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Antibody Response to Pulse Polio Immunization in Aligarh

The report by AS Hasan and colleagues is a timely reminder of the problems with OPV in India(1), first described in 1972(2). They measured seroconversion rates in three subgroups - vaccinated exclusively through NIP or pulse campaigns and vaccinated both ways(1). The highest response was in the last subgroup that received the highest number of doses (mean 8.4) of OPV. Seroconversion rate increases with increasing doses(3). This correlation is illustrated by Hasan's data, presented differently. For simplifying inter-group comparison, 'seroconversion index', a single variable, is useful in place of three variables of type-specific seroconversion rates(3). The mean of seroconversion rates will suffice as surrogate for seroconversion index(3). The *Table I* gives the results.

A similar gradation is also present with geometric mean antibody titres (GMT) - lowest in the NIP subgroup, and highest in the subgroup of NIP and pulse(1). Repeated infections do result in rise in GMT(3). Thus, without controlling for the number of doses the study is inadequate to compare differential responses of pulse versus NIP.

Loss of vaccine potency could not have been the reason for poor antibody response, since vaccine vial monitors were mandatory in 1999-2002(1). Their recommendation to investigate the cause of poor response is untimely in 2004, but what is urgently needed is to explore methods of improving immune responses for achieving interruption of wild poliovirus transmission. In spite of 96.5% coverage with 2-18 doses of OPV, gaps in immunity remained in the vaccinated and

TABLE I–*Relationship Between Number of Doses and Seroconversion Rate.*

Group/subgroup	Mean No. of doses	Mean sero conversion rate
NIP subgroup	3.8	80.8
Pulse subgroup	6.3	84.8
Vaccinated group	7.8	87.5
NIP and pulse subgroups	8.4	88.5

unvaccinated, showing that vaccine viruses did not spread extensively among children(1). The continued wild virus transmission has also questioned the theory that gut immunity induced by OPV would interrupt transmission rapidly. If 100% children must be individually vaccinated for interrupting transmission, gut immunity is not operative in a significant measure. If so, either OPV does not induce adequate gut immunity or faecal-oral is not the major route of wild virus transmission. As OPV does induce gut immunity, the conclusion is that the major route of wild virus transmission is not faecal-oral. The very young age of poliomyelitis (median age at 12-18 months) suggests high force of transmission, reminiscent of measles, the route of transmission being probably oral-nasal and pharyngeal. Fecal-oral transmission may be less important. Even when millions of viruses are fed in OPV, bypassing the nasopharynx, infection rate remains poor. If these arguments are valid, then IPV, which induces better pharyngeal mucosal immunity than OPV, is a valid tool for interrupting wild virus transmission(3). However, this choice (to use both vaccines as needed) should have been made in 1988; today we must try our best to eliminate wild virus in the endemic districts by applying intense vaccine pressure by immunizing infants who are the critical virus amplifiers, with 10 doses of OPV, or as near to ten as possible(4).

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