PublisherInfo		
PublisherName	:	BioMed Central
PublisherLocation	:	London
PublisherImprintName	:	BioMed Central

FAK, fibronectin and paxillin in chondrogenic cell condensation

ArticleInfo		
ArticleID	:	117
ArticleDOI	:	10.1186/ar-2001-66877
ArticleCitationID	:	66877
ArticleSequenceNumber	:	74
ArticleCategory	:	Paper Report
ArticleFirstPage	:	1
ArticleLastPage	:	3
ArticleHistory	:	RegistrationDate : 2001–1–15 OnlineDate : 2001–1–15
ArticleCopyright	:	Biomed Central Ltd2001
ArticleGrants	:	
ArticleContext	:	130753311

Lillian Shum,[Aff1]

Aff1 NIAMS, NIH, Bethesda, USA

Keywords

Chondrogenesis, condensation, FAK, fibronectin, integrin

Context

Chondrogenesis is a multi-step process that encompasses an initiation phase during which chondroprogenitor cells are recruited, followed by processes of proliferation, cell condensation and nodule formation, and culminates in overt differentiation. Cell condensation, the aggregation of a dispersed population of cells, is an essential stage during which cell - cell and cell - matrix interactions produce orchestrated signaling within and among precartilage cells directed towards differentiation. In many other differentiation systems, matrix signaling, with focal adhesion complex assembly and cytoskeletal rearrangement are pivotal events. The authors hypothesized that, during chondrogenesis, activation and assembly of focal adhesion complex are necessary steps for precartilage cell condensation.

Significant findings

Using chick wing bud mesenchymal cells at Hamburger-Hamilton stage 23/24, cell growth and condensation were observed to increase and peak at day 3 of culture, followed by cartilage differentiation. The timing of maximal cell condensation coincided with elevated expression of fibronectin and its receptor, a5 ?1 integrin, and increased phosphorylation of focal adhesion kinase (FAK). Phosphorylated FAK was associated with fibronectin, paxillin and src, which in turn was associated with phosphatidylinositol 3-kinase and phospholipase C-?. Upregulation of FAK phosphorylation upon transforming growth factor (TGF)-?1 or insulin treatment of mesenchymal cells resulted in increased levels of associated fibronectin and paxillin, consistent with published data which indicates that these growth factors support and enhance cartilage growth. Conversely, downregulation of FAK phosphorylation correlated with decreased levels of associated proteins. These data suggest that phospho-activation of FAK allowed focal adhesion complex assembly which was required for mesenchymal cell condensation and subsequent chondrocyte differentiation.

Comments

Numerous studies have provided us with a schema of the 'what, when and where' as they relate to chondroprogenitor cell condensation, cell shape changes, and subsequent differentiation. This paper focuses on the assembly of associated proteins in focal adhesion complexes during chondrogenesis, and contributes to our current understanding of the players in this process. It will be interesting and challenging to decipher the mechanisms of cell condensation, the hierarchy of the signaling cascades, and the causes and consequences of these molecular interactions.

Methods

Alcian Blue staining for cartilage differentiation; Alcian blue colorimetry; peanut agglutinin staining for cell condensation; immunoblotting; immunoprecipitation; tritiated thymidine incorporation; ^{35}S sulfate incorporation; primary cell culture; chick wing bud mesenchymal cells in a high density micromass culture system

References

1. Bang O-S, Kim E-J, Chung J-G, Lee S-R, Park T-K, Kang S-S: Association of focal adhesion kinase with fibronectin and paxillin is required for precartilage condensation of chick mesenchymal cells. Biochem Biophys Res Commun. 2001, 278: 522-529.