



From Polarization and Slogans to Common Sense and Science

Natural Immunity, Vaccination and Innate Immune Training Can Coexist

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Why this topic belongs at a Peace and Health conference

Debates about health have become a source of social conflict and have escalated dramatically since the Covid-19 crisis.

Trust, relationships, businesses, education but also dignity, trust, respect, integrity, solidarity, sovereignty and freedom can be damaged when health policy becomes coercive.

Even debates in medicine and science became increasingly offensive

Action
↔
Reaction

Polarization

Meaningful dialogue on *complex* issues collapses (e.g., climate change, migration & integration, AI, geopolitical dynamics, origins & response to SARS-CoV-2 pandemic)



Another highly polarized scientific and public health issue: Vaccination.

The COVID-19 crisis sharpened the conflict between pro- and anti-vaccine positions and reduced the vaccine debate to *'pro-vaccine' vs 'anti-vaccine'* without any room left for *scientific nuance*.

Instead, complex immunological phenomena and population-level dynamics are reduced to slogans. The conflict between pro- and anti-vaccine positions has become highly sensitive, both socially and politically and is ***largely rooted in a poor understanding of immunology***, particularly of two deceptively simple questions:

- **how and when does natural immunity work or fail**
- **how and when can vaccination work or fail?**

A serious scientific discussion must therefore move beyond ideological labels and return to mechanism, context and risk-benefit analysis.

From binary slogans to context-dependent biology

The COVID-19 crisis sharpened an older divide in vaccinology: “pro-vaccine” versus “anti-vaccine”.

Binary debate

- Vaccination is always good
- Vaccination is always bad
- Natural immunity is primitive
- Natural immunity is always sufficient

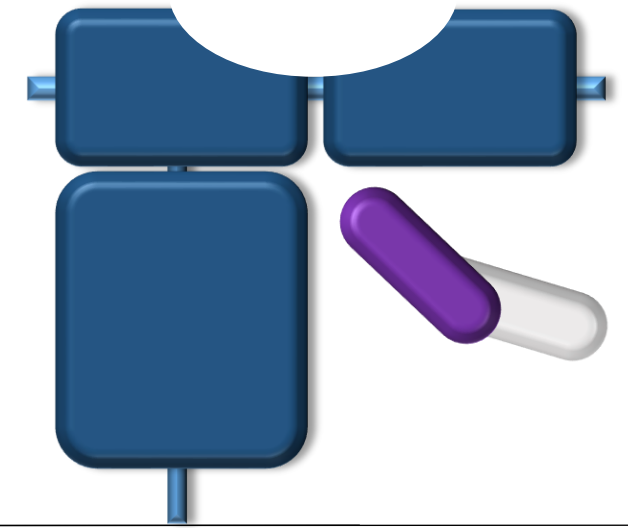
shift

Context-dependent biology

- Which pathogen?
- Which host immune status?
- Which epidemic phase?
- Does immunity block infection and transmission, or only disease?
- Does the benefit outweigh the risk?

For pandemics/ epidemics of certain acute, self-limiting viral infections (**ASLVIs**), *natural immunity* may be the more appropriate route toward protection, whereas *vaccination* can be highly valuable for maintaining immune protection after epidemics caused by other ASLVIs.

Why current vaccine approaches fail to reach the high-hanging fruit?



VACCINES RESEARCH AND DEVELOPMENT
November 18-20, 2019
Boston, USA

G. Vanden Bossche, DVM, PhD
Consultant Vaccine Research Collaborations

*I see vaccines for all human and animal beings, and vaccines
preventing and treating infectious, malignant or immune-mediated diseases
And I think to myself.....
What a wonderful world!*





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Autoimmune/Inflammatory Syn... x

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5256113/

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doi: 10.3389/fendo.2016.00150

Autoimmune/Inflammatory Syndrome Induced by Adjuvants and Thyroid Autoimmunity

Abdulla Watad,^{1,2,3} Paula David,^{2,4} Stav Brown,³ and Yehuda Shoenfeld^{2,3,5,*}

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J Autoimmun. 2013 Dec;47:1-16. doi: 10.1016/j.jaut.2013.10.004. Epub 2013 Nov 13.

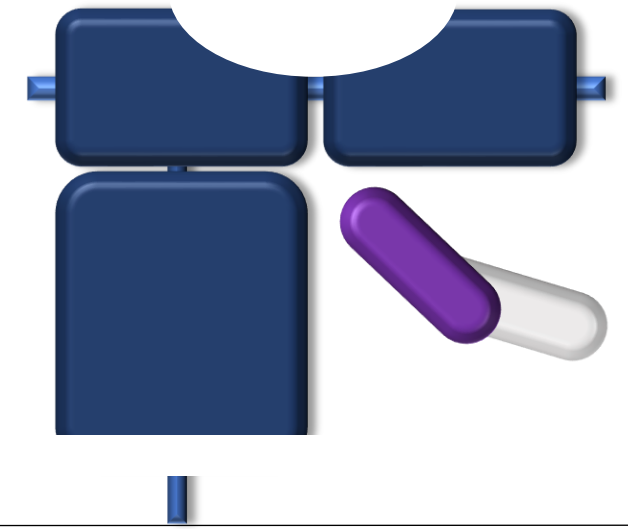
Autoimmune/inflammatory syndrome induced by adjuvants (ASIA) 2013: Unveiling the pathogenic, clinical and diagnostic aspects.

Perricone C¹, Colafrancesco S, Mazor RD, Soriano A, Agmon-Levin N, Shoenfeld Y.

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The major issue of contemporary vaccinology: Lack of understanding of pathogen – host *immunity* interactions



- Immunologists have learned a lot from vaccinologists but true learnings have rarely occurred the other way around
- Current 'progress' is largely based on *new technologies* and techniques including gene-based immunization and big data mining (-omics) rather than on a *fundamental understanding of host-pathogen interactions and immune subversive mechanisms which pathogenic agents have evolved to escape natural mechanisms of host immune defense*
- Increasing number of 'off target' studies and exercises in molecular stamp collection are slotting components into increasingly complex biochemical/ molecular pathways
- Increasing capacities, complex technologies and big data:
Anyone still sees the forest for the trees?

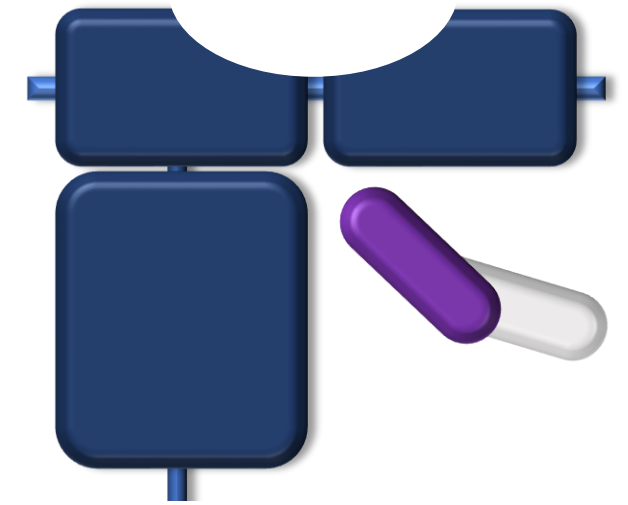


'Novel' vaccine approaches: An old lady in a *modern* dress?

Novel & modern? Yes, of course!.

- Structural & computational modeling/ biology
- Bioinformatics
- Systems Biology
- Characterization of immune signatures/ signaling pathways
- Inventories of adjuvant signatures/ microarrays
- The era of -omics and data mining
- Development of exciting CAR-T cells & checkpoint inhibitors; *nucleic acid-based vaccines*
- Personalized vaccinology
- *Artificial intelligence*

Ex. :“Human Vaccines Project” (to accelerate the development of vaccines and immunotherapies against major global infectious diseases and cancers *by decoding the human immune system*)

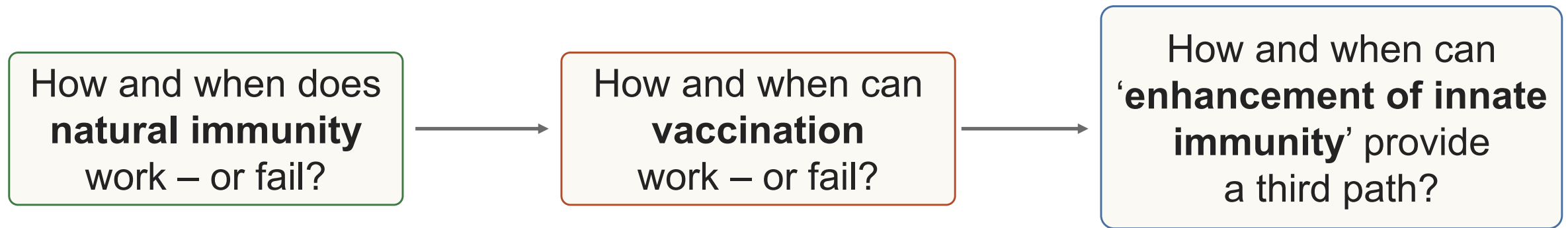


Despite all these efforts, vaccinology still remains an *empirical* business (*trial and error*) with poor understanding of individual and population-level immunity!





The real divide between pro-vax and anti-vax scientists is rooted in poor immunological understanding



The fight is not resolved by asking who is “pro-vax” or “anti-vax”; it is resolved by asking *which immune mechanism has the best risk-benefit profile in which biological and population-level context.*



3 routes to protect against overt disease: which one to choose, and when?

1. Natural immunity

- Immunization through natural infection (ASLVI)
- If protective **HI** is **built**
- Safety risk: case fatalities

2. Prophylactic vaccination

- Immunization independent of natural infection
- if protective **HI** is **preserved**
- Safety risk: adverse effects

3. Innate immune enhancement

- Immunization independent of both natural infection and vaccination
- **no HI involved**
- *No well-founded safety concerns*

HI: Herd immunity

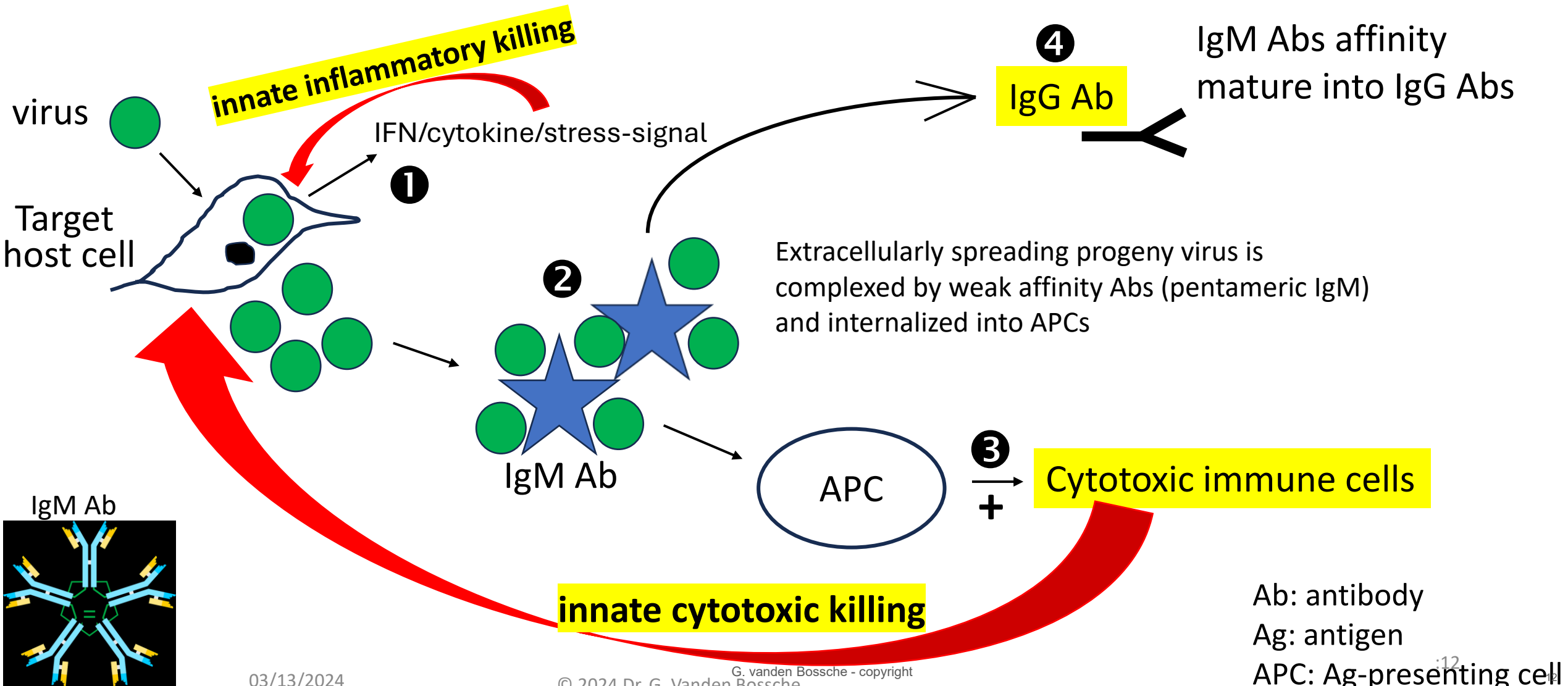
Protective HI illustrates the power of natural infection-primed immunity

To resolve a natural ASLVI pandemic, immunity must do more than reduce overt disease. It must evolve into a *collective immune response that sufficiently reduces infection and onward transmission to prevent sustained viral spread = HI*.

But why can naturally evolving ASLVI epidemics or pandemics eventually generate *large-scale transmission control* (= HI), whereas *large-scale vaccination* campaigns during pandemics/ epidemics fail to do so, even when high antibody (**Ab**) titers are induced?

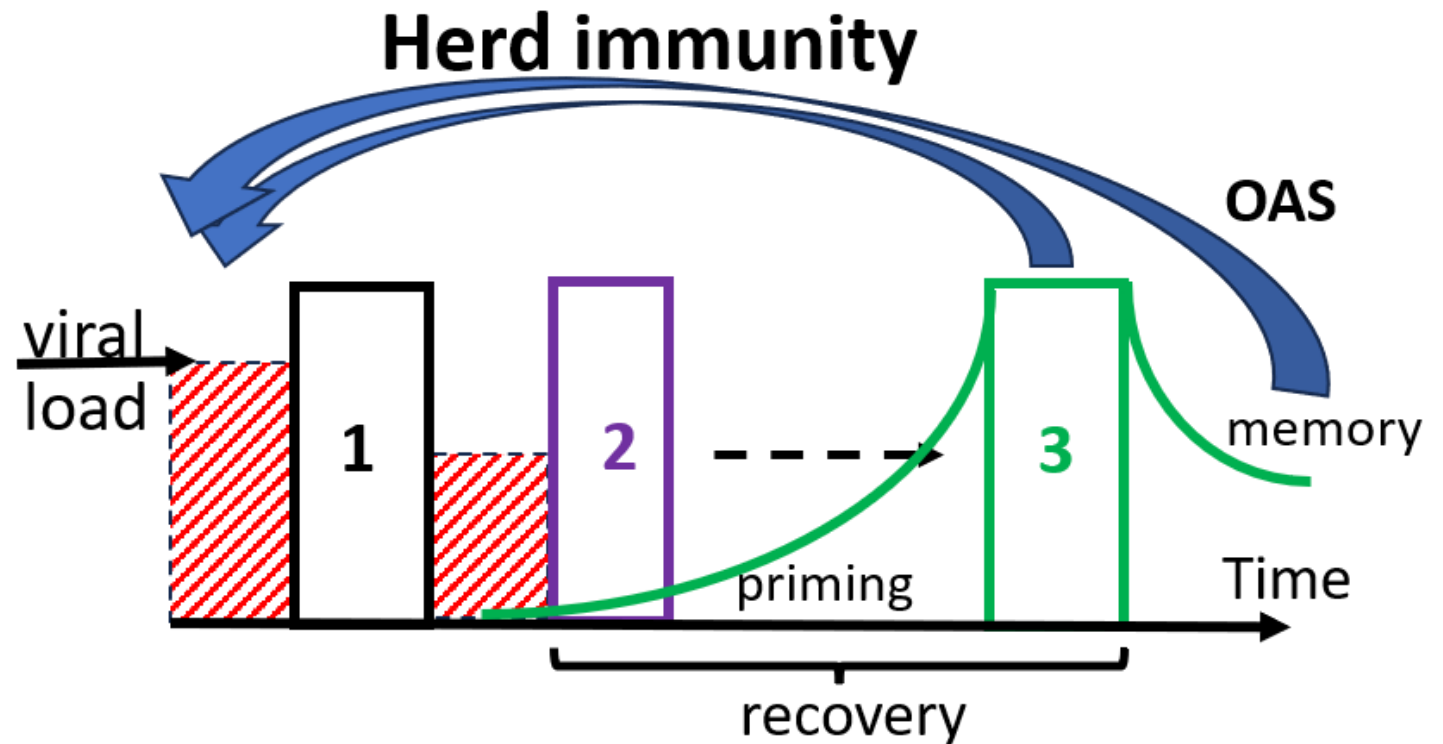
The answer is that infection-primed immunity engages a broader, sequential immune response than vaccine-primed immunity.

ASLVI generates sterilizing immunity & curtails infectious shedding



Immunization through natural infection

ASLVI of immunologically naïve individuals leads to sterilizing immunity and contributes to HI



1: **innate** inflammatory killing of infected cells

2: **innate** cell-mediated viral clearance

3: **Ag-specific virus-neutralizing Abs (NAbs)**

 : viral load

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Scenario 1: natural immunity can work during ASLVI pandemics



Natural immunity induced by ASLVIs spreading across a sufficiently large proportion of the population can generate *collective protection* against infection – i.e., *protective herd immunity*.

Importantly, this *collective* state of protection cannot be reduced to the sum of *individual* antibody titers!



Natural infection: the safety concern/objection is legitimate

Awaiting herd immunity can cost lives, especially among elderly, immunocompromised or otherwise vulnerable individuals.



This concern must be acknowledged, not dismissed.

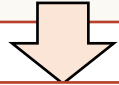
The question is *not* whether natural infection is risk-free. It is whether the biological cost can be reduced enough to justify reliance on natural immunity in a specific context.



Safety risk mitigation strategy: different viruses require different strategies

Do NOT “await herd immunity” when...

- case-fatality rate is high
- severe disease appears rapidly
- vulnerable groups cannot be protected
- *highly pathogenic viruses*: SARS-CoV-1, MERS



infection-prevention strategies!

More defensible when...

- virus is more transmissible but less pathogenic (e.g., SARS-CoV-2)
- severe disease is concentrated in identifiable vulnerable groups
- the biological cost of reaching HI is actively reduced (e.g., by reducing infectious pressure and early treatment).

Natural immunity is not anti-vax slogan; it is a context-dependent population-level strategy that is based on a sound scientific rationale.



Further safety risk mitigation: lower the price paid for HI

Reduce viral infectious pressure

- avoid *large* gatherings and crowding

Protect vulnerable individuals

- targeted measures/ shielding of immune-suppressed people without paralyzing society

Treat early

- antiviral, anti-inflammatory, anti-thrombotic

Promote innate immune resilience

- public-health messaging on healthy lifestyle

These ‘soft’ flanking measures do not prevent HI from developing; they reduce the biological cost of getting there.



Residual safety risk: why opponents retain a strong argument



Because severe disease and case fatalities cannot be ruled out, opponents will argue that at least vulnerable people (e.g., elderly in nursing homes) should be vaccinated when vaccines are available or could be made available.

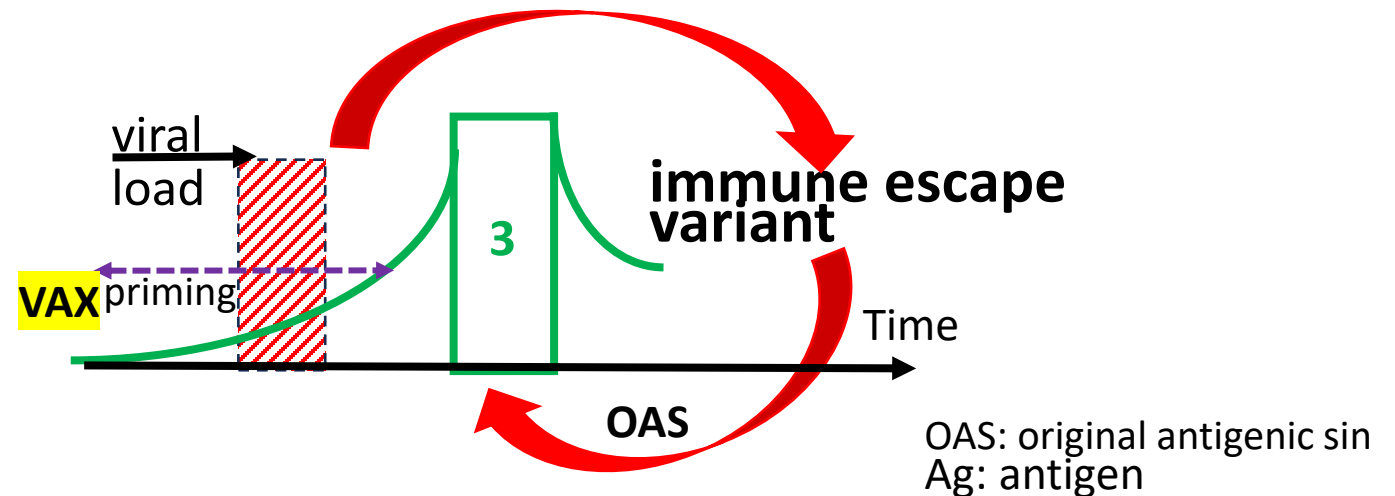
Vaccination during an ongoing ASLVI-driven pandemic/ epidemic raises serious efficacy and safety concerns...

Viral exposure of vaccinees with pre-existing suboptimal neutralizing Abs (NAbs) inevitably leads to viral immune escape



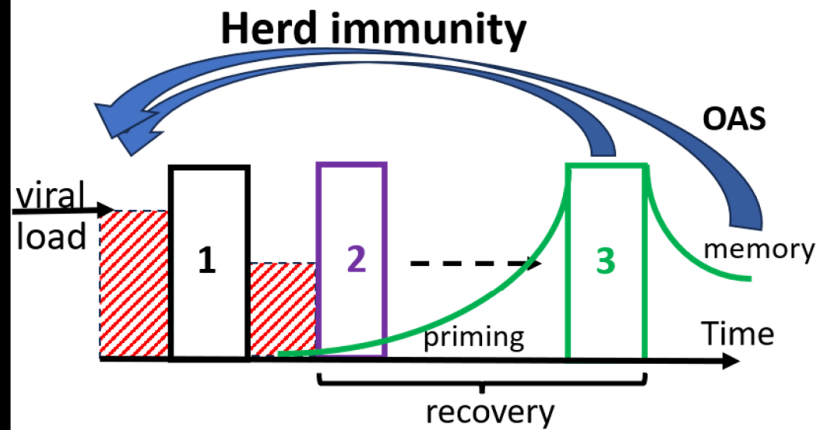
vaccine-primed NAbs

Immune selection pressure



SUBOPTIMAL neutralization by vaccinal NAbs

Immunization through natural infection



- 1: innate inflammatory killing of infected cells
- 2: innate cell-mediated viral clearance
- 3: Ag-specific virus-neutralizing Abs (NAbs)

red hatched box : viral load

Natural exposure versus vaccination during intense viral circulation

Natural exposure

low viral load when NAbs arise

1

Innate
inflammatory
viral
containment

2

Innate cell-
mediated
viral
clearance

3

IgG NAbs
priming

Vaccine-priming during circulation

immune selection pressure upon viral exposure

3

Suboptimal
innate
stimulation
upon
exposure

Suboptimal
NAbs

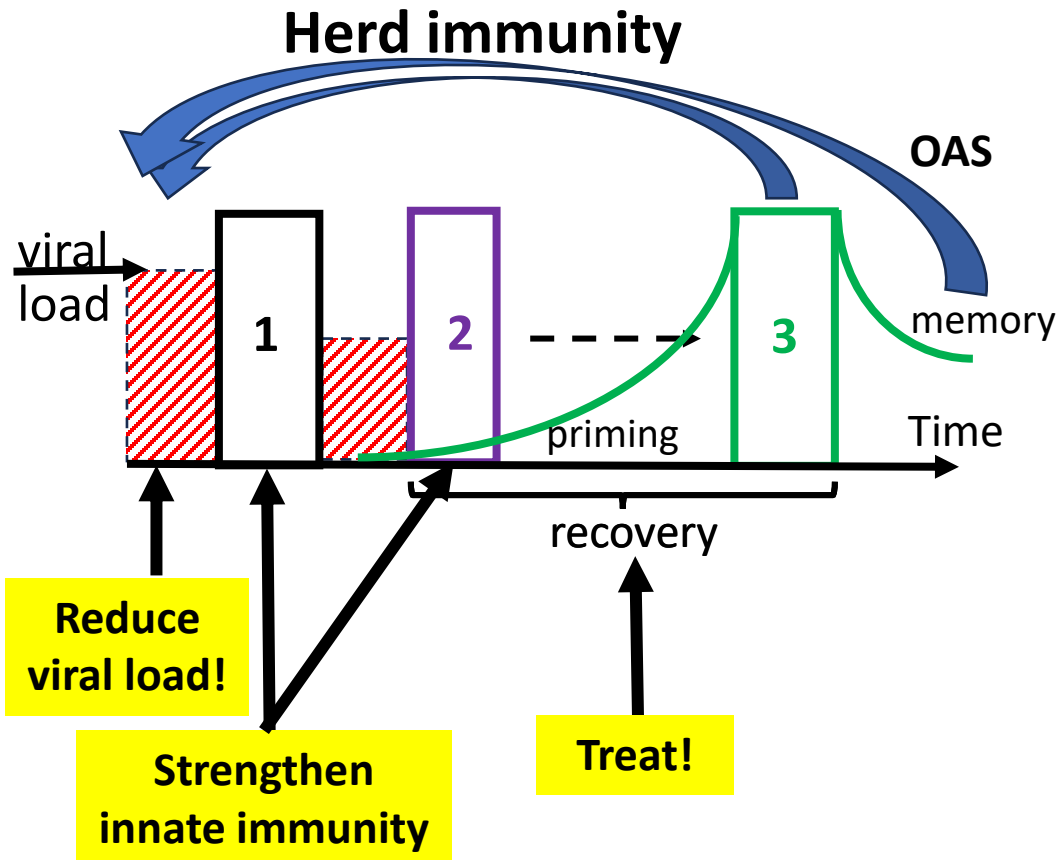
immune
escape
variants

Key distinction: **immune control of viral transmission** versus **immune pressure on viral transmissibility**

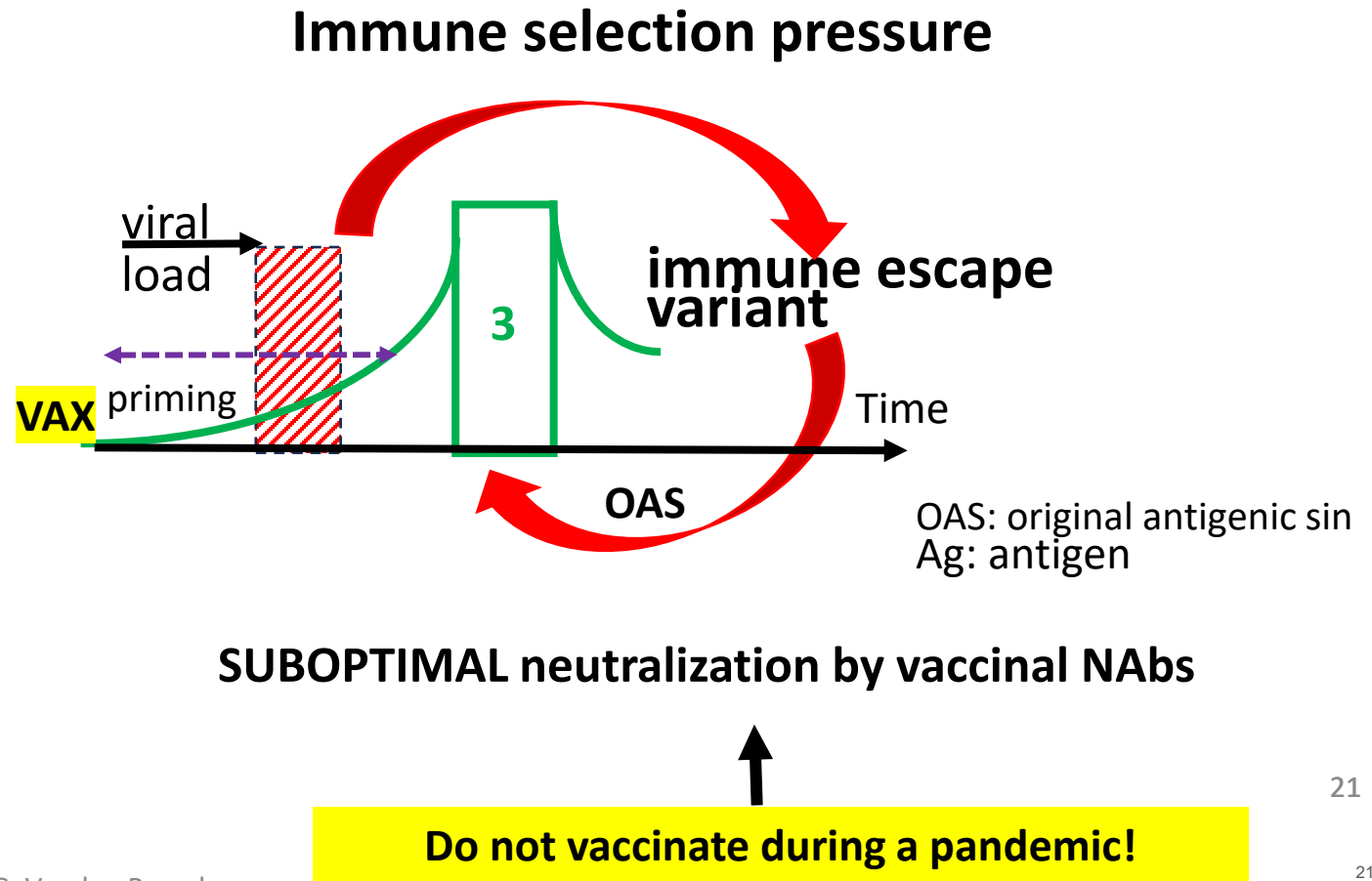
Viral exposure of vaccinees with pre-existing suboptimal neutralizing Abs (NAbs) inevitably leads to viral immune escape

03/13/2024

infection-primed NABs



vaccine-primed NABs



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From antibody thinking to protective herd immunity

A central blind spot is the assumption that stronger individual Ab titers automatically translate into stronger population-level protection, *even when the collective immune defense mounted by these Abs does not reliably prevent onward transmission.*

Any immunization strategy that elicits an antigen-specific adaptive immune response without effectively controlling transmission can be deceptively reassuring: it may protect against severe disease while simultaneously driving viral immune escape!

Failure

Individual protection

- may reduce severe disease
- may depend on Ab titer, affinity and memory
- does not necessarily prevent infection or transmission
-  may cause ADEI!

Transmission control

- requires reduction of infection and onward spread
- depends on the collective *immune response, viral load and timing*
- *is the **functional basis of protective herd immunity***

Failure

Immune selection pressure

- arises when virus still circulates under non-sterilizing immunity
- favors immune escape variants able to replicate or transmit despite prevailing immunity

Vaccination during exposure: the safety objection is legitimate!

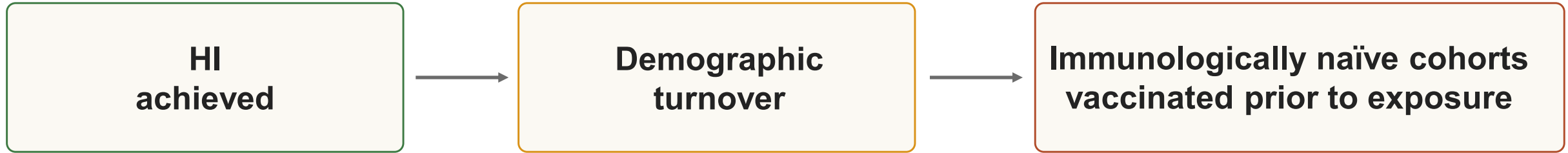
- non-sterilizing immunity can coexist with high viral replication
- immune selection pressure is imposed while transmission continues
- ***immune escape*** variants are selected to preferentially propagate
- Immune escape variants increasingly subvert the host immune system and may eventually sideline protective immune responses → ***efficacy & safety concerns!***



This concern must be acknowledged, not dismissed.

Vaccination policy should be guided not by ideology, empiricism, or the naïve belief that ‘more antibodies are better,’ but by whether **the immune-protection strategy can interrupt transmission in the relevant host–pathogen ecosystem.**

Scenario 2: Prophylactic vaccination after an epidemic may help preserve HI while mitigating the risk of vaccine-driven immune escape

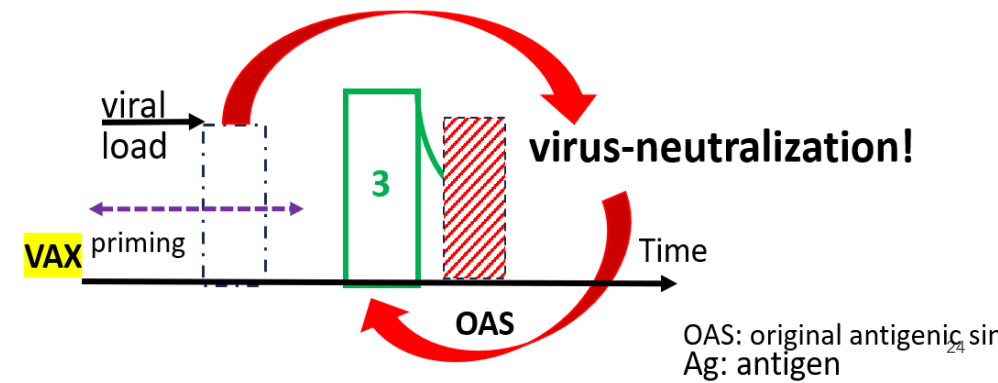


Pre-exposure vaccination of *immunologically naïve individuals* is most defensible when it helps preserve already established HI as it gradually erodes

vaccine-primed NAb

Immune selection pressure

Conclusion:
The same vaccine-induced response can have different consequences depending on epidemic phase, viral transmission intensity and immune status of the population.





Safety risk mitigation strategy: different viruses require different strategies

Strongest case for prophylactic vaccination

- antigenically stable virus
 - little/no asymptomatic transmission
 - no relevant animal reservoir
 - neutralizing immunity interrupts transmission
 - serious disease risk in susceptible children or immunocompromised persons
- Ex.: Measles

Problematic for large-scale prophylaxis

- high antigenic variability
- aerosol or asymptomatic spread
- animal or persistent reservoirs
- ongoing circulation despite Abs
- rapid immune escape risk

Ex.: Influenza, Corona viruses



Residual safety risk: why opponents retain a strong argument

Vaccinating young & healthy children can cause adverse effects in individuals who might otherwise remain healthy.



This concern must be acknowledged by vaccinologists.

A defensible vaccination policy does not deny safety risk; it strives to minimize risk and ***should only recommend vaccination when the context, the expected disease and transmission risk in the susceptible population can be scientifically justified.***



Further safety risk mitigation: lower the price paid for maintaining HI

Avoid questionable or problematic ingredients

- ***non-replicating antigens*** if possible
- native/particulate antigen where feasible
- avoid unnecessary inflammatory components & nucleic-acid-based active ingredient
- use naturally derived adjuvants
- avoid co-administration of adjuvanted vaccines



Residual safety risk: why opponents retain a strong argument and why mandates are hard to defend



If adverse effects cannot be ruled out entirely, vaccination of healthy children is difficult to justify when there is no circulation – and even more difficult to mandate.

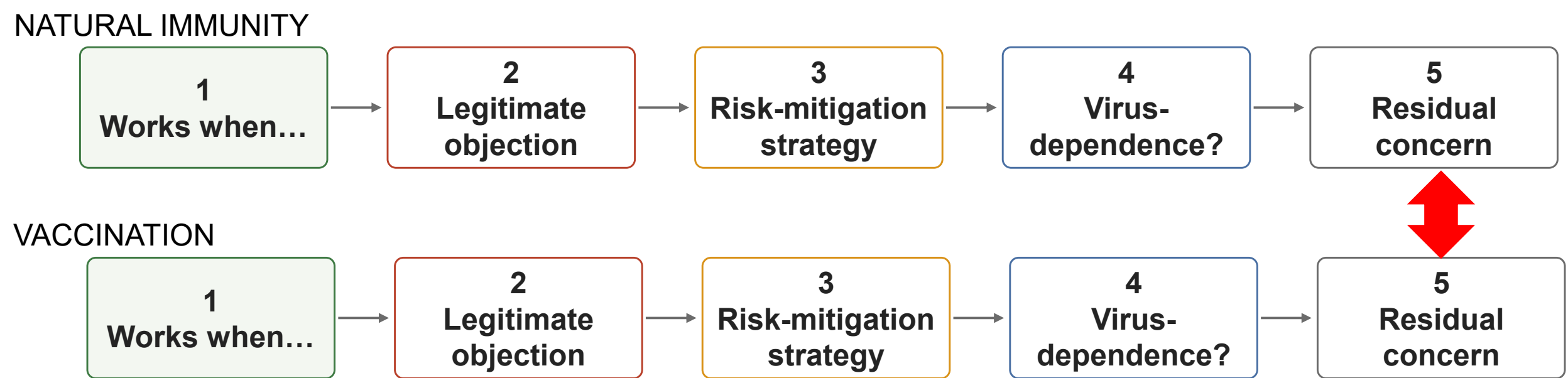


Residual safety risk: why opponents retain a strong argument, why mandates are hard to defend and why HI is so easily taken for granted

If adverse effects cannot be ruled out entirely, vaccination of healthy children is difficult to justify when there is no circulation – and even more difficult to mandate.

Paradoxically, the success of HI can undermine the perceived need for vaccination. Once sustained population immunity has made transmission rare, the pathogen appears to have disappeared and the individual risk of disease seems negligible. *But that very absence of disease is not evidence that protection is no longer needed – it is evidence that the population's collective immunity is working.* People often fail to appreciate that an increasing proportion of immunologically naïve individuals substantially increases the population-level risk of disease.

Both, immunization by natural infection or vaccination inevitably come with a 'certain' risk of potentially serious safety issues.



Rational approaches to generating protective immunity can substantially reduce safety risks. However, *no approach can eliminate the residual risks entirely and **opponents and proponents of vaccination perceive those risks very differently, regardless of the epidemiological context.*** Consequently, there is little reason to expect the fundamental divide between them to narrow anytime soon, unless we consider a novel immunization strategy: **enhancing innate immunity.**



The constructive shift: from contested routes to a third path

Natural infection
works in some settings
but carries *risk of case
fatalities*

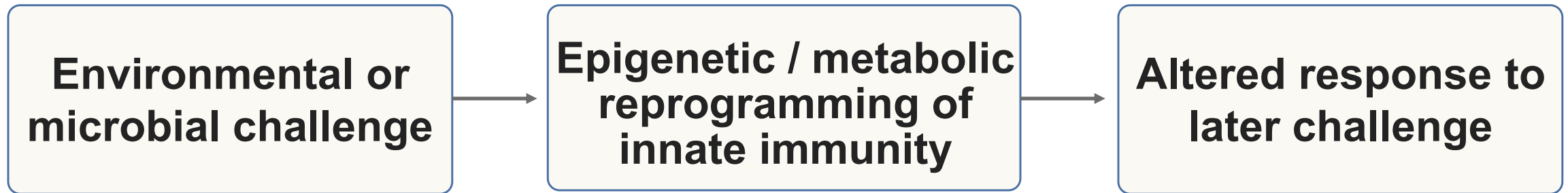
Vaccination
works in some settings
but carries *risk of vaccine-
associated disease*

Pathogen-nonspecific
innate immune enhancement
aims to protect against disease
while *avoiding both safety risks*

PNIIIE aims at strengthening the first line of immune defense without stimulating adaptive immune responses (i.e., without infectious challenge, vaccination or adjuvants).



Training innate immunity: established biological basis



BCG vaccination (and SC-2 infection) illustrates that immune interventions can induce nonspecific protective effects that cannot be reduced to classical adaptive immune responses (i.e., Ag-specific Ab or T-cells)



Scenario 3: PNIE via pathogen-derived self-mimicking peptides

What is avoided

- no replicating pathogen
- no classical vaccine platform
- no antigen-specific immune stimulation
- no adjuvants

What is used

- short synthetic peptides mimicking pathogen-derived '*altered self*' motifs

Intended effect

- educate and train cytolytic *natural killer (NK) cells*
- to recognize '*altered-self*' peptides on the surface of infected target host cells at an early stage of infection
- kill infected host target cells before progeny virus is released

Dual safety risk mitigation: by obviating the need to trigger or train innate immunity through exposure to infectious pathogens or conventional vaccines, it removes the associated safety liabilities.



NK-cell-centered mechanism: early recognition of altered self

Peptide-based
training signal
- no adjuvants-

Trained cytolytic
NK cells

Pathologically
altered host cell
recognized early

Killing before
viral progeny

Rapid recall
upon re-
exposure due
to memory

Trained NK cells could kill *pathologically altered host cells at an early stage of cellular alteration*, thereby preventing or curtailing both disease and viral transmission in pre- or post-exposure settings.

In this way, *individual immunization would neither contribute to neither depend on HI* ⇒ no incentive for public health authorities to 'push the patch.'



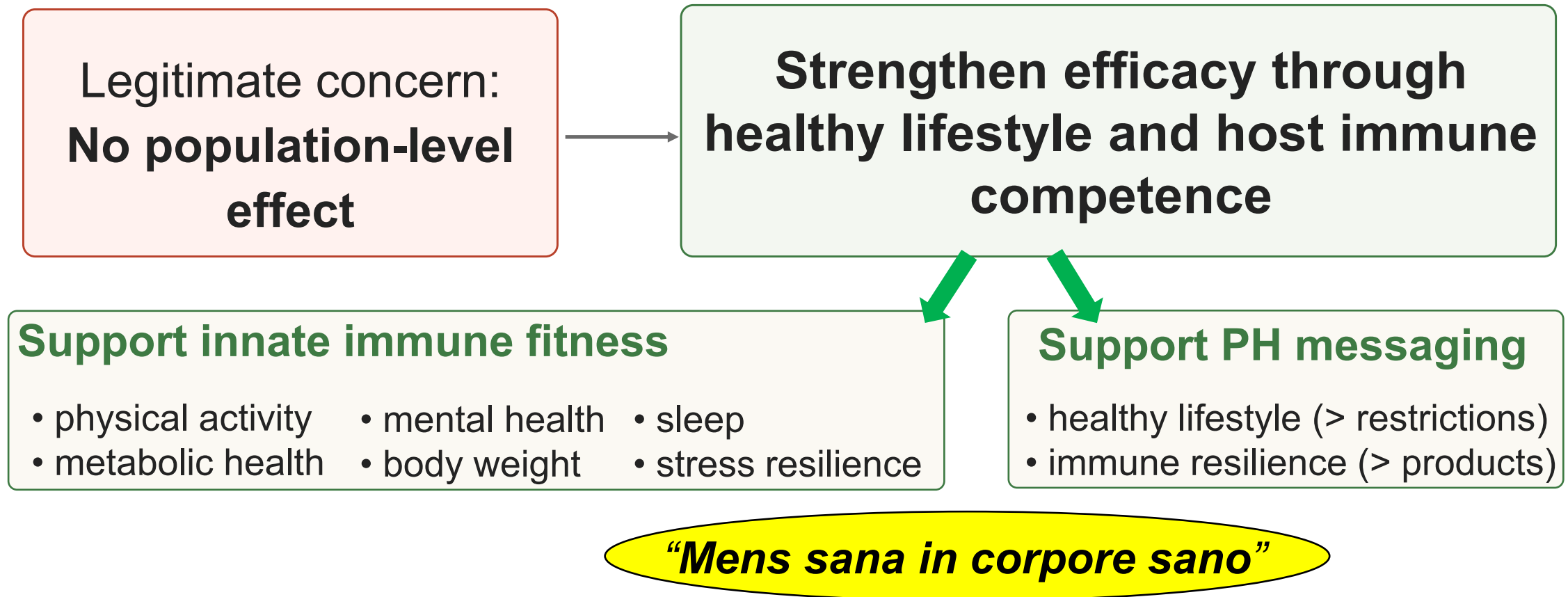
Innate immune enhancement: different viruses *do not require* a different basic logic

Classical vaccination targets *variable* viral antigens; therefore the *viral ecology* often determines whether vaccination is appropriate.

Innate enhancement aims at ‘altered-self’ / pathogenicity-associated motifs; *therefore the principle is intended to be pathogen-nonspecific.*

The key variable then shifts from “*which virus/ which context?*” to “*can the host recognize cells whose surface self-signature has been altered by infection or disease* and rapidly mount a sufficiently strong innate response to eliminate those cells?”

PNIE: The main limitation is a person's health status





Disease does not equal disaster

**Mild breakthrough
infection may
occur**

**Adaptive immune
boosting**

**More sterilizing
immunity later**

The biological goal is not necessarily to prevent every symptom but *to prevent serious disease*, uncontrolled transmission (due to immune escape) and immune pathology.



Conclusion:

No single gold standard for natural or vaccine-induced immunization
– but a common path beyond the pro-/anti-vax divide: protect without causing harm!

Natural immunity

- can generate HI and resolve *certain* ASLVI pandemics
- requires reduced infectious pressure
- *residual case fatality risk remains*

Pre-exposure Vaccination

- can preserve HI after outbreaks but only suitable for *certain* types of ASLVIs
- should use non-replicating Ag
- *residual adverse-event risk remains*

Innate immune training

- protection does not involve HI + depends on host health status
- *strong mechanistic rationale for avoiding safety concerns*
- uses pathogen-derived self-mimicking motifs

Conclusion:

Cont'nd

No single gold standard for natural or vaccine-induced immunization

– but a common path beyond the pro-/anti-vax divide: protect without causing harm!

As long as the mechanisms of immune protection remain poorly understood, vaccinology remains *empirical and potentially dangerous* – for example, **when vaccination is deployed during intense viral circulation or when LAVs are administered during the incubation period** – any immunization strategy may then be misapplied, discredited or rejected, each of which can itself carry serious risks.

The responsibility of public-health and regulatory authorities should therefore be to determine which immunization strategy, in which epidemiological and immunological context, *provides the greatest protection at the lowest avoidable biological and societal cost*.

It is unacceptable for such decisions to be made without a sound understanding of the role of natural immunity, the potential of trained innate immunity and the limitations of vaccine-induced adaptive immunity. These concepts are central to any scientifically rational assessment of epidemic or pandemic control and of large-scale vaccination programs.



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Lifestyle and immune competence

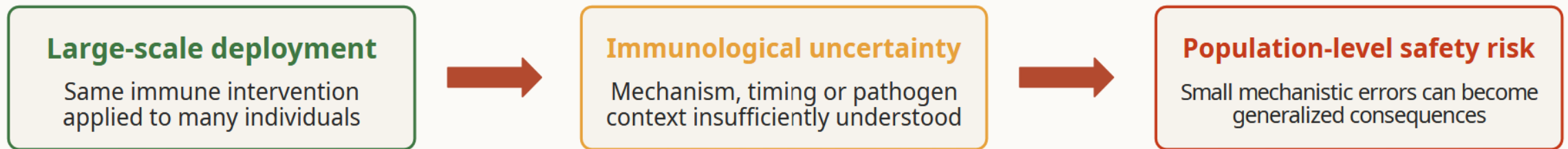
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Back-up slides....

Mass vaccination: individual safety is only half the story

Empirical large-scale vaccination programs transform immunological uncertainty into a population-level safety issue.



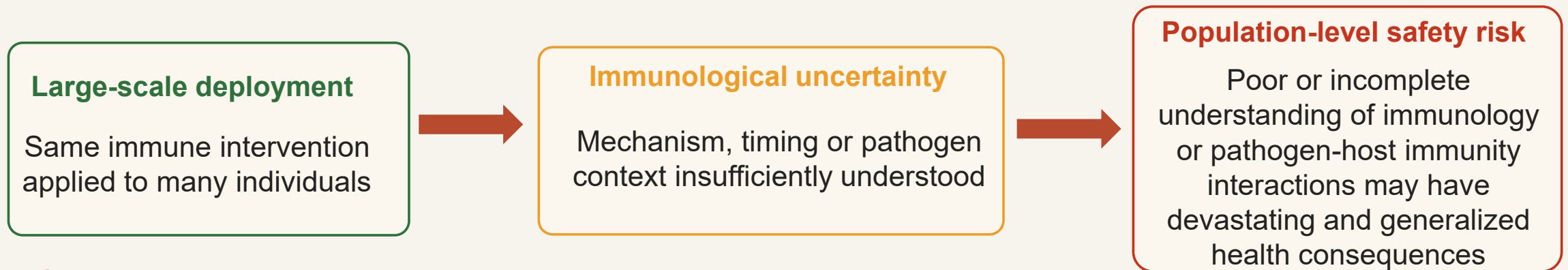
If the intervention...

- **drives non-sterilizing immune pressure** *continued replication under population-level immune pressure*
- **alters disease susceptibility** *risk of disease exacerbation or changed disease dynamics*
- **is deployed in the wrong epidemiological context** *timing and transmission ecology amplify biological consequences*

...the safety risks are scaled up too.

Mass vaccination: individual safety is only half the story

Empirical large-scale vaccination programs transform immunological uncertainty into a population-level safety issue.



If the intervention...

- drives non-sterilizing immune pressure → *continued replication under population-level immune pressure*
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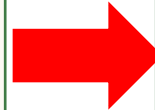
...the safety risks are scaled up too.

What about live attenuated vaccines (LAVs). Should they still be used?

Because LAVs transiently replicate and more closely mimic natural infection, they can induce *broader and faster functional protection after a single dose*. However, their replicative capacity also carries inherent risks, making them a *double-edged sword*.

When usage of LAVs may be considered

- natural infection is too risky
 - rapid outbreak control is required
 - LAVs may be best available option in specific outbreaks
- (example: large-scale measles outbreak)



Safety risk mitigation!

Given the potentially serious consequences, *safety-risk mitigation is critical*: never administer replication-competent vaccines/ viral vectors to close contacts of index cases as administration during the incubation period may exacerbate the disease! (e.g., rVSV-ZEBOV; West African Ebola epidemic [2015–16]).
<https://www.voiceforscienceandsolidarity.org/scientific-blog/guinea-the-ebola-vaccine-trial-and-the-reported-interim-results>

This is the exception that proves the rule: mechanism and context, not polarized slogans, should drive immunization strategy.

Backup slide: Can childhood vaccination maintain herd immunity for common viral infections?

Key criterion: does immunity sufficiently interrupt infection and onward transmission in the relevant host–pathogen ecosystem?

Infection / virus	Incubation	Herd-immunity potential from large-scale childhood vaccination
Measles	~10–14 d	Strongest: requires very high coverage; suited to maintaining established HI
Rubella	~12–23 d	Strong: childhood/adolescent programs can sustain population protection
Mumps	~12–25 d	Moderate: reduces spread, but waning immunity/outbreaks can occur
Varicella	~10–21 d	Moderate–strong: indirect protection, but VZV latency complicates framing
Rotavirus	~1–3 d	Partial: reduces disease burden and provides indirect protection
Influenza A/B	~1–4 d	Context-dependent: indirect effects possible, but antigenic drift and reservoirs limit durable HI
SARS-CoV-2	~2–5 d*	Weak for durable HI with current vaccines: transmission and immune escape remain central issues
RSV	~4–6 d	Limited/uncertain: protection against severe disease ≠ reliable transmission blockade
Rhinovirus / common cold	~1–3 d	Poor: many serotypes, short incubation, frequent reinfection
Seasonal HCoV	~2–5 d	Poor–limited: reinfection and antigenic/immune variability
Norovirus	~12–48 h	Poor–limited: short incubation, strain diversity, environmental spread
Enteroviruses / HFMD	~3–6 d	Limited: multiple viruses/serotypes and variable transmission ecology
Adenovirus	~2–14 d	Limited as broad childhood HI strategy: many types; disease-context specific
HPV	months–years	Yes , but slow: vaccination can reduce circulation and indirectly protect unvaccinated cohorts
Hepatitis B	~45–160 d	Yes , especially via infant vaccination and prevention of chronic-carrier transmission

Take-home: Childhood vaccination can maintain herd immunity only when it blocks transmission sufficiently; this is not true for “all viruses”.

*SARS-CoV-2 incubation has shortened for recent variants. Selected refs: Measles HI/coverage: WHO/UNICEF + recent reviews; Rotavirus indirect protection: Expert Rev Vaccines 2024; Influenza childhood indirect protection: J Infect Dis 2010 / systematic review; HPV herd effects: population studies; HBV: universal infant vaccination literature.

RECOMMENDED DOSES

U.S. CHILDREN CONCEPTION - 18 YEARS | SOURCE: CDC.GOV

1940

- DTP
- SMALLPOX

1960

- DTP
- OPV
- SMALLPOX

1983

- DTP (2 MONTHS)
- OPV (2 MONTHS)
- DTP (4 MONTHS)
- OPV (4 MONTHS)
- DTP (6 MONTHS)
- MMR (15 MONTHS)
- DTP (18 MONTHS)
- OPV (18 MONTHS)
- DTP (4 YEARS)
- OPV (4 YEARS)
- TD (14 YEARS)

1986 NATIONAL CHILDHOOD VACCINE INJURY ACT

THE CHILDHOOD VACCINE INJURY ACT WAS SIGNED INTO LAW, CREATING A COMPENSATION PROGRAM FUNDED BY TAXPAYERS AND RELEASING PHARMACEUTICAL COMPANIES FROM LIABILITY.

“

CHOICE IS SIMPLY THE
ABILITY TO PERSONALIZE
THIS SCHEDULE BY ANY
ONE OR MORE DOSES.

”

2021

- FLU (PREGNANCY)
- TDAP (PREGNANCY)
- HEP B (BIRTH)
- HEP B (2 MONTHS)
- ROTA (2 MONTHS)
- DTAP (2 MONTHS)
- HIB (2 MONTHS)
- PCV (2 MONTHS)
- IPV (2 MONTHS)
- ROTA (4 MONTHS)
- DTAP (4 MONTHS)
- HIB (4 MONTHS)
- PCV (4 MONTHS)
- IPV (4 MONTHS)
- HEP B (6 MONTHS)
- ROTA (6 MONTHS)
- DTAP (6 MONTHS)
- HIB (6 MONTHS)
- PCV (6 MONTHS)
- IPV (6 MONTHS)
- FLU (6 MONTHS)
- FLU (7 MONTHS)
- HIB (12 MONTHS)
- PCV (12 MONTHS)
- MMR (12 MONTHS)
- VAR (12 MONTHS)
- HEP A (12 MONTHS)
- DTAP (18 MONTHS)
- FLU (18 MONTHS)
- HEP A (18 MONTHS)
- FLU (30 MONTHS)
- FLU (42 MONTHS)
- DTAP (4 YEARS)
- IPV (4 YEARS)
- MMR (4 YEARS)
- VAR (4 YEARS)
- FLU (5 YEARS)
- FLU (6 YEARS)
- FLU (7 YEARS)
- FLU (8 YEARS)
- FLU (9 YEARS)
- FLU (10 YEARS)
- HPV (11 YEARS)
- HPV (11 YEARS)
- FLU (11 YEARS)
- TDAP (12 YEARS)
- FLU (12 YEARS)
- MEN (12 YEARS)
- FLU (13 YEARS)
- FLU (14 YEARS)
- FLU (15 YEARS)
- FLU (16 YEARS)
- MEN (16 YEARS)
- FLU (17 YEARS)
- FLU (18 YEARS)

2024

- RSV (PREGNANCY)
- FLU (PREGNANCY)
- TDAP (PREGNANCY)
- COVID (PREGNANCY)
- RSV (BIRTH - 19 MONTHS)
- HEP B (BIRTH)
- HEP B (2 MONTHS)
- ROTA (2 MONTHS)
- DTAP (2 MONTHS)
- HIB (2 MONTHS)
- PCV (2 MONTHS)
- IPV (2 MONTHS)
- ROTA (4 MONTHS)
- DTAP (4 MONTHS)
- HIB (4 MONTHS)
- PCV (4 MONTHS)
- IPV (4 MONTHS)
- HEP B (6 MONTHS)
- ROTA (6 MONTHS)
- DTAP (6 MONTHS)
- HIB (6 MONTHS)
- PCV (6 MONTHS)
- IPV (6 MONTHS)
- FLU 1 (6 MONTHS)
- FLU 2 (7 MONTHS)
- COVID 1 (6 MONTHS)
- COVID 2 (7 MONTHS)
- HIB (12 MONTHS)
- PCV (12 MONTHS)
- MMR (12 MONTHS)
- VAR (12 MONTHS)
- HEP A (12 MONTHS)
- DTAP (18 MONTHS)
- FLU (18 MONTHS)
- COVID (18 MONTHS)
- HEP A (18 MONTHS)
- FLU (2.5 YEARS)
- COVID (2.5 YEARS)
- FLU (3.5 YEARS)
- COVID (3.5 YEARS)
- DTAP (4 YEARS)
- COVID (4.5 YEARS)
- IPV (4 YEARS)
- MMR (4 YEARS)
- VAR (4 YEARS)
- FLU (5 YEARS)
- COVID (5.5 YEARS)
- FLU (6 YEARS)
- COVID (6.5 YEARS)
- FLU (7 YEARS)
- COVID (7.5 YEARS)
- FLU (8 YEARS)
- COVID (8.5 YEARS)
- FLU (9 YEARS)
- COVID (9.5 YEARS)
- FLU (10 YEARS)
- COVID (10.5 YEARS)
- HPV (11 YEARS)
- HPV (11 YEARS)
- FLU (11 YEARS)
- COVID (11.5 YEARS)
- TDAP (12 YEARS)
- FLU (12 YEARS)
- COVID (12.5 YEARS)
- MEN (12 YEARS)
- FLU (13 YEARS)
- COVID (13.5 YEARS)
- FLU (14 YEARS)
- COVID (14.5 YEARS)
- FLU (15 YEARS)
- COVID (15.5 YEARS)
- FLU (16 YEARS)
- COVID (16.5 YEARS)
- MEN (16 YEARS)
- FLU (17 YEARS)
- COVID (17.5 YEARS)
- FLU (18 YEARS)
- COVID (18.5 YEARS)



The question nobody dares to ask....

How can sufficient vaccine production and supply at an affordable price be ensured in the absence of mandates, strong recommendations, or advertising?