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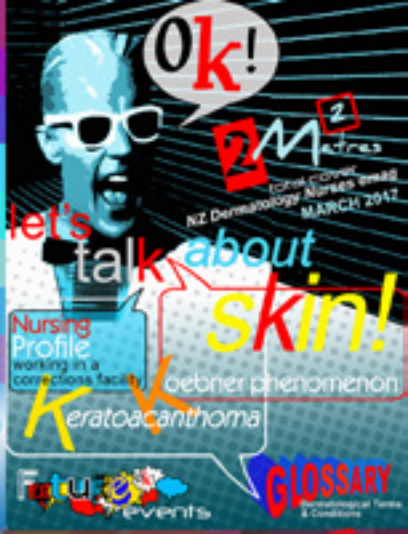
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NZ Dermatology Nurses **emag of the decade**

Full edition inside

# 2 Metres 2 total cover

the WAY it WA-Z

INSIDE TODAY


2 Metres total cover

NZ Dermatology Nurses emag

# the WAY it WA-Z

November 2013 to November 2020

this issue WXYZ

# editorial

Tracy Fenton

Ann Giles

Welcome to the last edition edited by Tracy and Ann of 2m$^2$ Total Cover, the official publication of the New Zealand Dermatology Nurses' Society Incorporated (NZDNS).

The NZDNS was founded in January 2010 following the dermatology nurses and allied health professionals group inaugural conference in 2006.

In 2013 at the eighth national conference the NZDNS members agreed to launch an online educational publication three times a year which would be available to everyone online. 2m$^2$ Total Cover which relates to the total area of skin that protects the whole human body began at the end of that year.

The emag aimed to promote excellence in the care of people with dermatological conditions through communication, education, research and professional development. To increase recognition of NZ dermatology nurses and nursing nationally and internationally and to benefit the public through providing education on skin conditions.

Starting something new meant starting at the beginning. Using the alphabet, focused each issue on a skin condition connected to that letter along with other interesting additions over seven years to enhance your professional development.

We have enjoyed the experience of editing and writing and are proud of the unique artistic design of the twenty two emags.

This issue has two professional development articles, one using X as one of the last four letters of the alphabet and an outline of life threatening dermatological conditions, along with an interesting nursing profile.

Save the date for the 15th annual NZDNS conference on 19 & 20 August 2021 in Queenstown.

We extend our best wishes to the NZDNS going forward.

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NZ Dermatology Nurses emag
NOVEMBER 2020




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**November 2020 contributors**
Tracy Fenton Ann Giles
Margie Martin

HS
HIDRADENITIS SUPPURATIVA
ONLINE

# The new online tool for people with HS is now live!

You'll find a range of tools at HS Online that are designed to help, educate, uplift and empower the HS community with information and support.

### HS facts
What exactly is HS and what causes it? Find the answers to some common questions

### Compare your symptoms
Learn about the stages of HS and view photos of the symptoms

### What's your sore spot
your patients can create guides to help discussion in clinic

### Living with HS
What is it like for other people with HS? Hear it straight from them in vlog-style stories

### Diet and lifestyle tips
Ideas to help maintain good health

### hs-online.co.nz
gives easy access to HS tools and information

Let your HS patients know they can check out information and guidance on their HS journey at **hs-online.co.nz**

# Xanthoma

## a skin sign of dyslipaemias

Tracy Fenton

The term 'xanthelasma' is derived from the Greek word xanthos (yellow) and elasma (beaten metal plate) (Nair & Singhal, 2018). A Xanthoma is a skin lesion caused by the accumulation of fat in macrophage immune cells in the skin and more rarely in the layer of fat under the skin (Ngan, 2005).

**Epidemiology**

Xanthomas are a common manifestation of lipid metabolism disorders. Epidemiologic data on cutaneous xanthomas is limited (Wanat & Hoe, 2020). They may occur in persons of any age however, xanthelasmas usually occur in people older than 50 years (Torres, 2019). Xanthomas associated with familial hypercholesterolaemia are an exception and often begin to develop prior to the age of 10 years (Wanat & Hoe, 2020). Equal prevalence of xanthomas is reported in males and females. Xanthoma disseminatum occurs before age 25 years in two thirds of cases and in a male-to-female ration of 2.4:1 (Torres, 2019).

**Etiology and pathogenesis**

Cutaneous xanthomas represent deposition of lipid and associated inflammation in the skin. The pathogenic mechanisms that lead to these are not fully understood and may differ based on the etiology and type of xanthoma (Wanat & Hoe, 2020).

Xanthomas are a result of lipoproteins passing through the blood vessel wall into the subendothelial layer where the lipoproteins are picked up by macrophages (Zaremba et al., 2012).

The characteristic histologic feature of cutaneous xanthomas is lipid-laden macrophages, also known as 'foam cells'. The number of foam cells and the presence of associated findings such as inflammatory cells, extracellular lipid deposition and fibrosis vary with type and age of the xanthoma (Wanat & Hoe, 2020).

**Diagnosis and differential diagnosis**

The diagnosis of cutaneous xanthomas involves determining the type of xanthoma and the underlying cause through the patient history, physical examination and relevant laboratory investigations (Wanat & Hoe, 2020). The diagnosis of xanthomas is not usually very difficult and generally are diagnosed clinically (Zaremba et al., 2012). The patient history should include an assessment for risk factors for xanthoma development such as:

- Underlying diseases including diabetes, thyroid disease, nephrotic syndrome and haematologic disease.
- Medications that may cause hyperlipidaemia such as estrogens, tamoxifen, prednisone, oral retinoids, cyclosporine, olanzapine, nilotinib and protease inhibitors.
- Family history of primary lipid disorders or diseases associated with hyperlipidaema.

Differential diagnosis includes (Torres, 2019):

- Acute complications of Sarcoidosis
- Amyloidosis, nodular localized cutaneous
- Cerebrotendinous xanthomatosis
- Dermatologic manifestations of juvenile xanthogranuloma
- Erdheim-Chester disease
- Generalised granuloma annulare (eruptive xanthoma)
- Histoid leprosy (eruptive xanthoma)
- Lichen amyloidoisis
- Macular amyloidosis
- Molluscum contagiosum (eruptive xanthoma)
- Multiple subepidermal calcified nodule (eruptive xanthoma)
- Necrobiosis lipoidica
- Necrobiotic xanthogranuloma

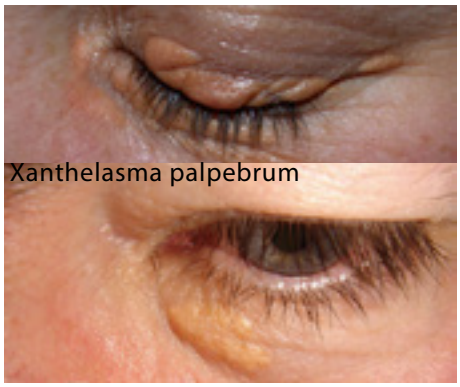
**Clinical manifestation and presentation**

Clinically xanthomas are yellowish papules, nodules or plaques. In relation to lesion localization and morphology they can be distinguished as (Zaremba et al.,

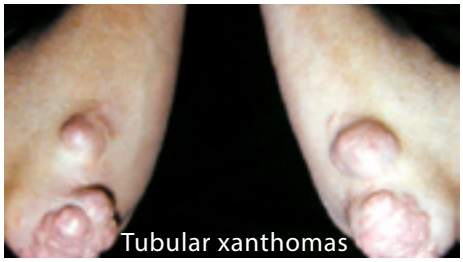
2012): flat, nodular, tendons and joint, subdermal, seeding, nodular-seeding, band-shaped of the hands and xanthomas of the eyelids.

Xanthomas are classified into types based on where they are found on the body and how they develop (Ngan, 2005).

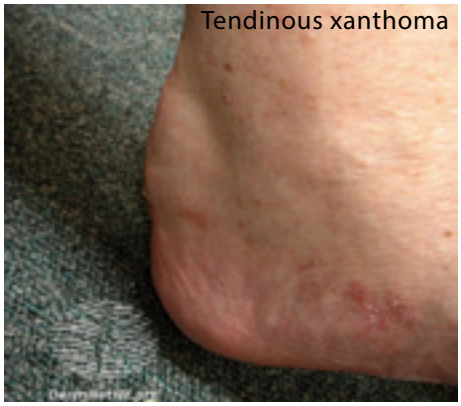
*Xanthelasma palpebrum* – The most common type of xanthoma. Soft, velvety, yellow flat papules or plaques arise symmetrically on upper and lower eyelids. They start off as a small bump and gradually grow larger over several months. They may or may not be associated with hyperlipidaemia (Ngan, 2005).


Xanthelasma palpebrum

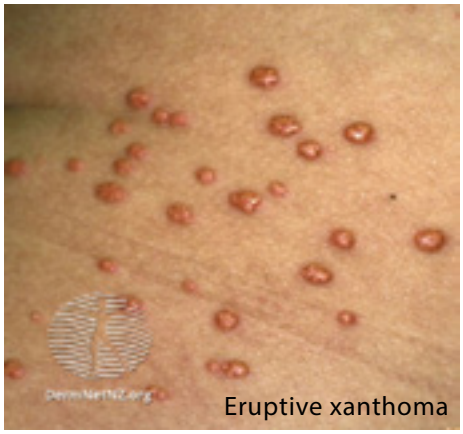
*Tuberous xanthomas* – These are yellow-orange or erythematous papules or nodules located over joints or extensor surfaces of the extremities, especially the elbows and knees. They may be grouped and can reach up to 3cm in size (Wanat & Hoe, 2020).


Tubular xanthomas

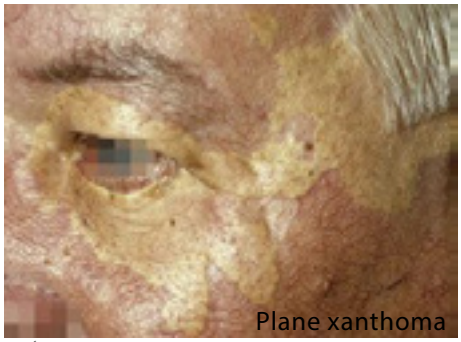
*Tendinous xanthoma* – These occur as subcutaneous nodules or papules in relation to tendons and are most frequently seen in familial hypercholesterolaemia. These occur most commonly to the extensor tendons over the knuckles and in the Achilles tendon (Griffiths et al., 2016).


Tendinous xanthoma

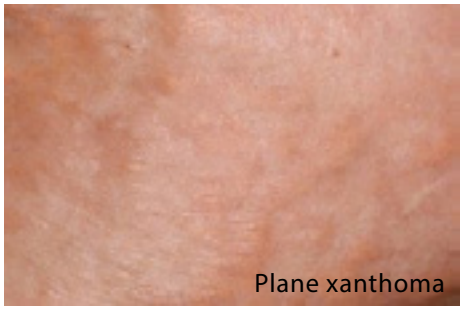
*Eruptive xanthomas* – These are 1-5mm erythematous yellow papules that appear in crops with an abrupt onset. The most common sites of involvement are the extensor surfaces of the extremities and buttocks (Freedberg et al., 2003). Eruptive xanthomas are highly suggestive of hypertriglyceridaemia (Wanat & Hoe, 2020).


Eruptive xanthoma

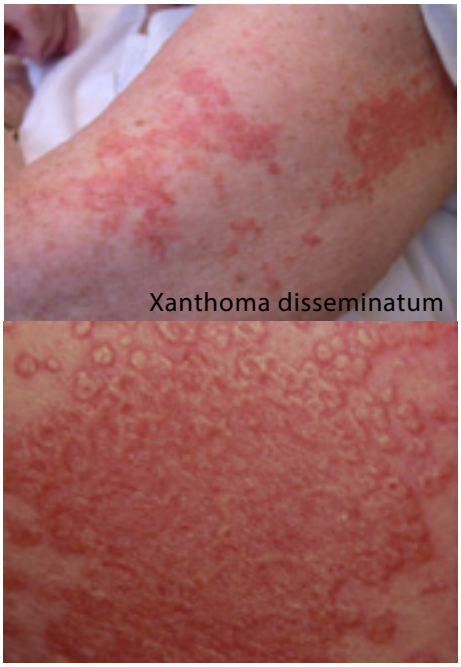
*Plane xanthoma* – These are flat, smear-like lesions that can occur anywhere on the body. They can be a feature of homozygous familial hypercholesterolaemia. (Griffiths et al., 2016). These can occur in both the presence and absence of primary or secondary hyperlipidaemia (Wanat & Hoe, 2020).


Plane xanthoma

Images from dermnet with permission http://www.dermnetnz.org


Plane xanthoma

*Xanthoma disseminatum* – This is due to a rare form of histiocytosis and lipid metabolism is normal. The skin lesions usually consist of hundreds of small yellowish-brown or reddish-brown bumps, which are usually evenly spread on both sides of the face and trunk. They may particularly affect the armpits and groins. The small bumps may join together to form sheets of thickened skin and it can also affect internal organs such as the liver, lungs and kidneys (Ngan, 2005).


Xanthoma disseminatum

**Treatment and management**

A fasting lipid panel to evaluate for dyslipidaemia should be performed in all patients with xanthomas (Nair, 2018). Patients with hyperlipidaemia should receive further evaluation to discover the cause. If they have eruptive or plane xanthomas and hyperlipidaemia this warrants evaluation for causes of secondary hyperlipidaemia such as diabetes, thyroid disease, liver

disease and renal disease (Wanat & Hoe, 2020).

Patients with tendinous or tuberous xanthomas should automatically be evaluated for an inherited dyslipidaemia (Wanat & Hoe, 2020). Those with plane xanthomas can also have a normal lipid profile but they can also be associated with haematologic disease and therefore should be evaluated for associated haematologic disease (Wanat & Hoe, 2020).

The main aim of treatment for xanthoma that is associated with an underlying lipid disorder is to identify and treat the lipid disorder (Ngan, 2005). In many cases treating the underlying disorder will reduce or resolve the xanthoma. More importantly treating hyperlipidaemia will reduce the risk of heart disease and treating hypertriglyceridaemia will prevent pancreatitis. Very effective medications may be prescribed to treat the underlying cause by the appropriate specialist.

Cutaneous xanthomas are not life-threatening and are usually asymptomatic, therefore treatment specifically for cutaneous xanthomas is not mandatory, although it is often desired for cosmetic reasons (Wanat & Hoe, 2020). Surgery or locally destructive modalities can be used to remove xanthomas that do not resolve spontaneously or with the treatment of the underlying cause (Ngan, 2005). However, recurrences post-surgical treatment of xanthomas are common (Torres, 2019) and impractical for patients with more extensive involvement (Wanat & Hoe, 2020). Other treatment modalities can include:

- Topical trichloroacetric acid (chemical peel)
- Cryotherapy
- Laser vaporisation
- Electrodessication

In some cases a skin eruption can be the first symptom of lipid abnormality and dermatology can be important in the process of diagnosing metabolic disease and preventing serious health consequences (Zaremba et al., 2012).

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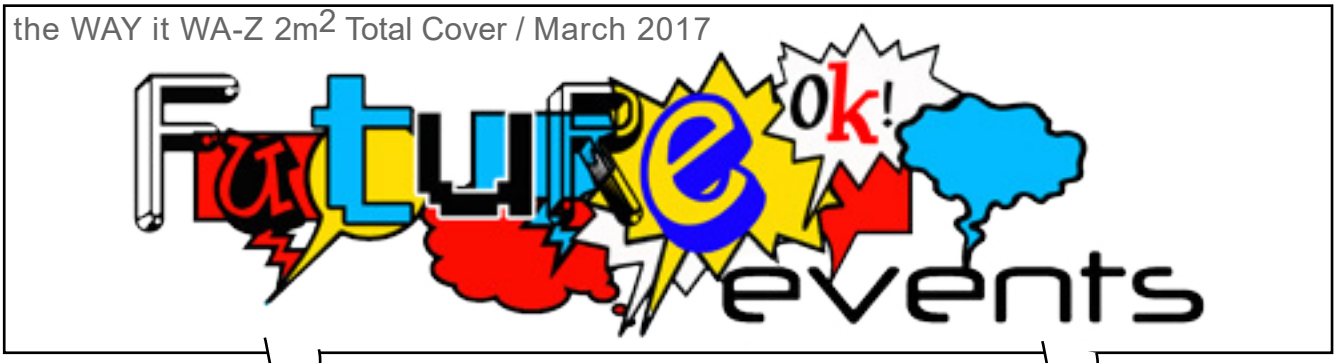



# 2020

**15th Annual Practical Course in Dermoscopy and Update on malignant melanoma 2020 Livestream**
4 - 6 December 2020
https://ce.mayo.edu/dermatology/content/15th-annual-practical-course-dermoscopy-and-update-malignant-melanoma-2020-livestream#group-tabs-node-course-default1

**45th Annual Meeting of the Japanese Society for Investigative Dermatology Virtual Meeting**
11-13 December 2020
https://jsid45.jp/En/index.html

# 2021

**Society for Investigative Dermatology Annual Meeting Virtual Conference**
13 -16 May 2021
https://www.sidannualmeeting.org/

**15th New Zealand Dermatology Nurses' Society Conference**
19 & 20 August 2021
Queenstown, New Zealand
https://www.nzdermatologynurses.nz/Events.html


# MENTAL HEALTH NURSING

## towards psychological well-being

**Why did you choose nursing as a career and when?**

From the age of 13 years old I worked in a rest home in the weekend and holidays. When I was 16 years old I worked in Paraparaumu Maternity Hospital as a Nurse Aid. When I was at school I wanted to be an air hostess, but I was too fat, so I decided to train as a nurse.

**Where did you do your nursing training and post graduate study?**

I trained as a Community Nurse at Hutt Hospital. At the time I was married to a policeman. In 1973 we were transferred to Porirua. There were two hospitals close by, Kenepuru Maternity Hospital and Porirua Psychiatric Hospital. I worked night duty at the psychiatric hospital in Porirua.

I trained as a Psychiatric nurse in 1979 and registered in 1982. I did post graduate study in Mental Health in 2006. During my time at Porirua Hospital I worked as a Staff Nurse and then as a Unit Manager. In 1996 I joined the newly formed Crisis Assessment and Treatment team (CATT) in the Wellington region.

In 2009 I became a Community Mental Health Nurse / Crisis Nurse for the Wairarapa District Health Board and in 2011 I helped in Christchurch following the earthquake.

I went to work on Christmas Island in 2012. This island, 1500 kilometres west of Australia is an Australian territory and close to Indonesia. I worked with refugees and later with detainees who were known as 501's due to their visa being cancelled on 'character' grounds and they were for return to their country of birth.

In 2015 I returned to NZ and took a position with the Auckland District Health Board in the Planned Acute Care Team and the Urgent Response Mental Health Team.

**What is a typical day in your work life like including the most important skills and abilities required?**

We have a daily handover where we discuss between 20 and 50 people needing our care. We accept referrals each day. These are generally for those who require short term support during a crisis and may return to their GP care or become part of the care and recovery service. The Care and Recovery Team support those in the community longer term. We also phone people, visit them in their homes and make decisions on care and treatment perhaps with daily face to face follow-up or by phone, review people in respite care, see clients and their extended family and review approximately three people in the afternoon with our psychiatrist to make decisions as a team in regards to immediate treatment and planning. We work shift work including night shifts. We educate clients and their families, support people with medication adherence and carry out duties as Duly Authorised Officers within the remit of the Mental Health Act. This includes supporting people through the committal process of the Mental Health Act maybe to hospital, home with support and follow up, or discharged from the Act process. We also assess and place clients in community respite facilities for 24 hour support.

**How does your role fit in with the nursing and medical team?**

We liaise with inpatient units within the hospital system who are treating clients for physical health issues, other community mental health nurses and emergency department staff as the Psychiatric Liaison Service. We continually assess people and make decisions on admission, utilising respite care, work with NZ Police and organise community support. We also work with universities, the justice system and Justice of the Peace. We speak to solicitors, GP's and employers.

**What kinds of nursing decisions do you have to make and who assists you when making difficult decisions with patients and families?**

We often have to rely on our own knowledge and call on our past experiences in making decisions. We work closely with others in our team including social workers, nurses and occupational therapists.

**What abilities or personal qualities do you believe contribute most to success in your job?**

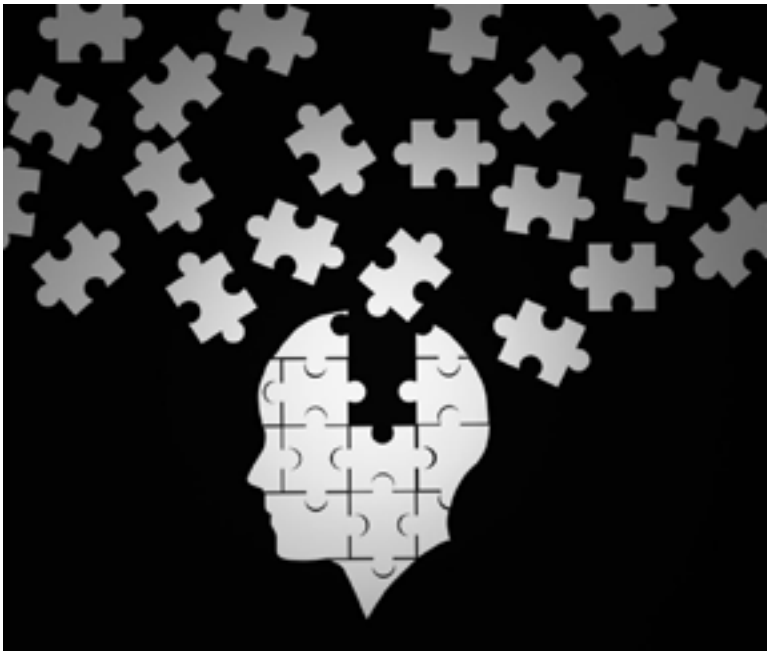
A good sense of humour, ability to listen, intuition based on many years of training and experience, adaptability, pragmatism, acceptance and an empathy for others.

**What are the most rewarding aspects or important personal satisfactions of your job?**

- Being part of a team.
- Seeing clients move forward in their lives and no longer needing acute input.
- Being part of the client and their family during a crisis or relapse.
- Watching and supporting a client who takes on board 'early warning signs' and is able to understand and use these effectively. Then they do not require mental health input except in a rare crisis.

**What part of your job do you find most challenging?**

Throughout my career probably the most challenging thing has been encouraging other health professionals to accept mental health illnesses as an illness.



Mental health clients have the same rights to acceptance, care and all treatments as everyone in the community including ourselves.

Stigma, whilst reducing, dramatically remains a part of health services.

**Do you ever advise patients on skin care. If so what do you tell them?**

Our role is very diverse and there would be very little in regards to physical health, mental health and social issues we don't get asked.

Our advice in many areas is general however, we do at times assist with getting clients reviewed by their GP. This may include making the appointment, facilitating them to attend the appointment and perhaps at times accompanying them.

Part of our home care and support is likely to include hygiene and diet.

Our psychiatric team may at times make a referral when required or seek advice from physical health specialists.

**What nursing preparation, educational or other work would you recommend for someone wanting to become a nurse in your specialty?**

My personal advice/ recommendation would be to spend at least a year in an acute inpatient Mental Health Unit before coming to work in the community.

To gain skills in feeling comfortable talking with clients and their families, being able to support in crisis and recovery.

Understanding medications, doses and side effects and being able to give an injection!

**If you could change one thing in your role, what would it be?**

To have more time to spend with clients and to do more intensive care. To spend less time at the computer. Is that two things?

Margie has worked as a Mental Health Nurse for 40+ years. She works at the Auckland District Health Board, Auckland, New Zealand

# DERMATOLOGY EMERGENCY

## LIFE THREATENING SKIN CONDITIONS

Ann Giles

The term dermatology emergency seems contradictory. Life threatening skin conditions are uncommon however, globally skin diseases are widespread, disabling, frequently difficult to live with and can significantly reduce a healthy life (Hay et al., 2014). Scarring, disfigurement and visual effects cause loss of self-esteem and confidence. Several may be associated with poor outcomes if treatment is delayed (Chowdhury, 2017; Nair, Moorthy & Yogiragan, 2005).

General life expectancy and death rates around the world varied in 2017. Across all countries life expectancy at birth ranged from 49.1 years in the Central African Republic to 82.1 years in Switzerland for men and from 54.9 years in Central African Republic to 87.6 years in Singapore for women (Global Burden of Diseases 2017, 2018). Life expectancy in 2017 in NZ was 80.2 years for men and 83.6 years for women (Stats NZ).

The prevalence of skin diseases in adults in NZ has not been widely researched or published (Oakley, 2009). The Royal New Zealand College of General Practitioners' Curriculum for General Practice 2014 states '*skin conditions account for approximately 15 percent of all consultations in general practice, which is where most dermatological consultations in New Zealand occur'* (RNZCGP, 2014).

Mortality directly caused by skin disease is similarly unpublished. Twenty four skin disease categories explored in the American Academy of Dermatology's Burden of Skin Disease study found half were fatal causing 22,953 deaths in 2013. The same study revealed that the average age of death from skin diseases was 68.2 years, five years younger than those who died of all other causes. Melanoma caused 41% of these deaths, 15% of deaths were caused by wounds followed by 10% related to cutaneous infections. Other skin conditions causing death included connective tissue disease, viral (HSV/HZV) and fungal disease, drug eruptions, cutaneous lymphoma, bullous disease and ulcers (Lim et al., 2017).

Although skin cancer is the most common of all cancers (Oakley, 2008) and melanoma has a high mortality rate, other potentially life threatening conditions can rapidly progress and require early recognition and supportive skin care to limit mortality (Oakley, 2009).

# Drug eruptions / Drug reaction with systemic symptoms (DRESS) / Drug induced hypersensitivity syndrome (DIHS)

**Prevalence**

Approximately 3% of patients admitted to hospital have rashes caused by adverse drug reactions.

**Information**

Rash improves when suspected drug is stopped. May take weeks to fully resolve. Many types of drug eruptions ranging from mild to severe cutaneous adverse reaction (SCAR). Skin features include redness, pain, blistering, urticaria with tongue or throat swelling, face and mucous membrane involvement. Other symptoms include lymphadenopathy, arthralgias or arthritis, abnormal blood counts, shortness of breath, wheezing or hypotension.

**Mortality**

Drug induced hypersensitivity syndrome, association with internal organ involvement and fever has a mortality rate of about 10%.

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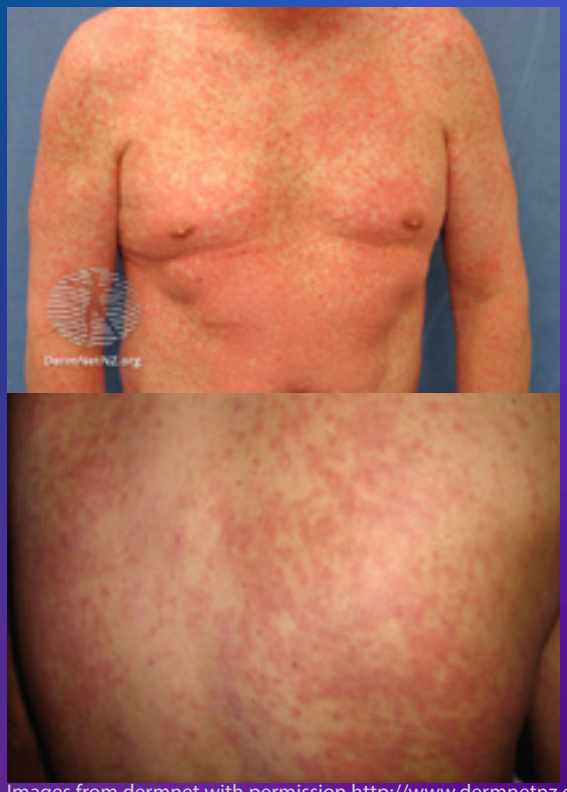

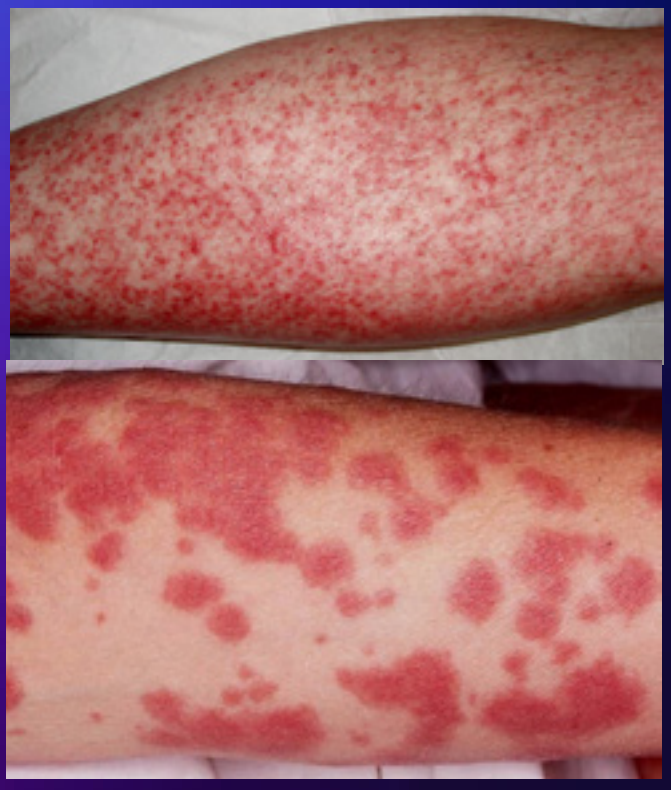

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# Erythema multiforme (EM) minor and major

**Prevalence**

EM prevalence unknown. Estimated at less than 1%. EM minor is a disease of young adults with median age of 24 years. More common in males.

**Information**

EM minor, is an eruption of classic target lesions on arms and legs with mild fever and malaise persisting for 1-3 weeks. Infection represents about 90% of cases. Herpes simplex virus (HSV) most common infectious agent. Drug-associated EM reported in less than 10% of cases. May recur with recurrent HSV.
EM major usually drug eruption with erosions and blisters mouth, lips, eyes and genitalia.

**Mortality**

EM major has mortality rate of approximately 1%.

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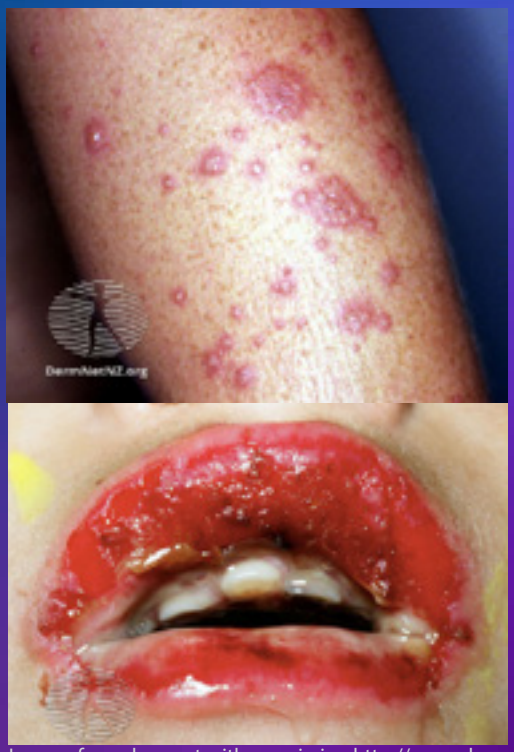

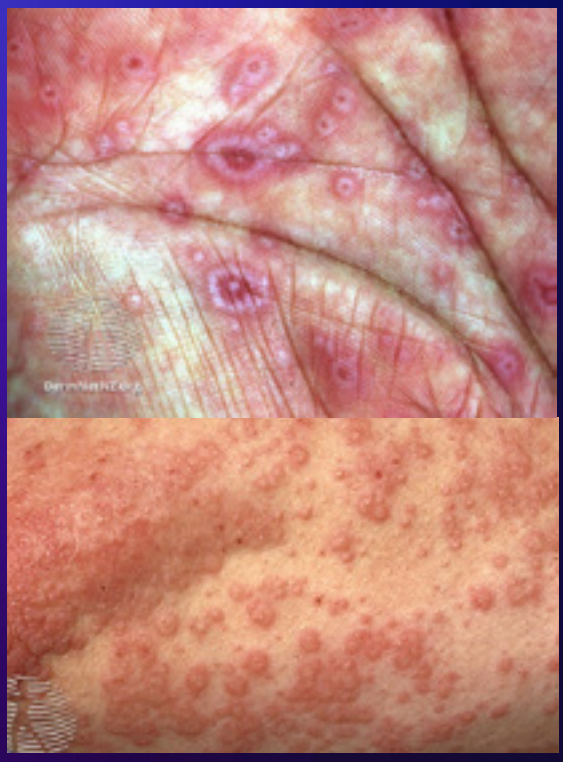

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# Toxic epidermal necrolysis (TEN) / Stevens - Johnson Syndrome (SJS)

## Prevalence

SJS/TEN is a very rare complication of medication use (estimated at 1–2 per million people each year for SJS and 0.4 –1.2 per million people each year for TEN).
100 times more common with human immunodeficiency virus (HIV) infection.

## Information

Severe cutaneous drug reaction characterised by flu-like illness followed by the rapid appearance of painful erythematous rash affecting 30% of skin surface. Desquamation of skin. Mucous membranes involvement severe including eyes, lips, mouth, esophagus (difficulty eating), gential area.
Drugs responsible for more than 80% of cases. Drug most often begun 1-3 weeks before presentation. Risk of recurrence if exposed to same drug or structurally related medicine. Family members at increased risk compared to the normal population. Most common drugs include sulphonamides, penicillins, cephalosporins, anti-convulsants, allopurinol, paracetamol and nonsteroidal anti-inflammatory drugs (NSAIDs).

## Mortality

Mortality rate approximately 30-35%, most commonly from bacterial sepsis.
More dangerous in those over 60 years old.

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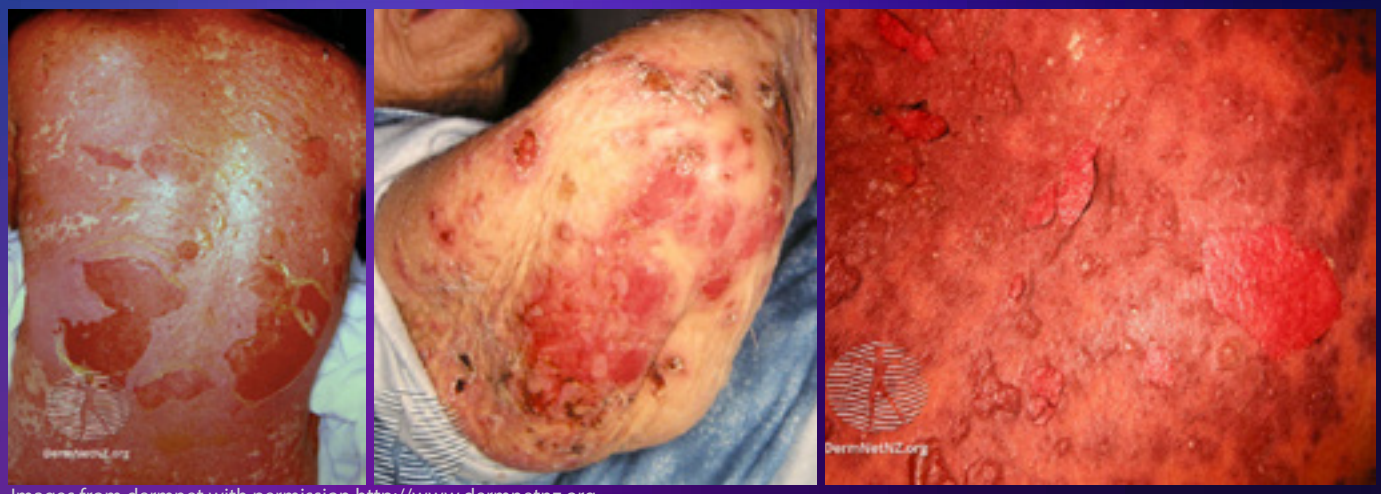

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# Erythroderma

**Prevalence**

Prevalence unknown. Rare but can occur at any age or race. Three times more likely in males. A third of cases have no cause.

**Information**

Serious inflammatory skin disease affecting entire skin surface. May be acute or chronic. May or may not be itchy. Red man syndrome term for idiopathic erythroderma. Clinical sign related to a wide range of cutaneous and systemic diseases including dermatitis, psoriasis, immunobullous disease, cutaneous T-cell lymphoma (Sézary syndrome), underlying systemic malignancy, HIV infection, graft vs host disease (GVHD). Blood tests show neutrophilia. Skin biopsy shows neutrophilic and mixed inflammatory infiltrate.

**Mortality**

Mortality rate ranges from 4 to 64%. Unfavourable prognosis related to advanced age, comorbidities and need for hospitalization.

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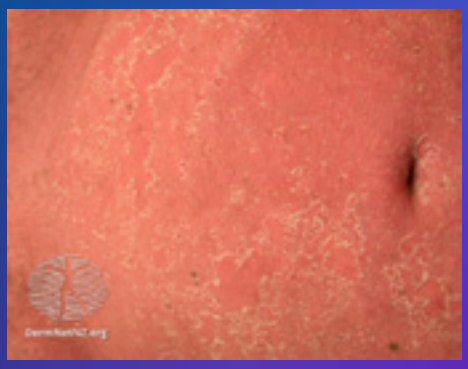

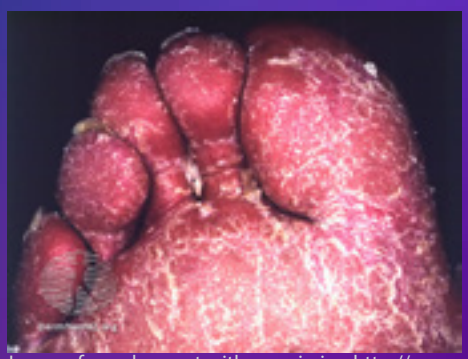

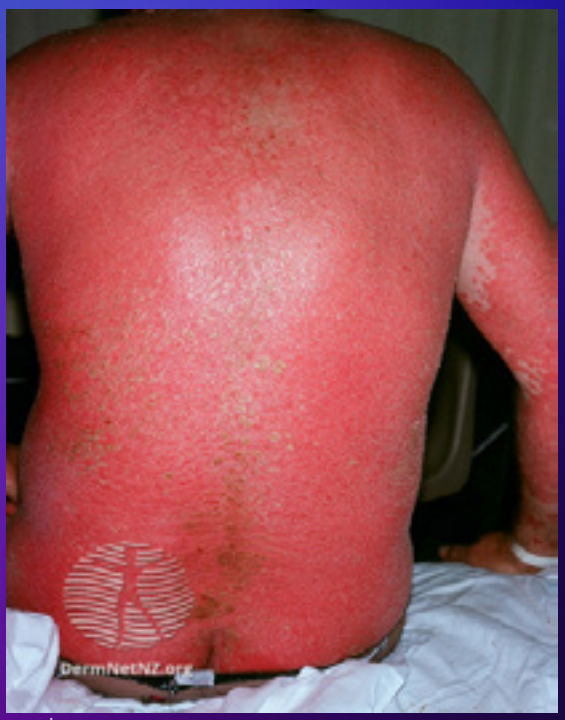

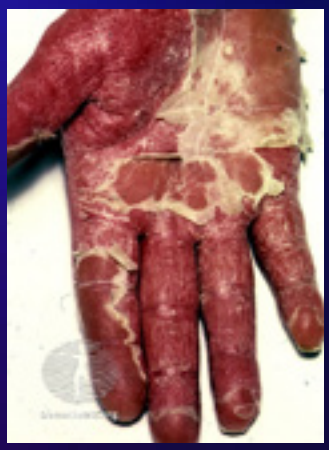

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# Acute febrile neutrophilic dermatoses or Sweet syndrome / Pyoderma gangrenosum (PG)

## Prevalence

Classical Sweet syndrome most common between 30 and 60 years old. More common in women.
PG second most common cutaneous sign of Inflammatory Bowel Disease (1–3%).

## Information

Sweet syndrome may be idiopathic or associated with infection often upper respiratory tract infection, malignancy (acute myeloid leukaemia) and drugs. Rash with systemic symptoms. Skin lesions evolve rapidly, juicy often 'pseudo-vesicular' plaques or nodules accompanied by fever, leukocytosis, conjunctivitis and arthralgia. Lesions often located asymmetrically on upper extremities, neck and face. May be painful. There may also be oral ulcers. Associated inflammatory disease most commonly Crohn's disease and ulcerative colitis in about 16% of patients with Sweet syndrome.
PG associated with rapidly enlarging, very painful ulcer. It is not an infection (pyoderma). It does not cause gangrene (gangrenosum). Characterised by full-thickness ulcer with blue undermined borders. New lesions appear after local trauma. May be related to internal disease. Common complication of ulcerative colitis rather than Crohn's disease. Also rheumatoid arthritis, blood disorders and active hepatitis.

## Mortality

Death from Sweet syndrome is rare.
PG mortality reported as 16%

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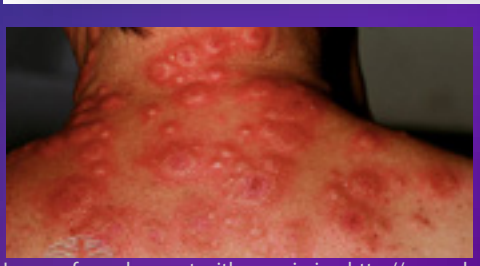

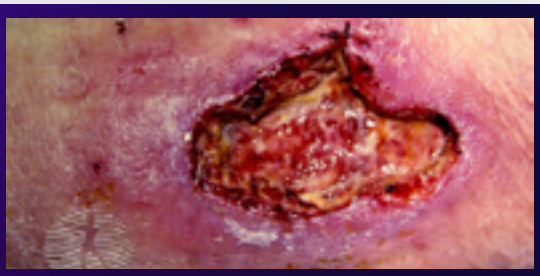

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# Urticaria and angioedema in adults

## Prevalence

Prevalence differs geographically and globally from 1.4% in Asia to 0.5% in Europe and 0.1% in North America. Women more affected than men. Another study estimated 15% to 23% of adults have experienced at least one episode of acute urticaria in their lives. Prevalence of chronic urticaria in adults estimated at 0.5% to 5%. Angioedema accompanies urticaria in 40% of cases or occurs on its own in 10%.

## Information

Skin signs of urticaria / hives: recurrent transient oedematous dermal papules and plaques (weals) which may be asymptomatic, often very itchy and sting or burn. Acute lasting less than 6 weeks or chronic lasting more than 3 months. If allergic, may progress to anaphylaxis. Allergens: drugs (5%), food (3%), contact allergy or bee/wasp stings. Non-allergic causes: infections (50%) other medical problems, drugs (opiates, aspirin, NSAIDs) or food. Oral steroids treat acute urticaria. Avoid triggers and antihistamines for chronic forms. Angioedema is redness and subcutaneous swelling of the deep dermis commonly around eyes and lips as a reaction to urticaria. Small blood vessels leak fluid into tissue. Painful and tender with or without itch. Happens quickly and briefly (24-48 hours). Can be acute /allergic, non-allergic drug reaction, idiopathic / chronic relapsing, rarely hereditary or acquired during lifetime. Breathing difficulties due to airway involvement may need emergency respiratory support in an intensive care unit.

## Mortality

Urticaria and angioedema manageable and improves with prompt and proper treatment. Mortality increases with angioedema of upper airways.

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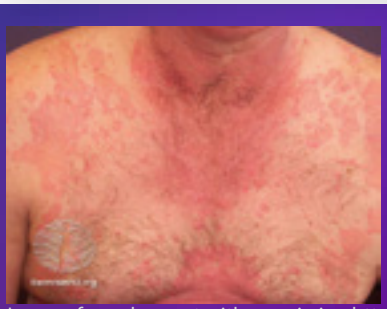

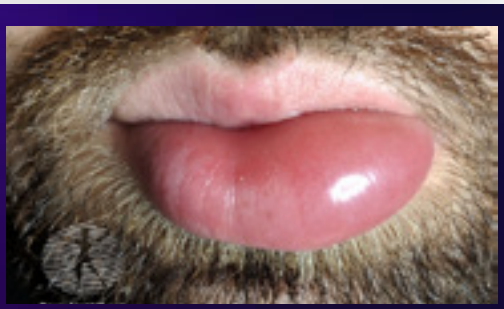

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# Inherited and immunobullous blistering skin eruptions

## Prevalence

More than 150 New Zealanders have Epidermolysis Bullosa (EB) with mild to moderate symptoms. Prevalence in the USA 1986-2002 was 11 per million live births. Prevalence of autoimmune bullous diseases (AIBDs) are scarce. Incidence in some European countries, US and UK estimated between 13.3 and 66 cases per million people annually. Men and women equally affected. Highest incidence in people older than 80 years.

## Information

EB is a group of rare inherited disorders usually diagnosed at birth. Minor skin trauma or friction causes fragile skin to painfully tear, blister and erode on the whole body including mouth and throat.

At least 9 distinct AIBDs broadly divided into two groups depending on location of blisters in the skin. The bodies immune system mistakenly attacks proteins of epidermis and dermal-epidermal junction that stick skin layers together. Categorised into subepidermal (pemphigoids; bullous pemphigoid, pemphigoid gestationis (bullous eruption of pregnancy), cicatricial pemphigoid (affects mouth, nose, eyelids and gentials), linear IgA disease, dermatitis herpetiformis (gluten-sensitive enteropathy) and epidermolysis bullosa acquisita (blistering following trauma) or intraepidermal (pemphigus group; pemphigus vulgaris (commonly caused by medications) and paraneoplastic pemphigus (may occur from underlying tumour). AIBDs causes painful blisters (subepidermal blistering) or erosions on the skin, mouth, throat, nose, eyes, scalp or gentials. Cause unknown. Associated with medications, ultraviolet light and genetics. Primary treatment is prednisone corticosteroids, immunosuppressive medications and supportive skin cares.

## Mortality

Mortality with pemphigus ranges from 5-30%. Death rarely related to disease. Usually secondary to systemic treatment, older age, infections, pneumonia and septicaemia.

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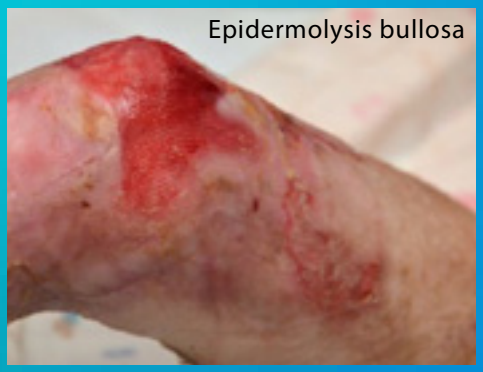





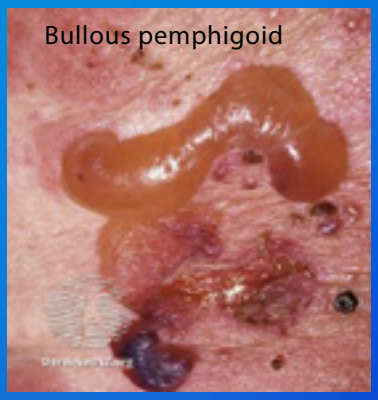



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# Dermatological Terms and Conditions



**Wart** - a small non-cancerous skin growth caused by a virus that appears on the surface of the skin.

**Weal** - a transient elevation of the skin due to dermal oedema, often pale centrally, with an erythematous rim or flare and without surface change. Wealing indicates urticaria or an urticaria-like condition.

**Well-defined** - having a clear sharp border separating it from its surrounds.

**Wet mount** - a microscopy examination technique where the specimen is held between a slide and a cover slip in a fluid. A wet mount would typically be used to assess the motility of live organisms in a swab, e.g. T*richomonas* in a vaginal swab.

**Whitehead** - also called Closed Comedone is a type of acne with a white or yellowish head on the skin. Whiteheads often appear in teenagers and young adults but can appear in people of all ages.

**Wide excision** - a surgical procedure in which an extra margin of normal tissue is removed in addition to the tumour. It is often a second procedure after a cancer diagnosis.

**Widespread** - an adjective pertaining to spread widely, being extensive or generalised.

**Winter itch** - a type of subclinical dermatitis that affects individuals during cold weather. It is also know as pruritus hiemalis.

**Wood lamp** - a Wood lamp emits long wavelength UVA light and is used to examine skin pigment changes (e.g. vitiligo or melasma) and fluorescent infections such as cat ringworm.

**Wrinkle** - a small crease or fold in the skin surface resulting from aging or frowning.

**Xeroderma** - the lower portion of the dermis. It is composed of coarse elastic fibres and thick collagen bundles parallel to the skin surface.

**Xerosis** - abnormally dry skin.

**Yaws** - a chronic tropical skin infection that may also infect bones in its late stages. It is caused by a spiral-shaped bacterium *Treponema pallidum pertenue*. Yaws belongs to the same family of bacteria as syphilis, *Treponema pallidum* subspecies *pallidum*.

**Zosteriform** - pertaining to herpes zoster, zoster-like, unilateral and restricted to a dermatome or distribution of a single spinal nerve.

**Zygomycosis** - a rare infection caused by a class of fungi called Zygomycetes. This a relatively primitive class of fungi which live on decaying organic matter.

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