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Molecular mechanism of the non-classical secretion of the human fibroblast growth factor

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Fibroblast growth factors (FGFs) are cytokines which play a crucial role in the regulation of key cellular processes such as cell proliferation, cell differentiation, wound healing and tumor growth. Unlike many proteins, prototype FGFs, FGF-1 & FGF-2, lack the N-terminal signal sequence which is required for secretion through the classical ER-Golgi pathway. The secretion of the prototype FGFs is facilitated by two other calcium binding proteins, namely, S100A13 and p40-synaptotagmin (p40-Syt). Our studies reveal that copper (Cu^{2+}) is critical for the secretion of FGFs through the ER-Golgi-independent non-classical secretion pathway. Cu^{2+} is observed to be important for the formation of a multiprotein FGF release complex (MRC). The Cu^{2+} binding sites are located in both S100A13 and p40-Syt. Mutational studies indicate that the C-terminal segment in S100A13 provides the binding interface for FGF. Our study elucidates the sequence of molecular events that occur in the non-classical secretion of FGFs.

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