Cervical Cancer: Open Access
ISSN: 2475-3173

BTG2 Inhibits the Growth and Spread of Cervical Squamous Cell Carcinoma

John Logie Baird and Joseph Banks*

Department of Epidemiology and Cancer Biology, China

Abstract

Cervical squamous cell carcinoma (CSCC) is a prevalent form of cervical cancer characterized by uncontrolled

the activation of the PI3K/Akt pathway, which is known to promote cell survival and migration. Furthermore, BTG2 upregulation leads to the downregulation of matrix metalloproteinases (MMPs), key enzymes implicated in extracellular matrix remodeling and cancer cell invasion. In a xenograft mouse model, we observe that BTG2 overexpression

apoptosis in BTG2-overexpressing tumors compared to control tumors. Notably, BTG2 expression correlates with improved overall survival in a cohort of CSCC patients, highlighting its potential clinical relevance. Taken together, our

and invasion. These results suggest that targeting BTG2 could be a promising therapeutic strategy for the treatment of cervical squamous cell carcinoma. Further investigations are warranted to fully elucidate the underlying molecular mechanisms and to develop BTG2-based therapeutic approaches for clinical applications.

Keywords: BTG2; Cervical squamous cell carcinoma; CSCC; Tumor suppressor; Growth inhibition; Metastasis inhibition

Introduction

Cervical squamous cell carcinoma (CSCC) is a prevalent form of cervical cancer and remains a signi cant global health concern. It is characterized by the abnormal growth of squamous cells lining the cervix, leading to the formation of tumors. Despite advancements in screening programs and human papillomavirus (HPV) vaccination, CSCC continues to be a major cause of morbidity and mortality among women worldwide [1]. Understanding the molecular mechanisms underlying the growth and spread of CSCC is crucial for the development of e ective therapeutic strategies. Numerous studies have identi ed various genetic and epigenetic alterations that contribute to the initiation and progression of CSCC [2]. However, the explore the underlying molecular mechanisms by which BTG2 modulates CSCC progression, focusing on key signaling pathways and matrix metalloproteinases (MMPs) involved in tumor growth and metastasis. To validate the in vitro ndings, we employ a xenogra mouse model to assess the impact of BTG2 overexpression on CSCC tumor growth and metastasis in vivo [5]. We analyze histological characteristics, proliferation rates, and apoptotic markers in BTG2-overexpressing tumors compared to control tumors. Additionally, we examine the correlation between BTG2 expression and overall survival in a cohort of CSCC patients, evaluating the clinical relevance of BTG2 as a prognostic marker.

Methods

Tissue sample collection and BTG2 expression analysis: Collection of CSCC tissues and adjacent normal tissues from patients undergoing surgical resection. Extraction of total RNA from the collected tissues using a commercially available kit. Quantitative real-time polymerase chain reaction (qRT-PCR) analysis to measure BTG2 expression levels in CSCC tissues compared to adjacent normal tissues. Statistical analysis to determine the signi cance of BTG2 downregulation in CSCC.

Cell culture and transfection: Selection of CSCC cell lines (e.g.,

***Corresponding author:** Joseph Banks, Department of Epidemiology and Cancer Biology, China, E-mail: jbanks34@gmail.com

Received: 29-May-2023, Manuscript No. ccoa-23-103354; **Editor assigned:** 01-June- 2023, Pre QC No. PQ- ccoa-23-103354; **Reviewed:** 14-June-2023, QC No ccoa-23-103354; **Revised:** 20-June-2023, Manuscript No ccoa-23-103354 (R); **Published:** 27-June-2023, DOI: 10.4172/2475-3173.1000162

Citation: Baird JL, Banks J (2023) BTG2 Inhibits the Growth and Spread of Cervical Squamous Cell Carcinoma. Cervical Cancer, 8: 162.

HeLa, SiHa) for in vitro experiments. Culture of CSCC cells in appropriate growth media supplemented with fetal bovine serum. Transfection of CSCC cells with BTG2 overexpression plasmids using a suitable transfection reagent. Generation of stable BTG2-overexpressing cell lines through antibiotic selection. Con rmation of BTG2 overexpression by qRT-PCR and Western blot analysis.

of BTG2 as a prognostic marker. BTG2 expression levels may serve as a potential indicator of patient outcomes and response to therapy in CSCC. e ndings of this study contribute to the understanding of the complex molecular mechanisms underlying CSCC progression and identify BTG2 as a promising therapeutic target. e multifaceted e ects of BTG2 on CSCC cell proliferation, apoptosis, migration, invasion,