78, 109 and 111, as well as Pro 36 and Gly 46 (Fig. 2*c*; numbering as in TGF-β2). All the cysteines are involved in the disulphide pattern of TGF-β2 as well as in the intermolecular disulphide bridge between the monomers. Amino acid 46 in the eight-residue ring has to be a Gly because of steric hindrance and because of the atypical conformation angles ($\phi = 135.1°$, $\psi = 160.9°$). Proline 36 was found to be the only *cis*-Pro in TGF-β2 and is necessary for ending the long loop and continuing in a β-strand. Many of the amino acids involved in the dimer interface are invariant or show conservative changes in most members of the TGF superfamily. We therefore propose the general fold, including the TGF-β knot, of all the proteins of the TGF superfamily to be the same with variations in the loop regions. The five more closely related members of the TGF subfamily (TGF-β1 to TGF-β5) show an overall sequence homology of 64 to 76% (compared to TGF-β2). The residues involved in the helix–sheet interactions or in the hydrogen-bonding network with the water molecules in the interface are invariant or show conservative changes. Of the 29 amino acids of TGF-β2 that are involved in the dimer interface (Fig. 2*b*), 24 are absolutely invariant in all five isoforms. Conservative changes were observed for three positions (residues 43 (Phe to Tyr), 58 (His to Tyr) and 74 (Ala to Ile)), whereas only two amino acids (residues 57 (Gln to Thr) and 68 (Ile to His)) show nonconservative changes. Both variable residues are located in the long α-helix H_3. This might lead to small positional changes of the α-helix H_3 of one subunit and the β-sheet of the other subunit. All other varying residues are found fairly evenly distributed over the whole surface of the dimer. The general fold of all five TGF-β isoforms, including the TGF-β knot, is therefore likely to be the same and only small changes are expected. The fact that heterodimers TGF-β1.2 (refs 1, 16), as well as TGF-β2.3 (ref. 16), have been isolated from natural sources support this proposal.

The first crystal structure of a protein of the TGF superfamily reveals a new fold including a very unusual disulphide pattern, the TGF-β knot. With this information, other three-dimensional structures of TGF isoforms or even of more distantly related proteins could be solved and modelled. Future work must include the analysis of the TGF-β receptor complexes. This will help us understand the biochemical roles of these members of the TGF superfamily and will help in the development of new therapeutic agents, such as for wound healing, bone formation and immune modulation. □

Received 14 May; accepted 17 July 1992.

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ACKNOWLEDGEMENTS. We thank J. P. Priestle for discussions during structure determinaion and critical review of this manuscript, N. Cerletti, D. A. Cox and G. K. McMaster for providing TGF-β2 and for discussions, K. von Meyenburg for encouragement and support for this project, M. Carvajal for technical assistance in the crystallization experiments and F. Winkler for bringing the structure of the potato inhibitor of carboxypeptidase A to our attention. The coordinates will be deposited in the Brookhaven Data Bank.

CORRECTIONS

Cloning of cDNAs for Fanconi's anaemia by functional complementation

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Nature **356,** 763–767 (1992)

WE have discovered that the sequence of our *FACC* gene in Fig. 1*c* is in error. The corrected sequence appears below, starting at position 787 of the cDNA.

GAG	CGA	GTG	GCG	TCC	CTG	TCA	CGA	GTT
E	R	V	A	S	L	S	R	V
TGT	GTC	CCA	CTT	ATT	ACC	CTG	ACA	
C	V	P	L	I	T	L	T	

The change does not alter any of the conclusions of the paper. The length of the open reading frame remains 557 amino acids. No new significant homologies to the cDNA sequence nor to the translated protein were detected in GenBank, EMBL or Swiss-Prot databases, nor were any new functional motifs detected with the corrected sequence. The correct version has been submitted to the EMBL database. The accession number is X66184. We regret any inconvenience caused by this mistake.

A new type of synthetic peptide library for identifying ligand-binding activity

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Nature **354,** 82–84 (1991)

IN this paper we inadvertently omitted to cite the work of Fukura and colleagues (A. Fukura, F. Sebestyen, M. Asgedom and G. Dibo *14th Int. Congr. Biochem.* FR013; 1988), who independently described a similar synthetic method for producing multiple peptide sequences (which we called "split synthesis"). However, Fukura *et al.* did not describe the concept of 'one bead, one peptide' which was central to our approach.