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ELEVENTH ANNUAL MEETING

Therapeutic Potential of Niclosamide in Suppressing Immune Escape during the Progression of Diffuse Large B-Cell Lymphoma Carcinogenesis

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INTRODUCTION

Diffuse large B-cell lymphoma (DLBCL) is a common type of non-Hodgkin lymphoma (NHL) with an increasing incidence in recent years. Immune escape is an essential characteristic of cancer development and metastasis. Niclosamide (NIC) is an oral antiparasitic drug that has been found to block immune checkpoints and is believed to have potential in cancer treatment.

AIM

This study aims to investigate whether NIC therapy can mediate DLBCL carcinogenesis by regulating immune escape.

1. Analyzed DEGs in microarray datasets for exploring the potential pathways of DLBCL progression after NIC treatment.
2. Explored the potential pathway for the immune escape of DLBCL mediated by PD-L1 after NIC treatment.
3. Validated whether NIC treatment could modulate PD-L1 mediated immune escape and the significance of target genes in pathway in DLBCL cells.

METHOD

DLBCL is classified into germinal center B-cell like (GCB) DLBCL and activated B-cell like (ABC) DLBCL. ABC DLBCL is characterized more chemo-resistant and poor prognosis than GCB DLBCL. Two cell lines SU-DHL-2 (ABC-like DLBCL) and SU-DHL-4 (GCB-like DLBCL) were used for validation.

ACKNOWLEDGEMENTS

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CONTACT INFORMATION

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RESULTS

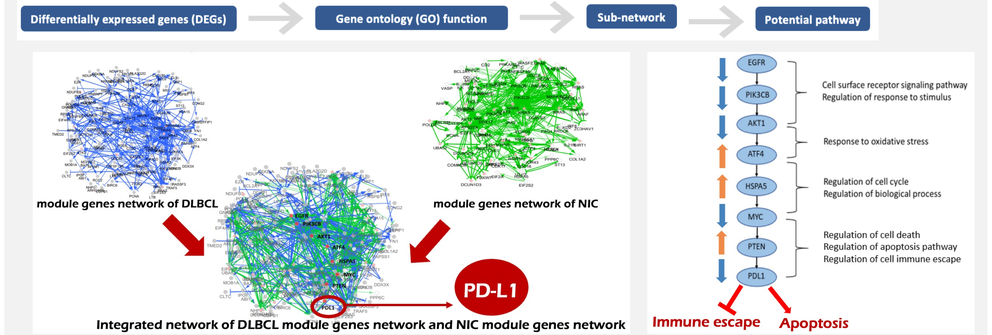


Fig. 1. A potential pathway of DLBCL progression underlying NIC treatment by analysis of microarray databases.

Fig. 2. A potential pathway of DLBCL progression underlying NIC treatment via PD-L1 regulation.

RESULTS

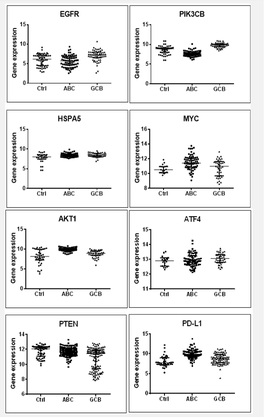


Fig. 3. Differential expression of target gene expression in potential pathway between ABC and GCB patients.

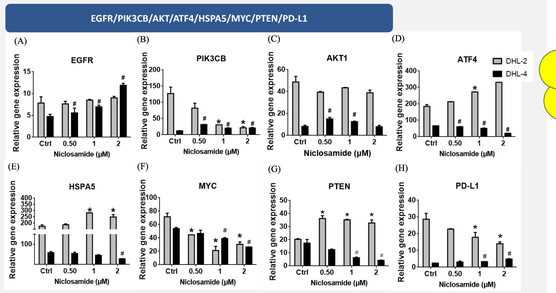


Fig. 4. Expression of target genes in two DLBCL cells with NIC treatment.

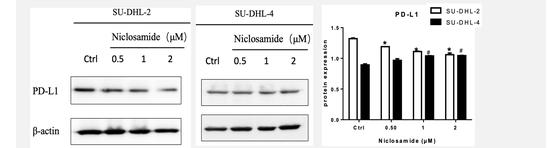
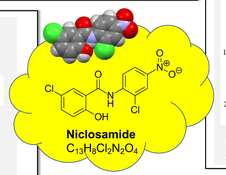


Fig. 5. Protein level of PD-L1 in two DLBCL cells with NIC treatment.



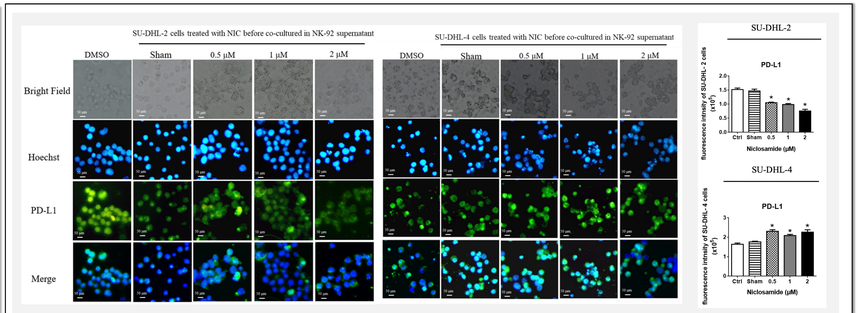


Fig. 6. Immunofluorescence PD-L1 level in DLBCL cells with NIC treatment before co-cultured in NK-92 supernatant.

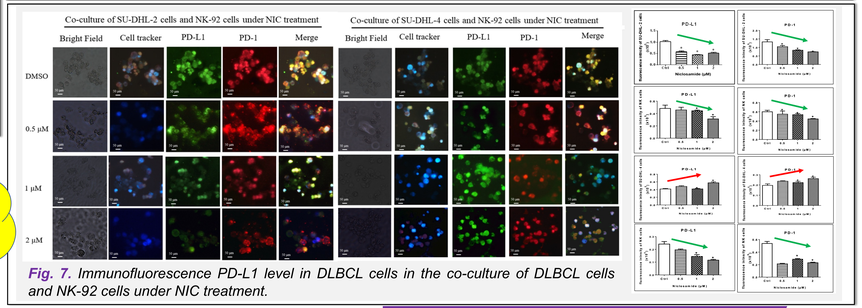


Fig. 7. Immunofluorescence PD-L1 level in DLBCL cells in the co-culture of DLBCL cells and NK-92 cells under NIC treatment.

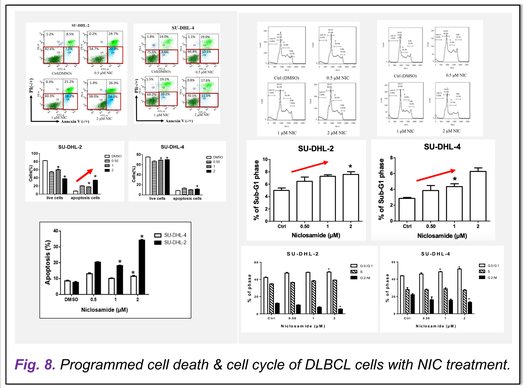
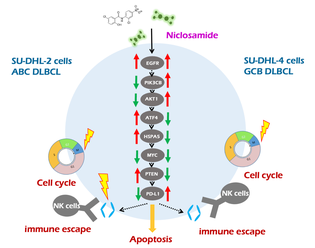


Fig. 8. Programmed cell death & cell cycle of DLBCL cells with NIC treatment.

CONCLUSIONS



This study found that NIC inhibits immune escape in SU-DHL-2 cells and induces cell apoptosis by regulating PD-L1 expression, leading to reduced cell proliferation. NIC has the potential to be used in DLBCL treatment.