netization effect wherein a sample that is magnetic in the presence of a field, becomes diamagnetic in the absence of a field. However, these bimetallic organic magnets are known to exhibit spontaneous magnetization below T_c even in the absence of an applied field. It is possible that a negative remanent magnetization arises in our samples **A** and **B** due to the antiferromagnetic coupling of hard and soft regions.

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Candidate live oral cholera vaccine strains produce a new cholera toxin

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When six candidate live oral cholera vaccine strains deleted for one or all known virulence factors are tested for enterotoxin production, two of them caused fluid accumulation in the initial rabbit ileal loop (RIL) test, the others did so after 1–3 serial passages through RIL. Culture filtrates also showed similar secretory response. Ten times concentrated culture filtrates of these strains gave precipitin band against anti-new cholera toxin showing reaction of identity. These observations clearly indicate that vaccine strains produce a secretogen antigenically similar to the new cholera toxin.

STRAINS of *Vibrio cholerae* 01 have been reported to secrete a number of extracellular products, such as haemolysin (Hly), zonula occludens toxin (Zot), accessory cholera enterotoxin (Ace) including the well-known cholera toxin (CT)[1]. Although CT has been suggested to be the factor responsible for severe cholera, however, if its gene is deleted the other toxic factors when present in some strains have been demonstrated to cause a mild secretory response[1]. However, it was evident from a recent volunteer study using a Tox-mutant of the biotype El Tor 01 Ogawa strain E 7946 designated CVD 110 that was deleted of all the above toxic factors, was still capable of causing diarrhoea in seven of ten volunteers[2]. These observations clearly indicate that such strains produce another secretogen.

It was shown earlier that CT gene-negative and positive strains of *V. cholerae* 01, biotype classical or El Tor, serotype Ogawa or Inaba of clinical or environmental origin or genetically engineered in the laboratory, produce a new cholera toxin (NCT) and the disease cholera can be caused by either CT or NCT or both[3,4]. An attempt was, therefore, made in this study to examine if the candidate vaccine strains of *V. cholerae* (obtained from J. B. Kaper, CVD, Maryland) deleted for one or all the other toxic factors produce NCT (Table 1).

Live cells of two of the six candidate vaccine strains tested in ligated ileal loops of adult albino rabbits (Belgian strain) following the method of De and Chatterjee[5], caused fluid accumulation in the initial tests, the others did so after 1–3 consecutive passage/s through RILs (Table 2), and thereafter outpouring of fluid by every strain increased on each passage (data not shown).

Culture filtrates of all the strains also caused fluid accumulation, although slightly less than that of the

Table 1. Virulence patterns of candidate vaccine strains

Vaccine strain	Parent strain	Virulence patterns				
		CTA	CTB	Hly	Zot	Ace
JBK 70	El Tor N 16961	–	–	+	+	+
CVD 104	El Tor N 16961	–	–	–	+	+
CVD 101	Classical 395	–	+	+	+	+
CVD 105	Classical 395	–	+	–	+	+
CVD 109	El Tor 7946	–	–	+	–	–
CVD 110	El Tor 7946	–	+	–	–	–

Table 2. Enterotoxicity of the genetically engineered vaccine strains

Vaccine strain	Range of fluid accumulations (ml/cm of RIL*)		Number of passages
	Live cells	Culture filtrates	
JBK 70	0.58–1.00	0.54–1.2	0
CVD 104	0.60–1.10	0.56–1.1	2
CVD 101	0.60–1.20	0.50–1.0	3
CVD 105	0.50–0.90	0.53–1.2	1
CVD 109	0.71–0.98	0.66–1.0	0
CVD 110	0.66–0.84	0.60–0.9	3
Positive control†	0.80–1.50	0.92–1.4	0
Negative control‡	0.0	0.0	–

*Range of accumulated fluid in ileal loops of two rabbits.
†BHIB culture of toxigenic strain 569B of *V. cholerae* 01.
‡BHIB.

toxigenic *V. cholerae* 01 strain no. 569B. These data indicate that the genetically engineered strains, including CVD 110 that lacks genes of all the known toxic factors, except NCT, produce a secretogen. Enhancement of secretory response upon passage suggests that if such a strain circulates in the community as is expected of a live oral vaccine, its virulence may increase further.

In the gel-diffusion test, 10-times concentrated culture filtrates of CT⁻ strain X-392 that produces NCT[3] and the candidate vaccine strains gave a precipitin band against anti-NCT showing reaction of identity (Figure 1). This observation suggests that the secretogen produced by these strains is antigenically similar to NCT.

Volunteer studies using CT and Hly, or CT, Hly, Zot and Ace genes deleted mutant strains did not support the role of Hly or other toxic factors in the pathogenesis of cholera, since no difference in the ability to cause diarrhoea was observed between them and their parent strains[1]. There is, therefore, every likelihood that the diarrhoeal episodes observed among the volunteers were due to the elaboration of NCT by the genetically engineered mutant strains. These observations indicate that vaccine strains produce the NCT and is supported by our earlier observation that anti-NCT completely neutralizes the enterotoxic activity of CT⁻ strains[6], which has been demonstrated to produce the NCT.

The present results indicate that the candidate vaccine strains of *V. cholerae* 01, biotypes classical and El Tor, serotypes Ogawa and Inaba, are able to produce NCT even when the genes for CT, Hly, Zot and Ace are deleted and thus possess the potential to cause diarrhoea. These observations are important not only for our understanding of the pathogenesis and epidemiology of the disease but also for developing an effective candidate live oral vaccine strain against cholera.

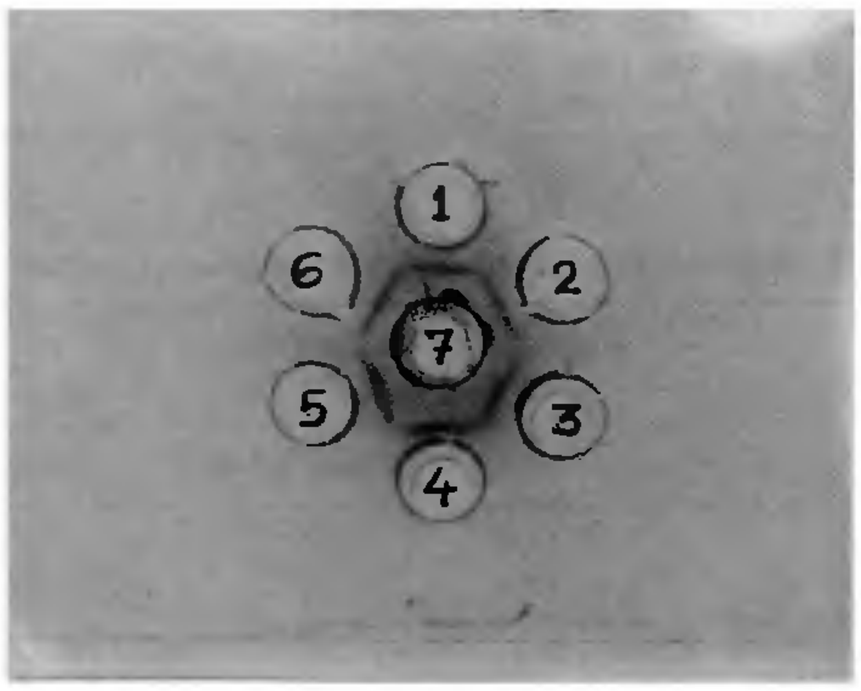

Figure 1. Immunological identity of the NCT with candidate live oral cholera vaccine strains. Ouchterlony immunodiffusion analysis of concentrated CF of CT gene-negative *V. cholerae* strain X-392 (well 1) and vaccine strains JBK 70 (well 2), CVD 104 (well 3), CVD 110 (well 4), CVD 101 (well 5), CVD 105 (well 6) against antiserum of X-392 enterotoxin (NCT) (well 7).

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Deformation tectonics of the diffuse Indo-Australian plate boundary using centroid moment tensor data

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The deformation tectonics of the Indo-Australian plate boundary has been fairly well explained by the Euler pole models. The Harvard centroid moment tensor (CMT) data concur with all the essential features described by these models. Additionally, it indicates the presence of widespread left lateral strike-slip faulting in the northern portion of the deformation zone from the Central Indian ridge up to the northern Ninety-east ridge, which incidentally is found to agree with the model of 'Wrench Fault Tectonics'. This is evidenced by the presence of focal mechanisms in this region with a consistent left lateral strike-slip faulting along NE–SW fault planes which are also continuous with the trend of the transform faults at the Central Indian ridge. However, it appears that this shearing phenomenon only complements the overall deformation process, but cannot explain it independently.

SEISMICITY in the north-eastern Indian ocean has for long been considered to be too high to be intraplate. Gutenberg and Richter[1] first reported this anomaly followed by other seismicity studies of this region[2–13].

Sykes[4] proposed the development of a nascent island arc between Sri Lanka and Australia to explain the unusual seismicity, which was refuted by later workers[6,7]. Stein and Okal[6] suggested a major left lateral strike-slip motion along the Ninety-east ridge as a result of greater resistance of the western part of the Indo-Australian