Activation of aryl hydrocarbon receptor suppresses invasion of esophageal squamous cell carcinoma cell lines

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ABSTRACT

Aims and background. Esophageal cancer is the eighth most common malignancy and sixth leading cause of cancer deaths in the world. Recent studies have shown the potential role of the aryl hydrocarbon receptor (AhR) in tumor development; however, little is known about its role in esophageal squamous cell carcinoma. In the present study, we evaluated AhR expression in esophageal tumor tissues as well as cell lines and investigated the effects of AhR activation by its agonist BNF on esophageal squamous cell carcinoma invasion using Eca109 and TE-13 cells as a model.

Methods. Western blotting was performed to detect the AhR and CYP1A1 protein expression. Transwell migration assays were carried out to study the effects of BNF on esophageal squamous cell carcinoma cell invasion. AhR-specific siRNA was used to knock down the expression of AhR protein.

Results. Our results showed that AhR was highly expressed in esophageal squamous cell carcinoma tissues and cell lines when compared with its expression in normal tissue. AhR siRNA robustly decreased AhR protein expression in both Eca109 and TE-13 cells. BNF significantly inhibited invasion of human esophageal squamous cell carcinoma cells via activation of AhR.

Conclusions. The obtained results provide critical information on the roles of BNF in mediating esophageal squamous cell carcinoma invasion. This information could be useful for future therapeutic intervention in this lethal human disease.

Key words: AhR, esophageal squamous cell carcinoma, BNF, invasion.

The authors declare that they have no competing interests.

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