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**Re: Association between intestinal neoplasms and celiac disease - beyond celiac disease and more**

Okumura K. Letter to intestinal neoplasms and celiac disease

Kenji Okumura

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**Author contributions:** Okumura K performed writing the paper.

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**Abstract**

The association between celiac disease and enteropathy-associated T cell lymphoma has been known. The pathogenesis of the development of malignant neoplasms remains limited. In addition to celiac disease, we believe that other underlying mechanisms contribute to the developing malignant neoplasms.

**Key Words:** Celiac disease; Entropathy-associated T cell lymphoma; c-MYC; JAK-STAT

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**Core Tip:** The pathogenesis of enteropathy-associated T cell lymphoma (EATL) remains limited. This letter suggests oncogene mutations were reported and would be pertinent to develop malignant neoplasms in EATL.

**TO THE EDITOR**

I read with great interest the paper by Wang *et al*[1] in the issue 13 of World Journal of Gastrointestinal Oncology, a review article regarding the association between intestinal neoplasms and celiac disease. The authors showed that the total risk of small bowel cancer (SBC) and enteropathy-associated T cell lymphoma (EATL) increased in celiac disease (CD) patients. I have agreed with the authors opinions and they mainly mentioned EATL type I, which is associated with CD. The pathogenesis of EATL remains limited, however, as the authors mentioned in the manuscript that CD disrupts cell-level regulation and chronic intestinal inflammation, which leads to the proliferation of intestinal intraepithelial lymphocytes. The presence of chronic inflammation leads to increase the turnover of cell cycle and contribute to the development of neoplasm due to gene mutation in oncogenes or tumor suppressor genes in EATL.

We previously showed that c-myc mutation was seen in EATL type 2[2]. Our findings support that gene mutation is one of the factors developing malignant neoplasm in the absence of celiac disease. JAK/STAT3 signaling pathway was also reported as the main drivers of CD associated lymphomagenesis[3]. JAK/STAT pathway regulates MYC expression[4], which lead to proliferation of malignant cells.

CD is one of the significant gastrointestinal diseases and increases the risk of malignant neoplasms. In addition to CD, we believe that other underlying mechanisms contribute to the developing malignant neoplasms[3]. We believe that these facts would be a helpful to understand CD and EATL and these findings are highly pertinent and provide a context that helps understand those reported by Wang *et al*[1].

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**Footnotes**

**Conflict-of-interest statement:** The authors have declared no conflicts of interest.

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