

Australian Vaccinology Course 2026

How Vaccines Work

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WHY IT MATTERS

Vaccines: Medicine's Greatest Success

154 million

lives saved by childhood vaccines since 1974 (14 diseases; 101 million were infants)

94 million

of those from measles vaccine alone, about 60% of the total

>99%

fewer polio cases since 1988; >20 million people can walk who would have been paralysed

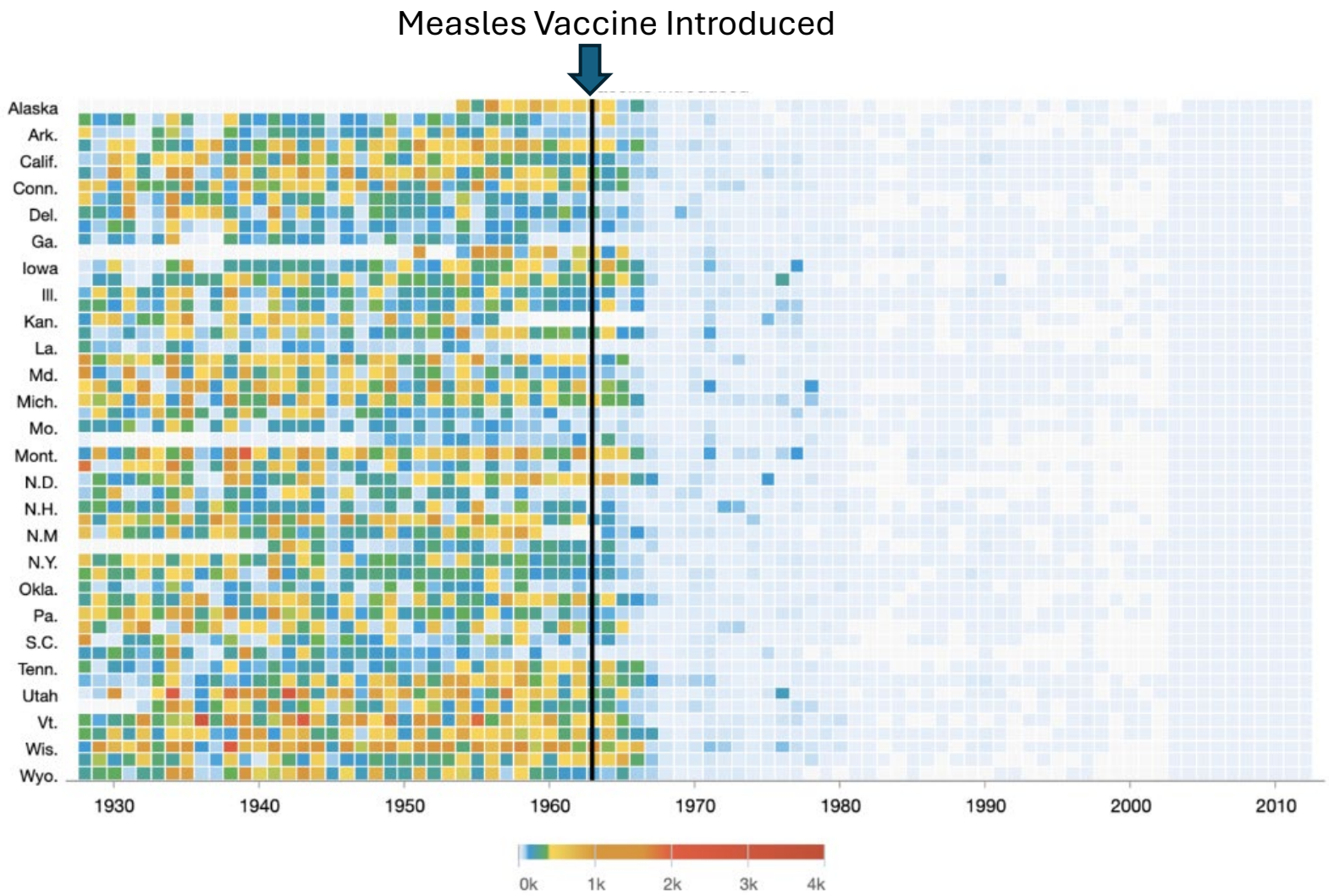
Eradicated

Smallpox: last natural case 1977; declared eradicated by WHO in 1980

Sources: Shattock AJ et al. Lancet 2024 (led from Telethon Kids Institute, with WHO); WHO polio and smallpox fact sheets.

WHY IT MATTERS

What a Vaccine Can Do: Measles in the USA



Based on Project Tycho data (University of Pittsburgh). US measles was declared eliminated in 2000.

Each row is a state; each column is a year.

Colour shows measles cases per 100,000 people.

The black line marks the measles vaccine introduction in 1963.

Within a few years, epidemics that occurred every year almost vanished.

The Immune System Has Two Arms

Innate Immunity

- **Fast:** minutes to hours
- **General:** the same cells respond to many different microbes
- **Sensors:** recognise ‘danger signals’ shared by many pathogens
- **Cells:** neutrophils, macrophages, dendritic cells, NK cells
- **Memory:** Innate immune training

Adaptive Immunity

- **Slower:** about 1–2 weeks the first time
- **Specific:** each cell recognises one specific target (antigen)
- **Cells:** B cells (make antibodies) and T cells (make cytokines)
- **Memory:** remembers the pathogen for years or a lifetime

Vaccines work by using the innate arm to switch on the adaptive arm, and creating immune memory

Innate Immunity: The Alarm System

1 Sensors detect danger

Pattern recognition receptors (e.g. Toll-like receptors) on dendritic cells and macrophages recognise microbial ‘danger signals’ (PAMPs): bacterial wall, viral RNA, unmethylated DNA

2 The alarm sounds

Signalling inside the cell switches on genes for interferons and inflammatory cytokines; the injection site becomes inflamed

3 Dendritic cells / APCs mobilise

Activated DCs and other antigen presenting cells mature and travel to the lymph node, carrying antigen to meet T and B cells

Why it matters: without an innate ‘danger’ signal, the adaptive response is weak. This is the job of adjuvants, and the reason live vaccines (and mRNA vaccines) are naturally immunogenic.

Adaptive Immunity: B Cells Make Antibodies

What happens to a B cell

1. **Recognise**

   A naive B cell with a matching receptor (co-express IgM and IgD) binds the antigen

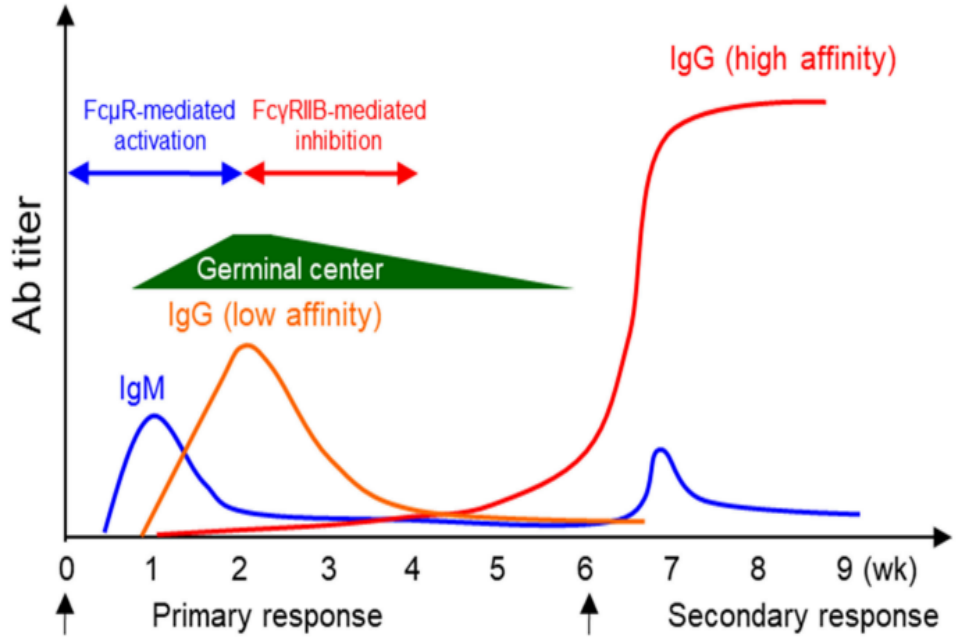
2. **Activate**

   With help from CD4 T cells it divides and matures in germinal centres and produces the appropriate Ab (class-switching)

3. **Differentiate**

   Plasma cells secrete antibodies immediately
   Memory B cells wait for the next exposure to the antigen

Second exposure: faster, bigger, better



Vaccine

Vaccine / Infection

Illustrative only. Later antibodies also bind more tightly (affinity maturation), which is why booster doses work.

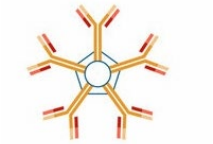
What Antibodies Do

Neutralise	Tag for eating (opsonise)	Call in killers
Bind the virus or toxin and block it from entering cells or acting	Coat the microbe so macrophages and neutrophils engulf it	Activate complement and NK cells (ADCC) to destroy microbes and infected cells

The main classes

IgM

First to appear; large pentamer



IgG

Main long-lived antibody in blood; crosses the placenta



IgA

Protects mucosal surfaces (gut, airway)



IgE

Allergy and parasites



T Cells: Coordinators and Killers

CD4 helper T cells

- **Recognise:** longer peptides (about 12–25 amino acids) on HLA class II expressed by APCs
- **Action:** help B cells make high-quality antibodies; produce cytokines to shape the response (Th1, Th2, Th17); activate CD8 T cells and macrophages
- **Activated by:** almost every vaccine, including conjugates

CD8 killer T cells

- **Recognise:** short peptides (about 8–11 amino acids) on HLA class I, found on amost every nucleated cell
- **Action:** find and kill infected cells
- **Activated by:** viral infections and live vaccines in particular; limit disease severity (e.g. shingles, COVID-19, measles)

Both subsets develop into short acting effector cells and long-lasting memory T cells after activation. People differ in their HLA genes, so T cell responses vary between individuals and populations.

From Injection Site to Lymph Node

Vaccination	Dendritic Cell / APC	Lymph node	Presentation
Vaccine antigen enters muscle or skin	Antigen presenting cells take up antigen; chop proteins into peptides	Mature DC/APC travels to LN	APCs show peptides on HLA (MHC) to T cells B cells bind intact antigen that has travelled in lymphatic system to the LN

T cells see fragments

A T cell cannot recognise a whole pathogen. It only sees short peptides displayed on HLA molecules, so the antigen must first be processed by an antigen-presenting cell.

B cells see the whole antigen

B cells recognise native vaccine antigen directly through their surface receptor, which is an antibody on the cell's surface. They then need help from T cells to make a strong response.

What a Vaccine Does: Putting It Together

Hours	Days	1–3 Weeks	Weeks–Months	Months-Years
Innate alarm	**Dendritic cells**	**T and B cells**	**Memory**	**Natural infection**
Sensors detect the vaccine; inflammatory cytokines (IL-6, TGF), type 1 interferons	Carry antigen to the lymph node	Activation in LN, germinal centres for Ab production T cell zone (paracortex) for T cells	Plasma cells in bone marrow; memory B and T cells in LNs and spleen	Memory responds in hours to days, to fight the infection

Protection depends on B cells, antibody and T cells working together to create immune memory, with antibody the best-established correlate of vaccine-induced protection.

Types of Vaccine: Live and Non-Live

	Live attenuated	Non-live (inactivated, subunit, toxoid, conjugate)
Examples	MMR, varicella, BCG, yellow fever, rotavirus	Influenza, hepatitis B, tetanus and diphtheria toxoids, pneumococcal and meningococcal conjugates
What it is	Weakened organism that replicates in the body	Killed organisms or parts of them (protein, toxoid) that cannot replicate
Immune response	Resembles natural infection; strong innate, antibody and T cell response	Needs an adjuvant or conjugation; mainly antibody, T cell response varies
Doses	Often 1–2	Usually several, with boosters
Cautions	Avoid in severe immunocompromise and pregnancy; rarely causes disease	Safe in immunocompromise, though responses may be lower

Newer Vaccine Platforms

Platform	How it works	Examples
mRNA in lipid nanoparticle	Our cells make the antigen briefly; the particle also alerts the innate system	COVID-19 (Comirnaty, Spikevax); RSV (mRESVIA); mRNA influenza (FDA-approved Aug 2026, under TGA evaluation)
Recombinant protein + adjuvant	Purified antigen made in cell culture; adjuvant provides the danger signal	Shingrix, Arexvy (RSV), Nuvaxovid, Fluad
Virus-like particles	Empty protein shells that look like the virus but contain no genome	HPV (Gardasil), hepatitis B, R21 malaria vaccine
Viral vector	A harmless (replication incompetent) virus carries the gene for the antigen into cells	Several COVID-19 vaccines (ChAdOx1); Ebola (Ervebo)

Not every product listed is registered or funded in Australia; see TGA and the National Immunisation Program for current status.

mRNA Vaccines in Four Steps

1

Delivered

mRNA is packaged in a lipid nanoparticle and injected into muscle

2

Taken up

Muscle cells and dendritic cells take up the particles and release the mRNA into the cell

3

Antigen made

Host cell machinery reads the mRNA and makes the coded antigen (e.g. a spike protein) for a few days

4

Response

The antigen is presented to T and B cells; the mRNA itself is quickly degraded

Strengths

- Fast to design: quick to swap in a new sequence
- Strong antibody and T cell responses
- Lipid particle acts as a built-in adjuvant

Limitations

- Cold-chain needs; reactogenicity
- Rare myocarditis, mainly in young males
- Durability of protection varies by pathogen

2023 Nobel Prize in Physiology or Medicine: Katalin Karikó and Drew Weissman.

Adjuvants: Waking Up the Innate System

What they are

Substances added to a vaccine that provide the ‘danger signal’ purified antigens lack, stimulating innate immunity so the adaptive response is stronger.

What they can do

- Increase the magnitude of the response
- Improve its quality and durability
- Allow smaller antigen doses
- Overcome weaker responses in older adults

Antigen alone	Antigen + Adjuvant
Little innate activation, leading to a weak, short-lived adaptive response.	Strong innate activation, leading to a larger, longer-lasting response.

Live vaccines and mRNA vaccines have their own intrinsic adjuvant activity.

Which Adjuvants Are in Which Vaccines

Adjuvant	Type	Used in (examples)
Aluminium salts (alum)	Mineral salts; discovered 1926; most widely used; antibody-focused (Th2-biased)	DTP, hepatitis A/B, HPV, some pneumococcal conjugate vaccines
MF59	Squalene oil-in-water emulsion	Fluad (adjuvanted influenza vaccine for ≥65 year olds)
AS03	Squalene + α-tocopherol (vitamin E) emulsion	Pandemic influenza vaccines
AS01	MPL (TLR4 agonist) + QS-21 (saponin) in liposomes	Shingrix, Arexvy (RSV), RTS,S malaria
CpG 1018	TLR9 agonist	Heplisav-B (hepatitis B)
Matrix-M	Saponin-based nanoparticle	Nuvaxovid (COVID-19 vaccine), R21 malaria vaccine

How alum works is still debated (the role of the inflammasome is controversial). Not all products are available in Australia.

Why Mucosal Immunity Matters

The mucosal frontline

- **Entry points:** most pathogens enter through the airway, gut or genital tract
- **Huge surface area:** mucosal surfaces are the body's main contact with the outside world
- **Defences:** epithelium**,** mucous, secretory IgA (sIgA), tissue-resident T and B cells
- **IgA:** more is made each day than all other antibody classes combined
- **Role:** block attachment and neutralise microbes before they cross the epithelium
- **Mucosal vaccines** aim to stop infection at the entry point and reduce shedding

What injected vaccines achieve

- **Mainly serum IgG;** little sIgA or tissue-resident memory at the mucosa
- **Strong protection** against severe disease, as IgG reaches the lower airway
- **Weaker protection** against infection of the nose and throat, and onward spread
- **Examples:** COVID-19, influenza, acellular pertussis

Builds immunity where the pathogen presents, not only in the blood.

Sources: Lavelle EC, Ward RW. Nat Rev Immunol 2022;22:236–50; Russell MW et al. Front Immunol 2020.

How Mucosal Immunity Is Generated

1

Antigen Uptake

M cells (in MALT) and DCs take up antigen across the epithelium often at specific sites: Peyer's patches (gut), nasopharynx-associated lymphoid tissue and tonsils (airway)

2

Prime and Switch

T and B cells are activated in the local lymphoid tissue. Naive B cell class switching to IgA production involves TGF-β, retinoic acid, cytokines and T follicular helper cells

3

Home

Primed cells leave in lymph and blood, then return to mucosal tissue using homing receptors (e.g. α4β7 and CCR9 for the gut)

4

Protect

Plasma cells secrete dimeric IgA (blood and body fluids); secretory IgA = specialised IgA on epithelial surfaces and in body secretions. Tissue-resident memory CD4 and CD8 T cells reside in epithelium and lamina propria

Compartmentalised: oral vaccines best induce gut immunity; intranasal or inhaled vaccines best induce airway immunity. Some cross-talk occurs between mucosal sites, but it is partial.

Sources: Lavelle EC, Ward RW. Nat Rev Immunol 2022; Mowat AM. Nat Rev Immunol 2003; Brandtzaeg P. Front Immunol 2013.

Oral and Nasal Vaccines in Use

Vaccine	Route and type	Notes
Rotavirus	Oral; live attenuated	Induces gut IgA; widely used
Polio (OPV)	Oral; live attenuated	Gut immunity limits shedding; rare vaccine-derived disease; nOPV2 more stable
Cholera	Oral; killed or live attenuated	Gut IgA; used for travellers and outbreak control
Typhoid (Ty21a)	Oral capsules; live attenuated	Gut and systemic responses; travellers
Influenza (LAIV)	Nasal spray; live attenuated	Nasal IgA and T cells
COVID-19	Inhaled or nasal; adenovirus-vectored	Licensed in China, India and some other countries; not in Australia

Mostly live attenuated: replication mimics natural infection, so there is strong innate and mucosal stimulation without needing an adjuvant.

Availability in Australia changes: check the TGA register and the NIP schedule. Sources: Australian Immunisation Handbook; WHO position papers; Lavelle EC, Ward RW. Nat Rev Immunol 2022.

LAIV: How a Nasal Flu Vaccine Works

Design

- Live virus, cold-adapted, temperature-sensitive, attenuated
- Grows in the cool nose (about 25–33 °C), but poorly in the warm lung (37 °C)
- Needle-free spray into each nostril
- HA and NA match the season's strains

Immune response

- Interferon and innate activation in the nasopharynx
- Local sIgA plus serum antibody
- CD4 and CD8 T cells, including tissue-resident cells

Limits

- Generally equivalent protection compared to inactivated vaccine in young children
- Variable effectiveness by season and strain
- Avoid in pregnancy, severe immunocompromise
- Pre-existing immunity limits replication in adults

No accepted correlate of protection: serum HAI underestimates LAIV; mucosal IgA and T cells are harder to measure.

Sources: Belshe RB et al. N Engl J Med 2007;356:685–96; Australian Immunisation Handbook; CDC ACIP influenza recommendations.

Mucosal Vaccines: Challenges and What's Next

Challenges

- **Hostile environment:** acid, enzymes, mucus and rapid clearance
- **Tolerance:** the gut ignores harmless antigens, so non-live antigens need adjuvants or delivery systems
- **Safety history:** e.g. a nasal flu vaccine + toxin adjuvant linked to Bell's palsy (2000-01) and withdrawn; RotaShield and intussusception
- **Measurement:** no routine assays for mucosal IgA or tissue-resident memory T cells

What's next

- **Prime–pull:** injected prime, mucosal boost to 'pull' immunity into tissue
- **Candidates in trials:** inhaled and nasal COVID-19, RSV, influenza, and live attenuated pertussis (BPZE1), TB (inhaled BCG and other candidates)
- **Better tools:** mucosal adjuvants, nanoparticle and mRNA delivery

Promising for blocking infection and transmission, but still few licensed products.

Sources: Mutsch M et al. N Engl J Med 2004;350:896–903; Lavelle EC, Ward RW. Nat Rev Immunol 2022; Thorstensson R et al. PLoS One 2014 (BPZE1).

Why Vaccine Responses Vary Between People

Age	Sex	Genes (HLA)
Immature in infants; immunosenescence in older adults (hence adjuvanted and high-dose vaccines)	Females often make higher antibody responses and report more vaccine reactions	Determine which peptides T cells can see

Microbiome and infections	Medicines and health	The vaccine
Gut microbiota and chronic infections such as CMV can modulate responses	Immunosuppression, steroids, chemotherapy, chronic disease, nutrition	Antigen dose, adjuvant, route, schedule and spacing between doses

Sex Differences in Vaccine Responses

Antibody

- Females often make higher titres: influenza, hepatitis A and B, MMR, yellow fever, rabies
- Most prominent between puberty and menopause
- Half-dose influenza vaccine in women matched full dose in men

Reactions

- Females report more local and systemic reactions
- Anaphylaxis is reported more often in women (e.g. mRNA COVID-19 vaccines)
- Myocarditis after mRNA vaccines is more common in young males

Protection

- More antibody does not always mean better protection; results of studies investigating vaccine effectiveness by sex are mixed

Differences are real but modest and variable: a reason to analyse by sex, not (yet) to change schedules.

Sources: Klein SL, Flanagan KL. Nat Rev Immunol 2016;16:626–38; Flanagan KL et al. Annu Rev Cell Dev Biol 2017;33:577–99; Engler RJ et al. Arch Intern Med 2008;168:2405–14.

Why Do Responses Differ by Sex?

Biology (Sex)

- **Sex hormones** Oestrogen boosts B and T cell responses and interferon signalling; testosterone generally dampens immunity
- **Life stage:** puberty, pregnancy and menopause change the hormone environment
- **X chromosome:** many immune genes (e.g. TLR7, CD40L, FOXP3); some escape X-inactivation, so females may express more
- **Microbiome:** differs by sex and interacts with hormones

Gender and Context

- **Dose per kg:** fixed adult doses mean smaller people, on average women, get more vaccine per kilogram
- **Reporting:** women may be more likely to seek care and report reactions
- **Exposure:** occupation, caring roles, behaviour and prior infection differ
- **Evidence gap:** trans and gender-diverse people are rarely studied

Implications: report trial data by sex, study dose and schedule, and track responses across puberty, pregnancy and menopause.

Sources: Klein SL, Flanagan KL. Nat Rev Immunol 2016; Flanagan KL et al. Annu Rev Cell Dev Biol 2017; Fischinger S et al. Semin Immunopathol 2019;41:239–49; Heidari S et al. Res Integr Peer Rev 2016 (SAGER).

Immunity Changes Across the Lifespan

Michelle Giles

Pregnancy

IMMUNE FEATURES

Immune system adapted to tolerate the fetus; higher risk of severe infections. Maternal IgG crosses the placenta to protect the baby.

VACCINE STRATEGY

Vaccinate the mother to protect both mother & child: dTpa, influenza, RSV (+/-COVID-19).

Infants and children

IMMUNE FEATURES

Little immunological memory; maternal antibody wanes; polysaccharide antigens work poorly under 2 years.

VACCINE STRATEGY

Early multi-dose schedule, conjugate vaccines, boosters.

Julia Brotherton
Helen Marshall

Adolescents

IMMUNE FEATURES

Strong responses; new exposure risks (HPV, meningococcus); childhood pertussis immunity wanes.

VACCINE STRATEGY

School program: HPV, dTpa, MenACWY.

Adults

IMMUNE FEATURES

Peak immune function, but immunity wanes; occupation, travel and medical risks.

VACCINE STRATEGY

Boosters, catch-up, risk-based vaccines.

Katie Flanagan

Older adults

IMMUNE FEATURES

Immunosenescence and inflammaging; high risk of severe disease.

VACCINE STRATEGY

Adjuvanted or high-dose vaccines, extra doses, age-based recommendations.

Match the vaccine strategy to the immune system at each life stage.

The NIP Across the Lifespan

Life stage	Funded on the National Immunisation Program (Australia, 2026)
Pregnancy	dTpa (ideally 20–32 weeks); influenza (any stage); RSV vaccine Abrysvo (from 28 weeks). COVID-19 may be considered from 20 weeks
Birth to 6 months	Hepatitis B at birth; 2, 4 and 6 months: hexavalent (diphtheria, tetanus, pertussis, hepatitis B, Hib, polio) and rotavirus; PCV20 at 2 and 4 months; MenB for First Nations children
12–18 months	12 months: MMR, MenACWY, PCV20. 18 months: MMRV, DTPa, Hib. Annual influenza funded from 6 months to under 5 years
4 years	DTPa-IPV booster
School (Years 7–10)	Year 7: HPV (single dose) and dTpa. Year 10: MenACWY
Adults	Influenza annually if aged 65 or over, pregnant or medically at risk; other vaccines by risk (not all funded)
Older adults	From 65: influenza (adjuvanted), shingles (Shingrix), COVID-19 (from 01/10/26). From 70: PCV21. From 75: RSV (Arexvy, from 15/05/26). First Nations: PCV21 from 25, shingles and COVID-19 from 50, RSV from 60

Sources: Dept of Health NIP Schedule (current from October 2026); Australian Immunisation Handbook. Check the current schedule — it changes often.

Efficacy, Effectiveness and Impact

Efficacy

Benefit in a randomised controlled trial, under ideal conditions, most in healthy individuals.

Effectiveness

Benefit in routine use, in the real-world population.

e.g. influenza vaccine, usually 40–60% depending on the season

Impact

Effect on disease across the whole population, including herd (indirect) protection.

e.g. HPV vaccine impact on cervical cancer rates

Vaccine efficacy (VE) = (1 – risk in vaccinated ÷ risk in unvaccinated) × 100

Example: 10 cases per 1,000 vaccinated vs 100 per 1,000 unvaccinated.
Risk ratio 0.1, so VE = 90%: **90% fewer cases than expected without the vaccine.**

How We Measure Vaccine Effectiveness

Design	How it works	Strengths and pitfalls
Randomised trial	Randomly assign vaccine or control; compare disease	Gold standard for efficacy; costly; often not ethical once a vaccine is proven protective
Cohort	Follow vaccinated and unvaccinated people over time	Estimates risk directly; confounding and ‘healthy vaccinee’ bias
Case-control	Compare vaccination status of cases and matched controls	Efficient for rare diseases; control selection is critical
Test-negative	Among symptomatic people tested, compare vaccination in test-positive vs test-negative	Handles care-seeking bias; standard for influenza, COVID-19, rotavirus
Population impact	Compare disease rates before and after a program	Captures herd effects; cannot prove cause on its own

Common pitfalls: confounding, waning protection, changing strains, and how the outcome is defined (infection vs severe disease), underascertainment.

Correlates of Protection

What is a correlate of protection?

An immune marker, measurable in blood, that statistically predicts whether a vaccinated person will be protected from disease. Generally based on antibody levels.

- **Mechanistic:** the marker itself protects (e.g. antitoxin, anti-capsular antibody)
- **Non-mechanistic:** a proxy for the true protective response (e.g. measles antibody)

Why they matter

- Checking individual immunity (e.g. hepatitis B antibody in health workers)
- Licensing by immunobridging: show a new vaccine gives the same antibody level as a proven effective one, without a new efficacy trial (e.g. pneumococcal vaccines)
- Annual influenza strain updates, new conjugate valencies, some boosters
- Setting schedules and booster intervals

Caution: a correlate is not the same as the mechanism. It depends on the assay, the outcome (infection, disease or severe disease) and sometimes the strain, so thresholds are guides, not guarantees.

Established Correlates of Protection

Vaccine	Marker	Protective level (indicative)
Hepatitis B	Anti-HBs antibody	≥ 10 mIU/mL
Tetanus and diphtheria	Antitoxin	0.01–0.1 IU/mL (0.1 = full protection)
Measles	Neutralising antibody	≥ 120 mIU/mL
Rubella	Antibody	≥ 10–15 mIU/mL
Hib (conjugate)	Anti-PRP antibody	≥ 0.15 µg/mL short term; ≥ 1 µg/mL long term
Pneumococcal conjugate	Serotype IgG (ELISA)	0.2–0.35 µg/mL (children, invasive disease)
Influenza (inactivated)	Haemagglutination inhibition	Titre ≥ 1:40 ≈ 50% protection
Polio	Neutralising antibody	Titre ≥ 1:8
Rabies	Neutralising antibody	≥ 0.5 IU/mL

Sources: Plotkin SA. Clin Vaccine Immunol 2010; Plotkin SA. Front Immunol 2023. Thresholds are assay-specific and indicative only.

When There Is No Simple Correlate

Undefined correlates

- **Tuberculosis:** no validated correlate
- **Whooping cough:** no agreed threshold
- **Malaria, HIV, CMV:** partial or no correlate
- **COVID-19:** antibody titre correlates with protection, but there is no universal threshold and it shifts with variants
- **RSV:** higher neutralising antibody is linked to lower risk; no accepted threshold
- **Shingles:** cell-mediated immunity matters but is hard to measure routinely

What we do instead

- Large phase 3 efficacy trials with clinical endpoints
- Real-world effectiveness studies after rollout
- ‘Correlates of risk’: markers linked to risk without a clear threshold
- Controlled human infection (challenge) studies
- Systems vaccinology to find immune signatures of protection
- Measure more than antibody: T cells, mucosal IgA, memory B cells

What We Still Don’t Fully Understand

Open questions

- Many vaccines were developed empirically; how BCG and yellow fever vaccines work is still incompletely understood
- For many diseases we lack a reliable correlate of protection (antibody is the main one we can measure)
- The old rule ‘live = Th1, inactivated = Th2’ is too simple in humans
- Wider immunomodulatory (non-targeted) effects of some vaccines are apparent but poorly investigated

Systems vaccinology

Genomics, transcriptomics, proteomics and immune profiling, combined with computational modelling, show what vaccines do to the whole immune system.

- Predict responses early (yellow fever vaccine signature)
- Define correlates of protection
- Compare adjuvants and platforms
- Explain safety signals and reactogenicity
- Understand differences by age and sex

What's New and What's Next?

First mRNA flu vaccine

FDA approved mFLUSIVA (mRNA-1010) for adults ≥50 in Aug 2026; 26.6% higher relative efficacy than standard-dose vaccine

Personalised cancer vaccine

Intismeran + pembrolizumab met its primary endpoint in a phase 3 melanoma trial

Self-amplifying mRNA

Licensed for COVID-19 in Japan and the EU (Kostaive); lower doses may be possible

Combination vaccines

Flu + COVID-19 mRNA combinations in late-stage development or review

New Vaccines

R21/Matrix-M malaria vaccine being rolled out in Africa; M72/AS01E TB vaccine in phase 3; GBS vaccines; Norovirus vaccines

Better delivery

Needle-free and microneedle patches, thermostable formulations, mucosal vaccines in trials

Sources: company and FDA announcements and trade press, Aug–Oct 2026. Results are topline or regulatory status in the USA; check TGA for Australia.

SUMMARY

Take-Home Messages

1. Vaccines train the adaptive immune system safely to create immune memory
2. The innate system determines how strong the response is, which is why adjuvants and live or mRNA platforms work so well
3. Antibodies, CD4 and CD8 T cells work together; antibody is the best-established correlate of protection
4. Live vaccines mimic infection; non-live vaccines need adjuvants, conjugation and boosters
5. Match the strategy to the life stage: vaccinate mothers to protect newborns; use adjuvanted or high-dose vaccines in older adults
6. Efficacy comes from trials; effectiveness and impact from real-world studies
7. Correlates of protection let us license and monitor vaccines, but many diseases still lack one
8. New platforms and technologies (mRNA, protein + adjuvant, VLPs) are expanding what vaccines can do, even to cancer