area of active tectonics and contrary to popular belief, the active fault scars are rarely (generally not) sites of large fans/cones.


Charu C. Pant and A. K. Sharma reply:

We thank D. K. Ghosh for taking interest in our review paper and for giving suggestions on some aspects of the sedimentation in the Indus-Gangetic Basin (IGB). The observations he has made are of general nature and there are no specific comments. However, regarding his comment on the term 'Megacone', it may be stated that it has been used in sedimentological sense and not in the morphological context. It may be pointed out, a 'fan' has been defined as 'A sedimentary deposit whose surface is a segment of a cone that radiates downstream from a point, along a fault scarp or precipitous slope, a stream emerges from material in which its lateral movement is inhibited'.

It is well known from many comprehensive studies that the active-fault sites are usually the sites of active sedimentation and consequent deposition in the form of large fans/cones. As a matter of fact the rate and magnitude of the upliftment of the adjacent highlands controls site, rate and magnitude of the depositions of a fan.

We are sorry that some of the references cited by Ghosh have been inadvertently left out, as most of them have been published in proceedings of the seminars and not in refereed journals of wide circulation.

Indian strain of HCV


Authors of the paper, Indian strain of hepatitis virus: prevalence and detection1, made some remarks that 'practically no information is available on either the prevalence or nature of HCV in India' and they claimed to have found 'Indian strain of hepatitis virus'. There are, however, well documented prevalence of hepatitis C virus (HCV) in multitransfused, hence high risk recipients of HCV for blood and blood components from Eastern India2,3 and Western India4. Prevalence of HCV in voluntary blood donors from Eastern India varies from 0.9 to 1.1 per cent and in thalasaemia receiving programmed blood transfusion from a single centre is much higher5. The authors6 observed that sera from ELISA positive for HCV were also positive by their novel method. The presence of seropositivity by an apparently more sensitive method used in the study may not be a confirmatory criterion for a separate strain for HCV. An analogy may be drawn from other transmissible virus-hepatitis B (HBV) whose portal of entry and clinical behaviour are similar to HCV, wherein the high risk recipients screened for HBV by ELISA did show presence of hepatitis B markers either single or combined in almost 100% recipients of blood over a period. This could be due to the presence of small amount of viral antigen (HBsAg < 0.25 ng/ml) which may infective in repeated exposure in immunocompromised subjects. The authors7 who observed seropositivity for HCV marker in otherwise seronegative (ELISA) in transfused subjects who may have received small amount of reactants but not necessarily of different strains of HCV.

M. R. Das et al. reply:

With regards to the statement in the introductory paragraph of our paper (ref. I of Bhattacharya's letter that 'practically no information is available...'; we agree that it would have been better, had it read, 'very little information is available...'). The original statement was made in the context of the wealth of information (as for example, information on prevalence on a national level, information on sequences or phylogenetic classification) available on Hepatitis C strains for Japan, Western Europe or America in contrast to the very limited amount of such published information on HCV strains in India. However, we should have referred to references 2-4 of Bhattacharya's letter.

With regard to the second point raised in Bhattacharya's letter, we should emphasize that we have made no claims that what we have reported are new strains distinct from any other strains from India that might have been detected through ELISA. In fact, the emphasis in our paper is on the importance of detecting HCV by PCR (vide pp. 479-482 of ref. 1 mentioned above). The point to be noted is that the primers shown in figure 1 of our paper are unsuitable for detection of Indian strains of HCV by PCR. That is simply because these strains are different from the ones (Japanese and Americans) that can be detected using the primers shown in figure 1 referred to above. Consequently, the statement made in Bhattacharya's letter 'the presence of seropositivity by an apparently more sensitive method used in the study may not be a confirmatory criterion for a separate strain for HCV' is not relevant.

It may also be noted that in several countries where tests for Hepatitis C are mandatory, before release of samples from blood banks, in addition to ELISA, it is essential to do either PCR or RIBA or both. This is simply because none of the ELISA kits presently available in the market is fully reliable. We are also aware of the fact that in the United Kingdom, the Institute for Biological Standards and Control is doing strict comparisons of the reliability of several ELISA kits for Hepatitis C that have become available in the market, for appropriate recommendations.

Lastly, it may be noted that the number of PCR-positive samples that we have is almost double that of the sample which can be detected using ELISA kits, including the Abbott kit. This need not arise from the higher sensitivity of PCR alone. The majority of ELISA-negative, but PCR-positive cases happen to be patients who had received recent blood transfusions. In these patients viremia is present, but seroconversion has not yet happened. Consequently, kits designed to look for the presence of antibodies would not detect HCV. What is important to note is that the method we have reported is capable of detecting the presence of HCV before or after seroconversion.

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