

PRIMAL HEALTH RESEARCH

A NEW ERA IN HEALTH RESEARCH

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VACCINATIONS FROM A PRIMAL HEALTH RESEARCH PERSPECTIVE

Studies detecting in the pre- and perinatal periods risk factors for a great diversity of medical conditions predominate in The Primal Health Research Database. This is why one can conclude today that our health is to a great extent shaped in the womb and that 'womb ecology' is the most vital aspect of human ecology.

However, our definition of the 'Primal period' also includes the year following birth: we had assumed originally that any event occurring between birth and the age of one year might also have life-long consequences, although occurring comparatively late in the formation of a human being.¹ This is why we must raise questions about possible life-long effects of early vaccinations.

The efficacy of the dozen vaccines offered to modern infants is well documented. It is evaluated by clinical studies and also by immunological responses: for example it is indisputable that tetanus vaccine is highly effective, compared with whooping cough vaccine or tuberculosis vaccine (BCG). The short-term side effects of different vaccines have also been seriously investigated and reported. Our main interest is in the possible long-term health consequences of the multiple vaccines currently given to babies. For this purpose we'll explore the Primal Health Research database using the keyword 'vaccination'.

Randomised controlled trials

It is well accepted that the golden method to evaluate the ratio of benefits to risks and possible long-term consequences of any medical intervention is through randomised controlled

trials. Let us first emphasise that, in general, this golden method is not used for early vaccinations. This is one of the reasons why our database has so few valuable studies in this field of research.

However, we must keep in mind that, historically speaking, the first randomised controlled trial was not, as it is commonplace to claim, an evaluation of streptomycin treatment of pulmonary tuberculosis published in British Medical Journal in 1948.² In fact the first one explored the effects of tuberculosis vaccine (BCG). Details of this original trial were published as early as 1942.³ Between December 1935 and February 1938, 3,025 American Indian and Alaska Native children aged one month or more who had normal chest radiography and who did not react to tuberculin were allocated to receive either a single intracutaneous dose of BCG or a placebo. The trial was conducted in southeast Alaska, Arizona, North Dakota, South Dakota, and Wyoming. Two strains of BCG were used. The pre-1948 analysis of this trial showed that efficacy was slightly higher among women than men (79% vs 68%).⁴ In this analysis the efficacy of BCG to prevent tuberculosis was the only outcome measured.

Interestingly, the last follow-up of this trial took place from 1992 to completion of data collection in 1998.⁵ The abstract of the relevant article mentioned only the overall incidence of tuberculosis, which was 66 and 138 cases per 100,000 person-years in the BCG vaccine and placebo groups, respectively, for an estimate of vaccine efficacy of 52%. Adjustments for age at vaccination, tribe, subsequent BCG vaccination, chronic medical illness, isoniazid use, and bacille Calmette-Guérin strain did not substantially affect vaccine efficacy. There was a slight but not statistically significant waning of the efficacy of BCG vaccination over time, greater among men than women.

In fact, the most valuable data from a primal health research perspective are not found in the abstract of the JAMA article, but only in the full text. The overall mortality rates were higher in the non-vaccinated group; furthermore the rates of deaths unrelated to tuberculosis were also higher in the non-vaccinated group (4.07% versus 3.0%), and there was a slightly higher prevalence of diabetes in the placebo group, at 25.7% versus 21.8% in the BCG vaccine group ($P = .02$). From this unique 60-year follow-up study one can conclude that in this population BCG had a positive non-specific effect on health.

There have been other randomized trials regarding the effects of BCG. The only outcome measures were always efficacy of the vaccine to prevent tuberculosis. This was the case of the huge Madras study, in South India, that involved 260,000 individuals (at age 7 and a half there was no evidence of a protective effect of the BCG vaccine).⁶ This was also the case of a review of 10 randomised trials (the average efficacy more than 10 years after vaccination was 14%)⁷ and a meta-analysis of BCG in neonates and infants in 3 controlled trials and 6 case-control studies (it indicated that BCG vaccine efficacy in this age group may persist through 10 years after vaccination).⁸

We should not be surprised by the possible long-term non-specific effect on health of BCG. Such effects have also been suggested by a study in Guinea-Bissau, one of the world's poorest

countries, with one of the highest childhood mortality rates. In such a context child survival can be used as a criteria of health. A Danish team of researchers looked at child survival in order to study the non-specific effects on health of different vaccines.⁹ The study involved 15,351 women and their children born during 1990 and 1996. The mortality incidence over 6 month periods was evaluated.

The findings show that both BCG (and measles) vaccines halved child mortality. The significant reduction in mortality was unrelated to deaths from tuberculosis (or measles): it appears that BCG (and measles vaccines) have a non-specific beneficial effect on health. On the other hand, children who received the combination of diphtheria, pertussis and tetanus (DPT) and polio vaccines had a risk of death multiplied by 1.84.

The authors interpreted their findings with caution because the research was performed in difficult circumstances. However, selection biases were unlikely, because different vaccines were associated with opposite tendencies. The report of this study should be instrumental in conveying the concept of non-specific effects on health of early multiple vaccinations in infancy. It is noticeable that other studies in developing countries with high infant mortality rates seem to confirm that BCG has positive effects on health.¹⁰

There are also theoretical reasons to explore such non-specific effects of BCG. In the age of tap water and bathrooms, the immune system of human babies is not exposed to mycobacteria and therefore tends to be culturally locked into a particular state (state of 'T-helper-2-bias'). This tendency is reinforced by all vaccines used in infancy, except BCG, which tends—on the contrary—to deviate the immune system to toward the opposite direction (Th2). This is why the interactions between BCG and other vaccines commonly used in infancy are a possible avenue of research.

Our three-step inquiry suggests that these interactions may influence in particular the risk of asthma in childhood. We first analysed health criteria in a population of 446 children (mean age 8 years) who were homogeneous in terms of infant feeding. All children had been breastfed more than 1 year and had received only breast milk during the first 6 months. None of them had received BCG in infancy. In this particular population whooping cough vaccination appeared as a significant risk factor for asthma in childhood.¹¹ (at the time of our enquiry the whole cell vaccines were in use). Similar correlations were obtained among the pupils of Rudolf Steiner schools in Great Britain. However, whooping cough vaccination was not a risk factor among the pupils of a French Steiner school. None of the 17 pupils who had whooping cough vaccination and BCG were diagnosed as asthmatic (British and French pupils of Steiner schools share the same anthroposophic lifestyle). It is plausible that BCG can protect whooping cough-immunised children against asthma. We must add that, apart from the protective effects of BCG against leprosy and Buruli ulcer, BCG is associated with a reduced prevalence of nematode infestation and has been used as an adjuvant in the treatment of diseases as diverse as bladder cancers, interstitial cystitis, prostate cancers, malignant melanomas, small-cell lung cancers, visceral leishmaniasis, and chronic hepatitis B infection. This led us to conclude that BCG might have a future, although it is not very effective to prevent tuberculosis.¹²

The Danish Civil Registration System

Some of the studies available in our database have great scientific value, although they are not randomised controlled trials. Most of these studies—typically Scandinavian—involve huge numbers (hundreds of thousands of cases). A Danish series about childhood vaccination has been possible thanks to the data from the Danish Civil Registration System, which assigns a unique identification number to every live-born infant and new resident in Denmark.

The objective of the most recent study in this series was to explore a possible link between childhood vaccinations and the development of insulin-dependent diabetes.¹³ The authors evaluated a cohort comprising all children born in Denmark from January 1, 1990, through December 31, 2000, for whom detailed information on vaccinations and diabetes was available. Insulin-dependent diabetes was diagnosed in 681 children during 4,720,517 person-years of follow-up. The rate ratio for diabetes among children who received at least one dose of vaccine, as compared with non-vaccinated children, was 0.91 (95 percent confidence interval, 0.74 to 1.12) for Haemophilus influenzae type B vaccine; 1.02 (95 percent confidence interval, 0.75 to 1.37) for diphtheria, tetanus, and inactivated poliovirus vaccine; 0.96 (95 percent confidence interval, 0.71 to 1.30) for diphtheria, tetanus, acellular whooping cough, and inactivated poliovirus vaccine; 1.06 (95 percent confidence interval, 0.80 to 1.40) for whole-cell whooping cough vaccine; 1.14 (95 percent confidence interval, 0.90 to 1.45) for measles, mumps, and rubella vaccine; and 1.08 (95 percent confidence interval, 0.74 to 1.57) for oral poliovirus vaccine. The development of diabetes in genetically-predisposed children (defined as those who had siblings with diabetes) was not significantly associated with vaccination. Furthermore, there was no evidence of any clustering of cases two to four years after vaccination with any vaccine. It is clear that these results do not support any causal relation between childhood vaccination and insulin-dependent diabetes.

A previous study of this series looked at a possible association between vaccines containing a mercury derivative (thimerosal) and autism.¹⁴ The authors studied the 467,450 children born in Denmark from January 1, 1990, until December 31, 1996, comparing children vaccinated with a thimerosal-containing vaccine with those vaccinated with a thimerosal-free formulation of the same vaccine. During 2,986,654 person-years, they identified 440 autism cases and 787 cases of other autistic-spectrum disorders. The risk of autism and other autistic-spectrum disorders did not differ significantly between children vaccinated with thimerosal-containing vaccine and children vaccinated with thimerosal-free vaccine (relative risk 0.85 [95% confidence interval 0.60-1.20] for autism; relative risk 1.12 [95% confidence interval 0.88-1.43] for other autistic-spectrum disorders). Furthermore, there was no evidence of a dose-response association for autism and other autistic-spectrum disorders. Such results do not support a causal relationship between childhood vaccination with thimerosal-containing vaccines and development of autistic-spectrum disorders. Of course, we might claim that this study has a purely historical interest, since

the use of thimerosal-free vaccines is now the rule. In fact, such negative findings are important, because they reinforce the main conclusions of an overview of the studies accessed in our databank via the keyword autism. Let us recall that such an overview suggests that the critical period for genes-environment where autism is concerned is situated in the perinatal period, before the age of infant vaccinations.

Although measles, mumps, and rubella vaccination (MMR) is given to babies after the end of the primal period, we'll mention a study about the possible links of this vaccine with autism.¹⁵ This information was found in a retrospective study of all children born in Denmark from January 1991 through December 1998. Of the 537,303 children in the cohort (representing 2,129,864 person-years), 440,655 (82.0 percent) had received the MMR vaccine. The authors identified 316 children with a diagnosis of autistic disorder and 422 with a diagnosis of other autistic-spectrum disorders. The relative risk of autistic disorder in the group of vaccinated children, as compared with the unvaccinated group, was 0.92 (95 percent confidence interval, 0.68 to 1.24), and the relative risk of another autistic-spectrum disorder was 0.83 (95 percent confidence interval, 0.65 to 1.07). There was no association between the age at the time of vaccination, the time since vaccination, or the date of vaccination and the development of autistic disorder. This study provides strong evidence against the hypothesis that MMR vaccination causes autism. This study has been instrumental in putting an end to the 'MMR fuss'.

This review of the comparatively small number of valuable studies found in our database indicates that vaccinations are not widely investigated from a primal health perspective. At a time when babies are often given a mixture of up to a dozen of vaccines, we should be more curious about the possible lifelong non-specific effects on health of the current practices.

Michel Odent

References:

- 1 – Michel Odent. Primal Health. Century-Hutchinson 1986.
- 2 - MRC Streptomycin in Tuberculosis Trials Committee. Streptomycin treatment of pulmonary tuberculosis. BMJ. 1948;ii:769–783.
- 3 -Townsend JG, Aronson JD, Saylor R, Parr I. Tuberculosis control among North American Indians. Am Rev Tuberc. 1942;45:41-52. Aronson JD, Aronson CF, Taylor HC. A twenty year appraisal of BCG vaccination in the control of tuberculosis. Arch Int Med 1958; 101: 881-893.

- 5 - Naomi E. Aronson NE, Mathuram Santosham M, Comstock GW, et al. [Long-term Efficacy of BCG Vaccine in American Indians and Alaska Natives. A 60-Year Follow-up Study.](#) JAMA. 2004;291:2086-2091.
- 6 - Tuberculosis Prevention Trial, Madras. Trial of BCG vaccines in south India for tuberculosis prevention. Indian J Med Res. September 1979;70: 349-363.
- 7 - Sterne JAC, Rodrigues LC, Guedes IN. Does the efficacy of BCG decline with time since vaccination? Int J Tuberc Lung Dis. 1998;2:200-207.
- 8 - Colditz GA, Berkey CS, Mosteller F, et al. The efficacy of bacillus Calmette Guérin vaccination of newborns and infants in the prevention of tuberculosis: meta-analysis of the published literature. Pediatrics. 1995;96:29-35.
- 9 - Kristensen I, Aaby P, Jensen H. Routine vaccinations and child survival : follow up study in Guinea-Bissau, West Africa. BMJ 2000 ; 321 : 1435-9.
- 10 - Hall AJ. Vaccination and child mortality. Lancet 2004;364: 2156-2157.
- 11 - Odent M, Culpin E, Kimmel T. Pertussis vaccination and asthma: is there a link? JAMA 1994;272:592-593.
- 12 - Odent M. Future of BCG. Lancet 1999; 354: 2170.
- 13 - Hviid A, Stellfeld M, Wohlfahrt J, Melbye M. Childhood vaccination and type 1 diabetes. N Engl J Med. 2004 Apr 1;350(14):1398-404
- 14 - Hviid A Stellfeld M Wohlfahrt J Melbye M. Association between thimerosal-containing vaccine and autism. JAMA. 2003 Oct 1;290(13):1763-6
- 15 - Madsen KM, Hviid A, Vestergaard, M Schendel D, et al. A population-based study of measles, mumps, and rubella vaccination and autism. N Engl J Med 2002 Nov 7;347(19):1477-82.

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