glass, ceramics and plastics.

Epichlorhydrin and Bisphenol A (isopropylidene diphenol) the raw materials are both aggressive and actively sensitising. Glass fibre laminates by traumatising the skin encourage penetration of sensitisers.

Polyesters. The linear textile fibres are without hazard but occasional contact dermatitis may occur from solvent based lacquers or when used as adhesives on glass fibre laminates. The cure agents, or solvents, as also lacquers, are mainly at fault; the resins themselves having only a low sensitisation index.

Rubber. Artificial rubber may be made from isoprene (natural rubber)-like polymers as butadiene or chloroprene or from high elastomers of such synthetic resins as vinyl, and silicones polymerised to a rubbery state. As with natural rubbers the risks of contact dermatitis reside mainly in the compounding materials necessary to achieve vulcanisation cure for consumer products.

Occasional dermatitis may occur during manufacture of the polymers from catalysts as mercaptans, boron trifluoride and aluminium chloride (initiators

in isobutylene polymers), stabilisers (phenyl-alpha-naphthylamine and epichlorhydrin in chlorobutadiene "Neoprene" polymers) or plasticisers.

Only rarely is the synthetic rubber itself a contact risk as chloroprene, butadiene or polysulphide rubbers.

Most contact dermatitis occurs from consumer products relative to additives as accelerators, and antioxidants which, being uncombined, leach from the final article.

The large range of additives used makes exact designation difficult but commonly at fault are accelerators as tetramethyl thiuram monosulphide (also used as insecticides (TMT)), mercapto benzothiazole (MBT), dipenta methylene thiuram monosulphide (PTD), paraphenylene diamine (PPD) (Wilson, 1960); and the anti-oxidant monobenzyl ether of hydroquinone which has also caused contact leucoderma. Plasticisers and dyes are much less common causes.

Protective industrial gloves, clothing, boots and shoes, personal clothing, elasticised textiles and foam rubber may affect the wearer.

Other hazards from rubber are the eczematogenic effect of alkaline rubber latices, and solvent rubber adhesives or those requiring a vulcanisation system as used in the boot and shoe industry.

Diagnosis and Management

Where the dermatitis is contact in pattern diagnosis is made on the clinical appearance and industrial exposure to a known irritant or sensitizer. In the case of patchy eczema the diagnosis is often a matter of the physician's opinion and assessment of the importance of the operative occupational and non-occupational agent in relationship to the constitutional diathesis of the patient.

In both patterns allergic sensitisation may be assessed by patch testing. These are single specified tests or batch tests. For instance, in hand eczemas such batteries may include common sensitizers or nickel, chromate, formaldehyde, balsam of peru, rubber, antihistamines, antibiotics, ointment bases, etc., other group sensitizers or the main hazards of the suspect occupation such as rubber compounding chemicals.

Difficulties in interpretation include false irritant positive reactions and multiple false positives in "status eczematicus" from testing during active dermatitis; false negative reactions also occur through non-penetration. Eosin, for example, in lipsticks may react on the thin lip-skin but not on the thick skin of the back. Stripping with adhesive scotch tape has been used to overcome this difficulty. Delayed positive reactions, 72 hours or in 6 - 7 days after the test, usually indicate true allergic sensitisation. Delayed positives after 7 - 10 days indicate sensitisation by the patch test itself. A latent

sensitivity can be awakened by the patch test itself, which would wrongly suggest responsibility for the dermatitis. Eric Skog (1965) has shewn a significant increasing number of positive test reactions to para-phenylene diamine, sensitisation being induced by the test, more so by the higher concentrations. Thus pre-employment patch tests are not advised. Sensitisations may also have been initiated by non-industrial contacts.

Where contact dermatitis has occurred the workman should be left on his own or alternative work if possible unless the dermatitis is severe. In sensitisation he should come off contact unless it is known the de-sensitisation hardening will occur. Accommodation hardening to irritants as oils, paraffin, soaps and detergents can and does occur with continued contact.

Most recover from contact dermatitis after months but some remain sensitive and have further attacks on re-exposure. Where a patchy eczema or a constitutional eczema pattern has emerged effort should be made to get the man back to his own job (if not sensitised) or alternative work as soon as possible; Hellier's (1958) figures indicate 77% of those with industrial eczema had recurrences whether or not their jobs were changed.

Psychoneurotic factors soon begin adversely to affect complete recovery where there is a prolonged absence from work.

An increment on the normal sickness benefit in the form of industrial benefit may itself increase the chances of eczema being diagnosed as occupational (Bettley, 1965), and could also militate against an early return to work.

Prevention and Protection

Prevention of industrial dermatitis is mainly referable to safety precautions, machine design and protective equipment to reduce skin contact, with proper factory floor and personal cleanliness and adequate washing facilities.

Industrial eczema is commonly initiated by the ill use of abrasives, high alkali soap or detergent cleaners, or solvents as white spirit, paint thinners, paraffin, petrol or trichlorethylene used on the site.

Occasional cases of contact dermatitis are related to sensitivity to rubber or plastic gloves and to barrier creams. Barrier creams are *sub judice* as to their effectiveness. They certainly aid in cleansing the skin after work. They are not entirely useless but must be limited in protective faculty under conditions of use which tend to remove them or interrupt the continuous film needed for protection. They do not stop the penetration of sensitisers and are not effective in continuous exposure to oils, cement or wet working conditions.

Conditioning creams with lanoline, soft paraffin or oil/water emulsions are helpful in maintaining the keratin integrity to water content and water/oil emulsion creams such as Ung.Aquosum may benefit the person whose hands tend to chap in the winter or those with patchy eczema in atopic or ichthyotic skins.

REFERENCES

- BAER, R. L., and LEIDER, M. (1949): Effects of Feeding Certified Food Azodyes in Paraphenylenedi-amine-hypersensitive Subjects, *J. invest. Derm.*, **13**, 223.
- BETTLEY, F. Ray (1965): Diagnosis of Industrial Dermatitis, *Brit. med. J.*, **ii**, 1340.
- BOURNE, L. B. (1960): Industrial Health and Welfare, *Med. Press*, **244**, 186.
- BUCKLEY, W. R. (1963): Localized Argyria, *Arch. Derm.*, **88**, 531.
- BURBACH, J. P. E. (1964): In: Progress in the Biological Sciences in Relation to Dermatology — 2. Edited by A. Rook and R. H. Champion. pp. 443-450. London: Cambridge Univ. Press.
- FREGERT, S., and RORSMAN, H. (1964): Allergy to Trivalent Chromium, *Arch. Derm.*, **90**, 4.
- FROST, J., and HJORTH, N. (1959): Contact Dermatitis from Hydrazine Hydrochloride in Soldering Flux. Cross Sensitization to Apresoline and Isoniazid. *Acta derm-venereol. (Stockh.)*, **39**, 82.
- HELLIER, F. F. (1958): The Prognosis in Industrial Dermatitis, *Brit. med. J.*, **i**, 196.
- HJORTH, N. (1961): Eczematous Allergy to Balsams, *Acta derm-venereol. (Stockh.)*, **41**, Suppl. 46.
- MARCUSSEN, P. V. (1960): Ecological Considerations on Nickel Dermatitis, *Brit. J. industr. Med.*, **17**, 65.
- MAYER, R. L. (1928): Die Überempfindlichkeit gegen Körper von Chinonstruktur, *Arch. Derm. Syph. (Berl.)*, **156**, 331.
- MITCHELL, J. C. (1965): Allergy to Lichens, *Arch. Derm.*, **92**, 142.
- MORRIS, G. E. (1960): Nylon Dermatitis, *New Engl. J. Med.*, **263**, 30.
- NAYLOR, P. F. D. (1955): Experimental Friction Blisters, *Brit. J. Derm.*, **67**, 327.
- PASCHOUD, J. M. (1963): Quelques Cas d'Eczéma de Contact avec Sensibilisation de Groupe, *Dermatologica. (Basel)*, **127**, 349.
- ROOK, A. (1961): Plant Dermatitis. The Significance of Variety-Specific Sensitization, *Brit. J. Derm.*, **73**, 283.
- RUBIN, L. (1949): Hyperkeratosis in Response to Mechanical Irritation, *J. invest. Derm.*, **13**, 313.
- SKOG, E. (1965): Sensitization to p-Phenylenediamine, *Arch. Derm.*, **92**, 276.
- SUSKIND, R. R., and WHITEHOUSE, H. S. (1963): The Housewife and Her Exposure to Washing Products, *Arch. Derm.*, **88**, 130.
- SZAKALL, A. (1955): Über die Eigenschaften, Herkunft und Physiologischen Funktionen der die H-Ionenkonzentration Bestimmenden Wirkstoffe in der Verhornten Epidermis, *Arch. klin. exp. Derm.*, **201**, 331.
- THOMSON, S. (1958): Some Aspects of Industrial Dermatitis, *Brit. med. J.*, **i**, 1135.
- VICKERS, H. R. (1958): Dermatitis in the Steel Industry in Sheffield, *Brit. med. J.*, **i**, 199.
- WILSON, H. T. H. (1960): Rubber-Glove Dermatitis, *Brit. med. J.*, **ii**, 21.