

**Supplementary Table S1.** Selection of pathogens and pathogen-derived vaccines that elicit a trained immunity effect.

Pathogen Species	Pathogens (1st Stimulus)	Host Species	Cell Type	Biological/Immunological Effects	References
BACTERIA	BCG vaccine	Human and mice	Monocytes Macrophages NK-cells	<ul style="list-style-type: none"> <li>Induction of non-specific protection against various infections beyond tuberculosis, such as yellow fever and malaria, e.g., due to enhanced cytokine production upon a 2nd stimulus.</li> </ul>	[24,25,26]
	<i>Salmonella typhii</i> vaccine	Human	Monocytes	<ul style="list-style-type: none"> <li>Oral vaccination with attenuated <i>Salmonella typhii</i> causes a reaction to a second non-specific stimulus with increased cytokine production, and enhanced expression of TLR4/5, as well as multiple surface markers on monocytes.</li> </ul>	[27]
	<i>Pseudomonas aeruginosa</i>	Human	Epithelial cells	<ul style="list-style-type: none"> <li>Exposure of respiratory epithelial cells with <i>P. aeruginosa</i> modulated their response to a subsequent different stimulus.</li> <li>Employing inhibitors of histone acetyltransferase and methyltransferase, epigenetic regulation was assumed as the reason.</li> </ul>	[16]
	<i>Acinetobacter baumannii</i>	Mouse	Macrophages	<ul style="list-style-type: none"> <li>Intranasal vaccination can induce a training effect in alveolar macrophages and protected against subsequent infection not only with <i>A. baumannii</i> but also with <i>P. aeruginosa</i>.</li> <li>Enhanced TLR4- and TNF-<math>\alpha</math> expression was observed in RAG-1 deficient mice.</li> </ul>	[17]
	<i>Neisseria gonorrhoe</i>	Human	Macrophages	<ul style="list-style-type: none"> <li>Epigenetic modifications, like enriched H3K9ac, were detected, indicating that <i>N. gonorrhoe</i> leads to a trained immunity phenotype in macrophages.</li> </ul>	[18]
	Measles vaccine	Human		<ul style="list-style-type: none"> <li>Mortality studies in developing countries suggest that the measles vaccine may provide a broad general positive impact on mortality</li> </ul>	[28]
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				rates, independent of its targeted protection against measles.	
	Smallpox vaccine	Human		<ul style="list-style-type: none"> <li>Immunization correlated with improved long-term survival in a high-income country, not accountable for by the vaccine's specific protective effects.</li> </ul>	[29]
	Oral polio vaccine (OPV)	Human		<ul style="list-style-type: none"> <li>Vaccination with OPV reduces all course childhood mortality in developing-countries.</li> </ul>	[30]
	Influenza vaccine	Human	Monocytes	<ul style="list-style-type: none"> <li>Influenza vaccination was linked to a reduction in SARS-CoV-2 infections and an improved immune response concerning various viral stimuli.</li> <li>In addition to adaptation in surface molecules of monocytes, decreased production of IL-1<math>\beta</math> and IL-6 and additionally an increase in IL-1-RA could be shown.</li> </ul>	[31]
	Herpesvirus	Mice	Macrophages	<ul style="list-style-type: none"> <li>Protection against a subsequent infection with <i>Listeria monocytogenes</i> and <i>Yersinia pestis</i> due to enhanced production of IFN-<math>\gamma</math> and activation of macrophages.</li> </ul>	[19]
	Hepatitis B virus	Human	Monocytes	<ul style="list-style-type: none"> <li>Exposure of newborns to HBV in utero appears to trigger a state of trained immunity.</li> <li>In vitro analyses revealed that umbilical cord blood exhibited heightened responsiveness to bacterial infection, evidenced by alterations in cytokine production, for instance.</li> </ul>	[20]
FUNGI	<i>Candida albicans</i>	Mouse	Monocytes	<ul style="list-style-type: none"> <li><math>\beta</math>-glucan in fungal cell wall triggers alterations in the genetic expression of immune-related genes, leading to heightened cytokine production in monocytes.</li> </ul>	[21]
	<i>Cryptococcus neoformans</i>	Mouse	Dendritic cells	<ul style="list-style-type: none"> <li>Enhanced proinflammatory cytokine production, such as IFN-<math>\gamma</math>, associated with</li> </ul>	[22]

				epigenetic changes in response to a 2nd stimulus.	
PARASITES	<i>Plasmodium falciparum</i>	Human	PBMCs	<ul style="list-style-type: none"><li>• After exposure to <i>Plasmodium falciparum</i>, the response of PBMCs was increased when exposed to TLR2 stimulation. Characteristic epigenetic and metabolic alterations associated with a trained immunity phenotype have been identified.</li></ul>	[23]