THE COLLAGEN OF THE INTERVERTEBRAL DISC IN ADOLESCENT IDIOPATHIC SCOLIOSIS

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A defect in the collagenous matrix of the intervertebral disc has been proposed as a contributory factor in the pathogenesis or progression, or both, of the scoliotic deformity. In an attempt to resolve these questions the collagen extractability and distribution across normal and scoliotic discs was investigated. The collagen content of the scoliotic nucleus pulposus was found to be higher than normal, particularly at the apex of the curve, but no consistent correlation was found with the spinal mobility or degree of curvature. The collagen content of the annulus fibrosus from scoliotic discs was shown to be abnormally distributed, again only in those discs encompassed by the curve. Since these abnormalities in absolute collagen distribution are dependent on location within the spine it is considered that they represent a consequence of the curvature rather than the cause. These results contrast with the pepsin extractability of collagen in the annulus fibrosus, which was abnormal in all the scoliotic discs examined, and found to be independent of location. While the precise interpretation of these latter findings is complex, it would seem that a subtle defect in collagen does exist within the scoliotic disc which, coupled with extraspinal influences, may play an important role in progression of the scoliotic curve.

By far the most common form of scoliosis is the adolescent idiopathic curvature, for which the precise aetiology has not been established. A genetic background to this disorder has been clearly demonstrated (Riseborough and Wynne-Davies 1973) but the mode of expression remains obscure. Thus, while initiation of the curve would appear to be genetically determined in many instances, other factors—for example, growth rates—are clearly implicated in its progression.

Scoliosis is a feature of some inherited disorders of connective tissue in which abnormalities of collagen have been demonstrated, and these have been reviewed by Uitto and Lichtenstein (1976). In Ehlers–Danlos syndrome Type VI a hydroxylysine deficiency has been observed (Pinnell *et al.* 1972; Sussman *et al.* 1974) which results in a defect in the intermolecular cross-linking of collagen (Eyre and Glimcher 1972). Collagen metabolism is also impaired in Marfan's syndrome (Krieg and Müller 1977) where there are high levels of urinary hydroxyproline (Laitinen *et al.* 1968; Priest, Moinuddin and Priest 1973). In homocystinuria, collagen in the skin is more soluble than normal and a cross-linking defect, due to the accumulation of homocysteine, has been demonstrated (Kang and Trelstad 1973; Siegel 1975). A disturbance in the types of collagen synthesised seems to be implicated in the clinical manifestations of osteogenesis imperfecta (Penttinen *et al.* 1975; Sykes, Francis and Smith 1977), along with a defect in cross-linking (Fujii *et al.* 1977; Trelstad, Rubin and Gross 1977). In the severe form of osteogenesis imperfecta, in which scoliosis is common, there is a decreased stability of polymeric collagen in the skin (Smith, Francis and Bauze 1975).

In contrast to the scoliosis associated with these syndromes, the idiopathic type is not generally considered to be a manifestation of connective-tissue disorder. There is, however, some recent evidence to suggest that both the collagen and glycosaminoglycans of some connective tissues in scoliosis are abnormal. Francis, Sanderson and Smith (1976) examined the stability of polymeric collagen of the skin from patients with idiopathic and congenital scoliosis. The stability of the polymeric collagen from adolescents with idiopathic scoliosis was found to be significantly less than that of control subjects, except for two patients with mature skeletons, where the polymeric collagen was normal. One of three patients with congenital scoliosis also had polymeric collagen of low stability.

Glycosaminoglycans in serum (Balaba 1972; Kaz'min and Merkur'eva 1971) and in the intervertebral disc (Pedrini, Ponseti and Dohrman 1973; Ghosh *et al.* 1979) are reported to be abnormal in idiopathic

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