

Role of basigin in adaptor protein mediated signaling pathways

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Abstract. The potential role of basigin/EMMPRIN in activation of NF- κ B and AP-1 signaling pathway was investigated. The MyD88- and TRIF-dependent activation of NF- κ B signaling pathways were inhibited by basigin specific antibody and siRNA. TRIF dependent AP-1 activation was also inhibited by basigin specific antibody and siRNA. Taken together, the current study suggests that basigin regulates the activation of MyD88- and TRIF-dependent signaling pathways, and warrants further investigation in these basigin mediated cellular processes.

Keywords: Basigin, Signal transduction, NF- κ B, MyD88, TRIF, AP-1

1 Introduction

Basigin is involved in a variety of pathophysiological functions including regulation of lymphocyte responsiveness and cellular proliferation. The interaction between basigin and cyclophilin A, for example, initiates activation of ERK 1/2 signaling and induces the expression of IFITM1 through ERK and PIK [1-2]. Basigin and syndecan association induces activation of MAPK, which promotes cell adhesion and chemotaxis [3]. Recently we reported basigin cascades a survival signaling through Wnt/ β -catenin-dependant in CSC-like cells [4]. These evidences demonstrate that basigin plays a key regulatory role in cell signaling related with inflammatory diseases and cancers. Herein, we have investigated the potential role of basigin in MyD88 and TRIF adaptor protein dependent activation of signaling pathway. We have identified that basigin induces MyD88- or TRIF-dependent activation of NF- κ B and AP-1. To our knowledge, this is the first time to report the role of basigin in adaptor protein mediated activation of signal transduction, and provides additional insights of intracellular events of dynamic nature of basigin.

2 Materials and Methods

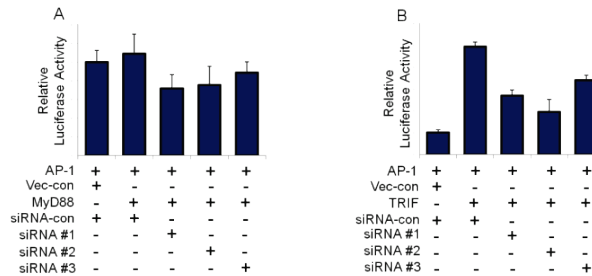


Fig. 2. Effect of basigin on AP-1 activation mediated by MyD88 (A) and TRIF (B). Data shown are representative of 3 independent experiments as described for Figure 1.

TRIF dependent, but independent of MyD88, as reported for TLR signaling pathway [7]. Current study provides the first evidence that basigin is coordinately associated with AP-1 activation in the TRIF-dependant manner.

In summary, our results show that basigin-mediated activation of signaling pathways is through either MyD88- or TRIF-dependent manner, and provides new insight into the basigin-mediated cellular functions.

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