

Facts about plague epidemic, 1994

A special report on 'The Plague Epidemic, 1994' (*Curr. Sci.*, 1996, 71, 781-806) was published, having contributions from the Chairman and members of the Technical Advisory Committee (TAC) appointed by the Government of India. Going through these articles we have noticed the following factual errors, which may inadvertently mislead the readers.

1. The Department of Microbiology, Government Medical College (GMC) and New Civil Hospital (NCH), Surat was FIRST to culture and report the finding of the causative organism *Yersinia pestis* on the basis of clinical picture, microscopic morphology, cultural characters and biochemical characters immediately after the admission of first case at NCH, Surat. The isolation of the causative organism *Y. pestis* was carried out subsequently from admitted suspected

patients of pneumonic plague. The isolated cultures were preserved in specific media in duplicates. The epidemic was officially notified by the state administration based on these reports. All other agencies including National Institute of Communicable Diseases (NICD), Delhi; World Health Organisation; TAC, etc., only reconfirmed the above reports.

2. The TAC had not collected any fresh or stored samples of clinical material as the epidemic had subsided before its appointment. As such, the isolation of *Y. pestis* by the Defence Research and Development Establishment (DRDE), Gwalior on behalf of TAC was from the cultures collected from the Microbiology Department, GMC, Surat. Thus the information given in the article, 'Isolation and identification of *Y. pestis* responsible for the recent plague outbreak in India' (pp. 789-791 by Batra *et al.*), that

SPUTUM SPECIMENS were processed from the patients of Surat, is factually incorrect. Hence it would be appropriate to mention that the scientists at DRDE had done only the reisolation of *Y. pestis* which was originally done at Surat. This fact has been acknowledged by the Chairman, Ramalingaswamy, in the 'Prologue' published in the same issue (pp. 781-782), where it is mentioned that DRDE had undertaken the main task of isolation and characterization of *Y. pestis* from STORED CULTURES provided by NCH, Surat. Of course, the DRDE could undertake further molecular characterization etc. having more sophisticated facilities.

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Are lowered maternal insulin levels a cause of birth defects in diabetic pregnancies?

Occurrence of congenital abnormalities in offsprings of diabetic mothers is well known. Incidence of such abnormalities is alarmingly high (8-12%) and they occur in vital organs like the brain, heart, and kidney¹. Most of the abnormalities occur during the first 4 to 6 weeks of pregnancy. This is not surprising since the crucial developmental events such as embryonic induction and pattern formation occur during this period. Neurulation or the set of events leading to the formation of the neural tube also occurs by the first 3 weeks of development of the human embryo².

Decades of extensive research effort have gone into identifying the possible causes of congenital malformations in diabetic pregnancies. Lack of availability of human embryos for experimentation, due to ethical and legal considerations, has compounded the problem. Experimenters have however tried to exploit animal models, and diverse approaches used by them have led to the general conclusion that congenital malformations in diabetic pregnancies have multifactorial etiology including the teratogenic effects of glucose and ketones, free oxygen radicals, and abnormal DNA synthesis¹.

There is another possibility that has come up due to recent work³⁻⁷ in the area of early embryonic development in vertebrates. It is entirely likely that induction of abnormalities in human embryo developing in a diabetic mother's womb could be due to the paucity in mother's blood of an appropriate level of insulin. Considerable evidence has gathered over the past decade showing that insulin and receptors for insulin are essential during early development in frog, chick and mammalian embryos³⁻⁵. Insulin appears to be particularly crucial for the development of the nervous system^{6,7}. Assume that a human embryo like the other vertebrates also requires minute amounts of insulin coming from the mother's blood for normal development. The diabetic blood practically devoid of insulin is unable to provide the requisite levels of insulin to the embryo. This would result in the formation of a defective neural tube; congenital malformations observed in the offsprings of diabetic mothers would be a direct consequence of this deficiency.

It did come as a surprise when it was shown that insulin is essential for pancreatic development when blood cir-

ulation is yet to be established. But now that this hitherto unknown role of insulin has been reasonably well established, it may be worthwhile to test the hypothesis - non-availability of sufficient quantities of insulin to the developing embryo leads to congenital malformations in the offsprings of diabetic mothers. The frequency of such malformations is generally mitigated by insulin injections.

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4. De Pablo, F., Scott, L. A. and Roth, J., *Endocrine Rev.*, 1990, 11, 558-577.
5. Ghaskadbi, S. and Ghate, H. V., *Int. J. Dev. Biol.*, 1993, 37, 359-362.
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7. De Pablo, F. and De La Rosa, E., *Trends Neurosci.*, 1995, 18, 143-150.

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