



ISSN : 2347 - 2243

Indo - American Journal of Life Sciences and Biotechnology



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Seroprevalence of Measles IgG Antibodies Among Vaccinated Children in Urban Settlements

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Received: December 6, 2015 Accepted: March 27, 2016 Published: July 24, 2016

DOI: [10.62644/iajlb.2016.v12.i3.pp37-46](https://doi.org/10.62644/iajlb.2016.v12.i3.pp37-46)

ABSTRACT

Measles vaccination programmes are usually judged by how many children have been vaccinated, but a vaccinated child is not necessarily a protected one, and surveys of measles IgG antibodies are the commonest way of asking the second question. This narrative review considers what such a seroprevalence figure can and cannot tell us when it is measured in vaccinated children living in urban settlements. It draws on published work on the virus and its immune effects, on antibody as a marker of protection, on the effect of age at vaccination and maternal antibody, on persistence and waning, and on population immunity in crowded urban settings, and it reports no new data. The review finds that an IgG result depends heavily on the assay used, that a negative or equivocal result in a vaccinated child has several distinct explanations of which true vaccine failure is only one, and that surveys with different methods and age groups cannot be compared at face value. Urban settlements add particular problems of denominators, documentation and mobility. We propose a short set of questions for appraising a survey and conclude that seroprevalence is best read as a measure of programme performance in a defined group, not as a verdict on the vaccine.

Keywords: Measles; IgG antibodies; Seroprevalence; Vaccinated children; Urban settlements; Vaccine failure; Narrative review

Citation: Seabra, Gupta and Guillemot [2016]. Seroprevalence of Measles IgG Antibodies Among Vaccinated Children in Urban Settlements. *Indo-Am. J. Life Sci. Biotechnol.* 12(3):37-46. DOI: [10.62644/iajlb.2016.v12.i3.pp37-46](https://doi.org/10.62644/iajlb.2016.v12.i3.pp37-46)

Article Class: Narrative Review

1. Introduction

Measles is one of the most contagious infections known, and a child who is not immune is very likely to catch it if the virus reaches the household (1). A live attenuated vaccine has been in use for decades, and it has been one of the great

successes of public health (2, 3). Because the virus spreads so easily, a pocket of unprotected children can be enough to sustain transmission.

Programmes are usually monitored by coverage, the share of children recorded as vaccinated. Coverage is a statement about



syringes and registers. Protection is a statement about immunity, and the two are not the same thing. The most direct way to look at immunity in a population is to measure antibody in blood, and IgG against measles virus has become the usual marker because it is simple to collect and widely available (4, 5). A survey of IgG in vaccinated children then asks, in effect, whether the vaccine has done what it was given to do.

The answer is rarely as clear as the question. A negative IgG result in a vaccinated child can mean several different things, and a positive one is not a guarantee of protection. The difficulty is sharper in urban settlements, where crowding favours transmission, populations move, and records of who was vaccinated, and when, are often incomplete (6, 7). The title of this review refers to such settings, but we report no survey of our own and no local data; we write about what the published literature allows a reader to conclude from this kind of survey. The aim is a fair account of what a seroprevalence figure can carry, not a verdict on any one programme.

2. Literature Review

2.1 The Virus and Its Effects

Measles virus infects the respiratory tract and then spreads through the body, producing the familiar fever and rash, and in a minority of children serious complications of the lung and brain (1). The risk of death varies a great deal with nutrition, age and access to care (8), and modelling of the global burden shows how much of what is reported rests on estimates (9, 10).

The infection also does lasting damage to the immune system. Measles suppresses immune responses for weeks, and there is long-standing evidence that it interferes with the cells that coordinate them (11,

12). More recent ecological work suggests that the effect on defence against other infections may persist for a good while after the child has recovered, which would mean that the cost of an outbreak extends well beyond measles itself (13). That argument is still being tested. It is one reason why even small gaps in immunity are taken seriously.

2.2 Antibody as a Correlate of Protection

A correlate of protection is a measurable response that predicts whether a vaccinated person will resist the infection. For measles, antibody that neutralises the virus is the best established correlate, and a conventional protective level was proposed from outbreak investigations around 1990 (4, 14). It is worth remembering that this level was derived from particular groups under particular conditions. It remains a useful benchmark, but not a physical constant, and cell-mediated immunity contributes in ways an antibody test does not capture.

Laboratories do not usually measure neutralising antibody directly. The neutralisation test is the reference method, but it is slow and needs cell culture, so most surveys use an enzyme immunoassay for IgG instead (15, 16). Immunoassays agree well with neutralisation at higher levels and less well at low ones, which is precisely where a survey of vaccinated children needs the answer to be right. Different commercial kits can also classify the same serum differently, and standardising assays and reference sera was needed before European seroprevalence data could be set side by side (5). Table 1 summarises the main approaches.

2.3 Vaccination, Age and Maternal Antibody



The vaccine contains live attenuated virus, which replicates briefly in the child and prompts an immune response that resembles the natural one in many respects. Reviews of field effectiveness describe a vaccine that works well, with protection rising when a second dose is given (18). The response of any individual child depends on the age at which the dose is given, and the reason is maternal antibody: babies are born with their mother's IgG, which protects them for a time but can also neutralise the vaccine virus before it has provoked a response (19).

Table 1. Laboratory approaches used to measure measles immunity.

Method	What it measures	Main caution
Plaque reduction neutralisation	Antibody that neutralises virus; the usual reference (14, 15)	Slow, costly, needs cell culture
IgG immunoassay (ELISA)	Binding antibody to viral antigens (16)	Less sensitive at low levels; kits differ
IgG avidity assay	Strength of antibody binding (17)	Research tool; not used in routine surveys
Standardised panels	Common reference sera across laboratories (5)	Needed before surveys are compared

Note: Qualitative comparison drawn from the cited methodological literature; no performance figures are given because they differ between kits and laboratories.

The timing of the loss of maternal antibody is therefore central. Longitudinal work in a country with long-standing vaccination found that antibody from vaccinated mothers faded earlier than antibody from mothers who had had the disease, so that infants of vaccinated mothers lost protection sooner (20). Studies in communities with contrasting levels of

vaccination reached a similar conclusion (21), and the decay of passively acquired antibody against the three viruses in the combined vaccine has been followed in infants over the first year (22). A dose given while maternal antibody remains may leave the child unprotected, even though the dose is recorded on the card.

Infants vaccinated at six months showed a weaker antibody response than those vaccinated later in a controlled study, and serial studies at six, nine and twelve months pointed in the same direction (23, 24). Work in Haiti found that maternal antibody, undernutrition and concurrent illness all affected response in infants (25), and the infant immune system is itself limited in the quality and duration of the antibody it makes (26, 27). Policy has to balance vaccinating early, when the response may be blunted, against vaccinating late, when the child goes longer unprotected.

2.4 Persistence, Waning and Host Factors

Once antibody has been made, how long does it last? Studies of long-term persistence after vaccination generally find that most people keep measurable antibody for many years, though levels fall and a minority lose it (28, 29). After two doses, follow-up in a setting free of circulating virus found that antibody persisted in most children, though the levels that remained varied a good deal from one child to the next (30). Antibody to measles appears to persist unusually well compared with several other antigens (31).

Long persistence does not make waning irrelevant. Measles antibody after vaccination has been found to be lower than after natural infection in some comparisons, and boosting through contact with wild virus can restore it (32). Where measles has become rare, children lose the natural boosts that once kept antibody



high, so population antibody may fall even when coverage is stable. Outbreaks in vaccinated people have been analysed as secondary vaccine failure, a child who responded at first and later lost protection, although primary failure is hard to exclude without a measurement soon after the dose (33). Avidity testing has been used in outbreaks to separate the two (17).

Host factors add further variation. Children infected with HIV were found to respond less well to standard measles vaccine than uninfected children in Zambia (34), and the effect of vitamin A given around the time of vaccination has been reviewed alongside its effects on disease (35). These matter here because dense, poorer districts often carry more of such burdens.

2.5 Population Immunity and Urban Settlements

Individual protection and population protection are related but not identical. When enough children are immune, transmission cannot be sustained and those who are not immune are indirectly protected, a point set out in classic work on herd immunity and in more recent critical reviews of how the idea is used (36, 37). The proportion needed for measles is high because the virus is so infectious, and theory shows how uneven coverage and clusters of susceptible children can keep it circulating even when the average looks good (38). Experience of elimination and its fragility has been summarised several times (39, 40, 41).

Urban settlements challenge these ideas in practical ways. Crowded housing raises contact rates, and rapid population change makes it hard to know who is living where and who has been vaccinated (6). A review of reasons for non-vaccination and under-vaccination in lower-income countries described a mixture of access, information and household circumstances rather than any single cause (7). Where

children arrive, leave and are looked after by different relatives, a card may be lost and the denominator for a survey may be uncertain.

3. Materials and Methods

3.1 Scope and Question

The review asks one question in two parts. What can a seroprevalence figure for measles IgG in vaccinated children actually tell us about vaccine performance, and what features of urban settlements change how that figure should be read? We divided it into sub-questions: how antibody is measured, why a vaccinated child may lack measurable antibody, how survey design affects the figure, and which urban features matter. None of these steps is difficult, but each is easy to skip when a result looks reassuring.

3.2 Literature Assembly

We drew on peer-reviewed work in four areas: the biology and immunology of measles; the immune response to the vaccine and its persistence; laboratory methods; and population immunity and the epidemiology of vaccination in crowded settings. Sources were chosen for relevance, standing in the field and clarity of method, and only work published up to the year of this issue is cited.

We did not run a formal systematic search and we do not claim one. This is a narrative synthesis, and the choice of sources reflects the judgement of the authors. The review is therefore a better guide to how to read a survey than to the size of any one figure.

3.3 Appraisal Framework

To judge how much a survey can be trusted, we used a short set of questions, listed in Table 2. Which assay and cut-off were used, and how were equivocal results handled (15)? Was the age at vaccination known from a record, and were the number



of doses and the interval stated (30)? Who was sampled, and could the sample represent the settlement? Over what time since vaccination was antibody measured (28)?

Table 2. Questions used to appraise a measles seroprevalence survey.

Question	Why it matters	Source of concern
Which assay and cut-off?	Results change with method (15, 16)	Equivocal results treated as negative
Age at vaccination known?	Early doses respond poorly (19, 23)	Recall instead of cards
Doses and interval stated?	Two doses differ from one (30)	Doses pooled
Who was sampled?	Representativeness of the group	Clinic or school convenience samples
Time since vaccination?	Antibody may wane (28, 29)	Ages mixed in one figure

Note: The questions are the authors' working framework, built from the cited literature; they are not a validated instrument.

4. Results

4.1 What a Positive Result Means

Reading the literature as a whole, the first thing to say is that a positive IgG result in a vaccinated child is good news but is not the same as proven protection. The conventional protective level came from neutralising antibody (14), and an immunoassay may agree with it only approximately (15). Results near the threshold are the most uncertain.

A positive result also reflects the whole path from the dose to the blood sample, shown in Figure 1. The child must have received a valid dose, the vaccine virus must have replicated, memory and antibody must have been made, and the antibody must have lasted until the survey. Each step has its own modifiers, and the

result at the end of the path cannot be traced back to any one of them. In a survey of children from settlements where exposure to wild virus has been rare, antibody reflects vaccination; where circulation continues, some antibody reflects infection or boosting, and the survey cannot say which. That uncertainty is easy to overlook, because a single number hides the whole path.

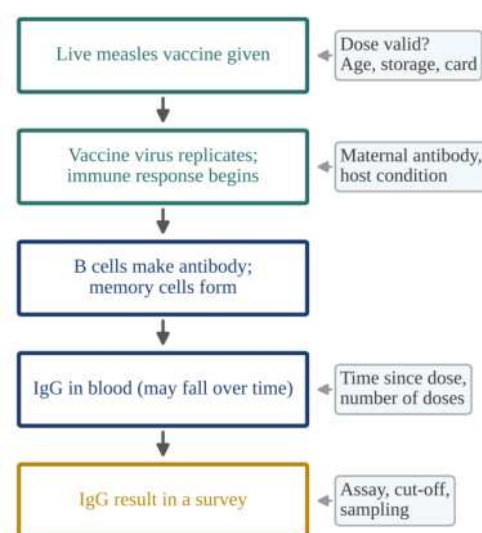


Figure 1. From vaccination to a measurable IgG result, with the factors that act on each step (schematic, not data).

4.2 Why a Vaccinated Child May Have No Measurable IgG

A negative or equivocal result in a child recorded as vaccinated has several explanations, set out in Table 3. The simplest is that the dose was given while maternal antibody was still present, or that the infant immune system was too immature to respond well, which are primary failures of response (19, 24, 26). Figure 2 shows the idea in schematic form: when maternal antibody and an early dose overlap, the response is blunted, whereas a later dose meets a clear field. In both cases the dose is on the card, yet the antibody never follows. Neither need be a fault of the vaccine itself.

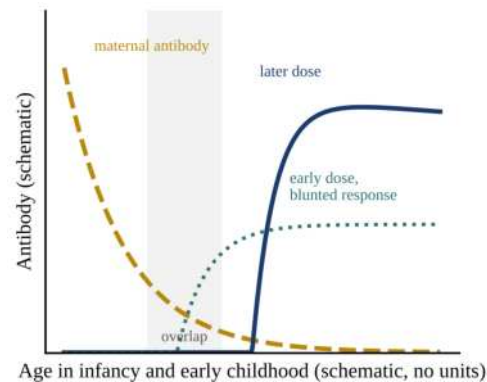
**Table 3.** Reasons a vaccinated child may have no measurable measles IgG.

Explanation	Mechanism	Vaccine failure?
Dose given too early	Maternal antibody interferes (19, 24)	Primary
Immune immaturity	Infant response is limited (26, 27)	Primary
Host condition	HIV or malnutrition (25, 34)	Possible
Waning	Antibody falls over years (32, 33)	Secondary
Assay limits	Low levels missed (15)	No
Not vaccinated	Card or recall is wrong (7)	No

Note: Several explanations can operate together, and a single serological result cannot separate them.

Other explanations do not involve the vaccine at all. A child may have been recorded as vaccinated without having been, or the card may carry a date that is wrong. The assay may have missed a low but real level of antibody (15). Waning after a good initial response is a third possibility and, as noted, avidity testing can help separate it from primary failure in outbreak settings but is not part of routine surveys (17). Illness and nutrition may also matter in some groups (25, 34). A health officer reading a survey report needs an answer to a plain question: who, exactly, was tested?

The practical lesson is that the proportion of vaccinated children without antibody cannot be read as the failure rate of the vaccine. It is an upper bound on one kind of failure and a mixture of several other things. Survey authors who report only the proportion, without stating the assay, the age at vaccination and the source of the vaccination history, leave the reader unable to apportion the result. The point is not that a survey is poor, only that its headline figure travels less far than its abstract suggests.

**Figure 2.** Qualitative course of measles antibody in early childhood, showing why the age at vaccination matters (schematic, not data).

4.3 Comparing Surveys

Surveys differ in the assay, the cut-off, the age of the children, the number of doses and the way the sample was drawn, and each of these alone can move the proportion. A major European effort showed that, without standardisation, the same sera could give different answers in different laboratories (5). A figure that cannot be reproduced with another kit is a finding about the kit as much as about the children. Age is a particularly important confounder because children vaccinated recently may have high antibody that falls with time, and an unstructured age range can hide this pattern (28, 29).

4.4 Reading a Survey from an Urban Settlement

In an urban settlement the problems above are joined by problems of sampling. Households may be hard to list, residents may be mobile, and the children most likely to be missed in a survey are often those least likely to have been vaccinated (6, 7). A convenience sample from a clinic or school over-represents children already in contact with services, and may look good without describing the settlement.

When a negative result is found, Figure 3 shows the order in which we would sort the explanations: first the reliability of the record, then the sensitivity of the assay,



and only then the timing of the dose, host factors and waning. The earlier explanations are the cheaper to check and the more often overlooked. Even then, a survey of this kind can say only that some children in the group lack measurable antibody, not which of them were failures of the vaccine. That is a modest claim, but it is the one the published evidence will currently bear.

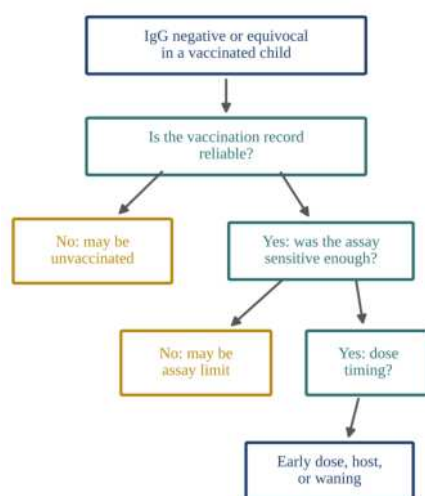


Figure 3. Questions that sort a negative result into groups of explanation when a survey is read (schematic, not data).

5. Discussion

5.1 Interpretation

The central finding is conditional. Seroprevalence of IgG in vaccinated children is informative about the programme as a whole, because it combines the quality of the vaccine delivered, the age at which it was given, and the persistence of antibody, with the quality of the survey that measured it. It is much less informative about the vaccine, which has an excellent record in the wider literature (2, 18). A low figure should lead first to questions about cold chain, age at vaccination and assay, and to a search for the pockets of unvaccinated children that population models suggest matter most (37, 38).

The second point concerns age at vaccination. Programmes in settings with high transmission have reason to vaccinate early, accepting a weaker response, and a second opportunity later is the usual way to recover protection in the children who responded poorly to the first dose (18, 23). A modest seroprevalence after an early first dose need not signal any fault in the programme. Read this way, a modest figure is a prompt for a closer look, not a conclusion.

5.2 Limitations

This is a narrative review and not a systematic one, and the choice of sources is ours. Reviews of this kind can overstate consensus and under-represent studies with unwelcome results. We have tried to name the points on which the literature is divided, but this is not a complete census of the evidence. Reviews of this kind are more useful when they say where the evidence stops than when they smooth it over.

The review also draws on evidence from different countries and eras, and on studies that were not carried out in urban settlements. Mechanisms such as maternal antibody decay and the effect of age on response are likely to apply widely, but the size of their effects depends on local conditions. We report no survey data, and we have avoided quoting proportions we could not check against the primary papers, which makes the review safer but less specific.

5.3 Directions for Future Work

Three needs stand out. First, a formal systematic review of seroprevalence in vaccinated children, with a transparent search, an appraisal that uses questions like those in Table 2, and an analysis that respects differences in assay and age. Second, repeated surveys in urban settlements with a documented method and



a sampling frame that includes mobile households. Third, wider use of standardised reference sera so that results can be compared (5).

For programme managers, the most useful step is probably to report the assay, cut-off, age at vaccination and number of doses alongside every seroprevalence figure. These details cost little to collect and make a large difference to what a figure can mean. It is a habit worth keeping even when the answer is the expected one.

6. Conclusion

IgG seroprevalence in vaccinated children is a sensible and widely used measure of how well a measles programme is performing, but it is easy to over-read. A negative result has several distinct explanations, only some of which concern the vaccine, and the figure depends on the assay, the age at vaccination, the number of doses and the way the sample was drawn. In urban settlements, difficulties with denominators, records and mobility add to these. The appropriate reading is of a programme measured in a defined group, with its uncertainties stated.

For practitioners, the message is practical rather than discouraging: describe the method, report the age and doses, check the records and treat comparisons between surveys with care. For researchers, the opportunity lies in standardised, repeated and well-sampled surveys that can separate waning, early dosing and missed vaccination, and that tell programmes what to do next.

7. Acknowledgements

The authors thank colleagues who commented on earlier drafts of this review.

8. Funding

This work received no specific grant from any funding agency in the public, commercial or not-for-profit sectors.

9. Conflict of Interest

The authors declare no conflict of interest.

10. Author Contributions

R.S. conceived the review and wrote the sections on measles virology and laboratory methods. An.G. wrote the sections on vaccine immunology and antibody persistence. Au.G. wrote the sections on population immunity and survey interpretation. All authors revised the manuscript and approved the final version.

11. Ethics Statement

This is a review of published literature. No human participants or samples were involved and no ethical approval was required.

12. Data Availability

No new data were generated. All sources are cited in the reference list.

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