IMD's forecast	Actual performance of 1991 monsoon
•Monsoon will set over Kerala in the first week of June 1991	•Monsoon broke over Thiruvananthapuram on 2 June 1991 and set over Kerala in the first week of June 1991
•Total rainfall for the season (June to September 1991) for the country as a whole: 94% of the long-period average value with model error within ± 4%, that is, within 90% to 98% of the long period average value	•Season's total rainfall for the country as a whole: 92.6% of the long-period average value
•About 75% of the 35 meteorological subdivisions likely to receive normal or excess rainfall	•Meteorological subdivisions with normal or excess rainfall: 80% (28 out of 35)
•Overall performance will be within the definition of normal monsoon (± 10%) but on the lower side of normal range	•Overall monsoon on the lower side but within the definition of normal monsoon

Assam and Meghalaya came into the normal category in the second half of the season. Nagaland, Mizoram, Manipur and Tripura also reached normal-rainfall status by the end of the season. However, J&K, HP, hills of West UP, Haryana, Rajasthan, and Saurashtra and Kutch are in the deficient category at the end of the season, although the deficiency is not large.

Thus, on the whole, the performance of the 1991 monsoon was within the definition of normal but on the lower side of the normal range, as was forecast by IMD.

1. Gowariker, V. *et al.*, *Mausam*, 1989, **40**, 115.
2. Gowariker, V. *et al.*, *Mausam*, 1991, **42**, 125.
3. Rangarajan, S., *Curr. Sci.*, 1991, **60**, 622.
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RESEARCH NEWS

Bacterial enterotoxins: a structural view

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Diarrhoeal diseases, of which cholera is the most feared, have flourished in the Indian subcontinent and the less-developed areas of the world. Oral rehydration therapy and improved medical care have to a great extent mitigated the consequences of these diseases. The recent reemergence of cholera in South America is a dangerous portend for the future. The origins of research in cholera and related diarrhoeal diseases date back to the last century, but a clear understanding of the factors mediating bacterial pathogenicity was achieved only in the last thirty years. In pioneering studies far ahead of their time, Sambhu Nath De, working in Calcutta in the fifties, established that the watery diarrhoea of cholera was indeed triggered by a soluble bacterial enterotoxin (see *Current Science* special issue, 1990, vol. 59, nos. 13&14). De was also able to show that strains of *E. coli* could also cause diarrhoea, although he did not pursue this work to the logical conclusion of demonstrating the presence of a soluble toxin in culture filtrates[1]. Later the identity of the cholera and *E. coli* enterotoxins as complex proteins was established[2], and a comparison of amino-acid sequences demonstrated a high degree of homology (~80%) between the two proteins[3]. Biochemical work clearly established that both toxins consist of one A subunit (M_r ~27,000) and five B subunits (M_r ~11,600), which together occur as a multimeric AB_5 complex, with a total molecular mass of 86,000 daltons. A schematic model for such a molecule is shown in Figure 1 (ref. 4). Now, almost four decades after De's seminal work, comes the first definitive report[5] of the three-dimensional structure of a diarrhoea-causing toxin, the heat-labile enterotoxin of *E. coli*.

Wim Hol and his colleagues at the University of Groningen, The Netherlands, describe the crystal structure of *E. coli* enterotoxin (LT) at a resolution of 2.3 Å. They worked with a protein purified from porcine *E. coli*, overcoming problems arising from non-isomorphism even between native crystals, and report a model that contains all 515 residues of the B-pentamer, 230 residues of the A-subunit, and 163 solvent molecules. The ribbon diagram in Figure 2 clearly illustrates the folding of the polypeptide chain of the AB_5 complex. How well does the present 3D structure allow us to visualize the various interactions involving the toxin? It has already been known for several years that, after release from the bacterial cell, the toxin binds to target intestinal cells. This association is through a specific interaction between the B-subunits and the gangliosides GM1 on the external surface of the intestinal cell membrane. A portion of the A-subunit is then transferred across the membrane bilayer, a process that requires 'proteolytic nicking' between residues A192 and A195, which results in fragments A1 (~ 192 residues) and A2 (~ 45 residues). The portion transferred across the membrane is the catalytically active A1 polypeptide, which then ADP-ribosylates an arginine residue of a regulatory protein component of the adenylate cyclase system in a complex process that requires other factors. As a consequence of this enzymatic modification the adenylate cyclase remains stimulated, in turn elevating levels of intracellular cyclic AMP, which then results in an efflux of ions and fluids from the cell[6]. The consequences are only too well known to anyone who has been infected with diarrhoea-causing *E. coli*.

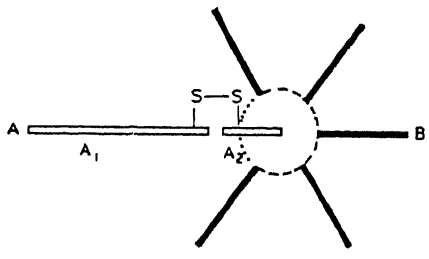

Figure 1. Schematic model of cholera toxin structure. (From ref. 4)

The crystal structure reveals many details likely to be of great interest to workers in the field. The five B subunits are arranged so as to generate a central