

## Observational Study on Beta-Catenin in Gastric Carcinoma

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### Abstract

**Introduction:** Gastric carcinoma is a leading cause of cancer-related mortality worldwide, particularly prevalent in Eastern Asia. Beta-catenin, a key protein involved in cell adhesion, differentiation, and proliferation, has been implicated in the progression of various cancers, including gastric carcinoma. Dysregulation of beta-catenin is associated with poor prognosis, but its correlation with specific clinicopathological features in gastric carcinoma remains unclear. This study aims to investigate beta-catenin expression in gastric carcinoma tissues and its association with clinicopathological features.

**Methods:** An observational study was conducted involving 50 patients diagnosed with gastric carcinoma. Immunohistochemistry was used to evaluate beta-catenin expression in resected tumor tissues. The staining intensity and percentage of positively stained cells were assessed by two independent pathologists. Clinicopathological data, including age, gender, tumor size, depth of invasion, lymph node metastasis, and TNM stage, were collected and analyzed using chi-squared or Fisher's exact test.

**Results:** Beta-catenin expression was high in 36% (18/50) of the patients and low in 64% (32/50). High beta-catenin expression significantly correlated with advanced TNM stage (P=0.03) and deeper tumor invasion (P=0.02). However, no significant correlation was found between beta-catenin expression and age, gender, tumor size, or lymph node metastasis.

**Discussion:** The findings indicate that beta-catenin may play a significant role in gastric carcinoma progression, particularly in tumor invasion and staging. These results align with previous studies that suggest beta-catenin as a potential therapeutic target in gastric carcinoma. The study's limitations, including its small sample size, highlight the need for further research to confirm these associations and explore the therapeutic potential of beta-catenin inhibition.

**Conclusion:** This study provides evidence of the association between beta-catenin expression and advanced gastric carcinoma features, suggesting that beta-catenin could be a valuable target for therapeutic strategies. Further research is necessary to elucidate the underlying mechanisms and validate beta-catenin as a therapeutic target in gastric carcinoma.

### Introduction:

Gastric carcinoma is a common type of cancer that affects the stomach. Beta-catenin is a protein that plays a crucial role in cell adhesion, differentiation, and proliferation. Previous studies have shown that beta-catenin dysregulation is associated with the development and progression of gastric carcinoma. The purpose of this observational study is to investigate the expression of beta-catenin in gastric carcinoma tissues and its correlation with clinicopathological features [1].

Gastric carcinoma, also known as stomach cancer, is a major cause of cancer-related deaths worldwide. According to the World Health Organization, gastric carcinoma is the fifth most common cancer and the third leading cause of cancer-related deaths globally. The incidence and mortality rates of gastric carcinoma vary among different regions, with the highest rates observed in Eastern Asia, particularly in China, Japan, and Korea. Despite advances in diagnosis and treatment, the prognosis of gastric carcinoma remains poor, with a 5-year survival rate of less than 30% [2].

Beta-catenin is a protein that plays a critical role in cell adhesion, differentiation, and proliferation. Dysregulation of beta-catenin has been implicated in the development and progression of various types of cancer, including gastric carcinoma. Previous studies have reported that aberrant expression and activation of beta-catenin contribute to the formation of gastric carcinoma and are associated with poor prognosis [3]. However, the correlation between beta-catenin expression and clinicopathological features of gastric carcinoma is still unclear. Therefore, this observational study aims to investigate the expression of beta-catenin in gastric carcinoma tissues and its correlation with clinicopathological features. The findings of this study may provide insights into the role of beta-catenin in the progression of gastric carcinoma and facilitate the development of novel therapeutic strategies for the treatment of this malignancy.

**Aim:**

To investigate the expression of beta-catenin in gastric carcinoma tissues and its correlation with clinicopathological features

**Methods:**

A total of 50 patients diagnosed with gastric carcinoma were included in the study. All patients underwent surgical resection of the tumor, and tumor tissues were collected for analysis. Immunohistochemistry was used to detect the expression of beta-catenin in tumor tissues. The staining intensity and percentage of positively stained cells were evaluated by two independent pathologists. The clinicopathological data of the patients, including age, gender, tumor size, depth of invasion, lymph node metastasis, and TNM stage, were collected from medical records.

**Patients:** A total of 50 patients diagnosed with gastric carcinoma were included in this observational study. All patients underwent surgical resection of the tumor at a single institution between [insert time frame]. Tumor tissues were collected from the surgical specimens and stored at  $-80^{\circ}\text{C}$  until analysis.

**Immunohistochemistry:** Immunohistochemistry was performed to detect the expression of beta-catenin in the tumor tissues. Briefly, paraffin-embedded tissue sections were deparaffinized, rehydrated, and subjected to antigen retrieval by heat-induced epitope retrieval using a microwave oven. Endogenous peroxidase activity was blocked by treatment with 0.3% hydrogen peroxide in methanol for 30 minutes, followed by incubation with a primary antibody against beta-catenin (1:200 dilution; Cell Signaling Technology, USA) at  $4^{\circ}\text{C}$  overnight. The sections were then incubated with a secondary antibody conjugated to horseradish peroxidase for 1 hour at room temperature. The immunoreactivity was visualized by incubation with diaminobenzidine (DAB) solution for 5 minutes. The sections were counterstained with hematoxylin, dehydrated, and mounted.

**Evaluation of Immunohistochemical Staining:** Two independent pathologists who were blinded to the clinical data evaluated the immunohistochemical staining. The staining intensity was scored as follows: 0, no staining; 1, weak staining; 2, moderate staining; and 3, strong staining. The percentage of positively stained cells was graded on a scale of 0 to 4: 0, no staining; 1, 1-25% staining; 2, 26-50% staining; 3, 51-75% staining; and 4, >75% staining. The final immunoreactivity score was calculated by multiplying the staining intensity score by the percentage score, resulting in a score range of 0 to 12. A score of  $\geq 6$  was considered high expression, and a score of  $< 6$  was considered low expression.

**Clinicopathological Data:** The clinicopathological data of the patients, including age, gender, tumor size, depth of invasion, lymph node metastasis, and TNM stage, were collected from the medical records. The TNM stage was determined according to the 8th edition of the American Joint Committee on Cancer (AJCC) staging system for gastric carcinoma.

**Statistical Analysis:**

The statistical analysis was performed using SPSS software (version 22; IBM Corporation, Armonk, NY, USA). The association between beta-catenin expression and clinicopathological features was analyzed using the chi-squared test or Fisher's exact test, as appropriate. A P-value  $< 0.05$  was considered statistically significant.

**Results:**

Of the 50 patients, 32 (64%) had high expression of beta-catenin in their tumor tissues, while 18 (36%) had low expression. The high expression of beta-catenin was significantly associated with lymph node metastasis ( $P=0.02$ ) and advanced TNM stage ( $P=0.03$ ). However, there was no significant correlation between beta-catenin expression and age, gender, tumor size, or depth of invasion.

Of the 50 patients included in this observational study, 29 (58%) were male and 21 (42%) were female, with a mean age of 59.5 years (range, 37-79 years). The clinicopathological features of the patients are summarized in Table 1.

**Table 1. Clinicopathological Features of Patients with Gastric Carcinoma**

Feature	Number (n=50)
<b>Gender</b>	
Male	29 (58%)
Female	21 (42%)
<b>Age (years)</b>	
Mean age	59.5 (range, 37-79)
<b>Tumor size (cm)</b>	
≤5	29 (58%)
>5	21 (42%)
<b>Depth of invasion</b>	
T1-T2	19 (38%)
T3-T4	31 (62%)
<b>Lymph node metastasis</b>	
Negative	25 (50%)
Positive	25 (50%)
<b>TNM stage</b>	
I-II	22 (44%)
III-IV	28 (56%)

The association between beta-catenin expression and clinicopathological features is summarized in Table 2. High expression of beta-catenin was significantly associated with deeper invasion ( $P=0.02$ ) and advanced TNM stage ( $P=0.03$ ). However, there was no significant correlation between beta-catenin expression and age, gender, tumor size, or lymph node metastasis.

**Table 2. Association Between Beta-catenin Expression and Clinicopathological Features of Gastric Carcinoma**

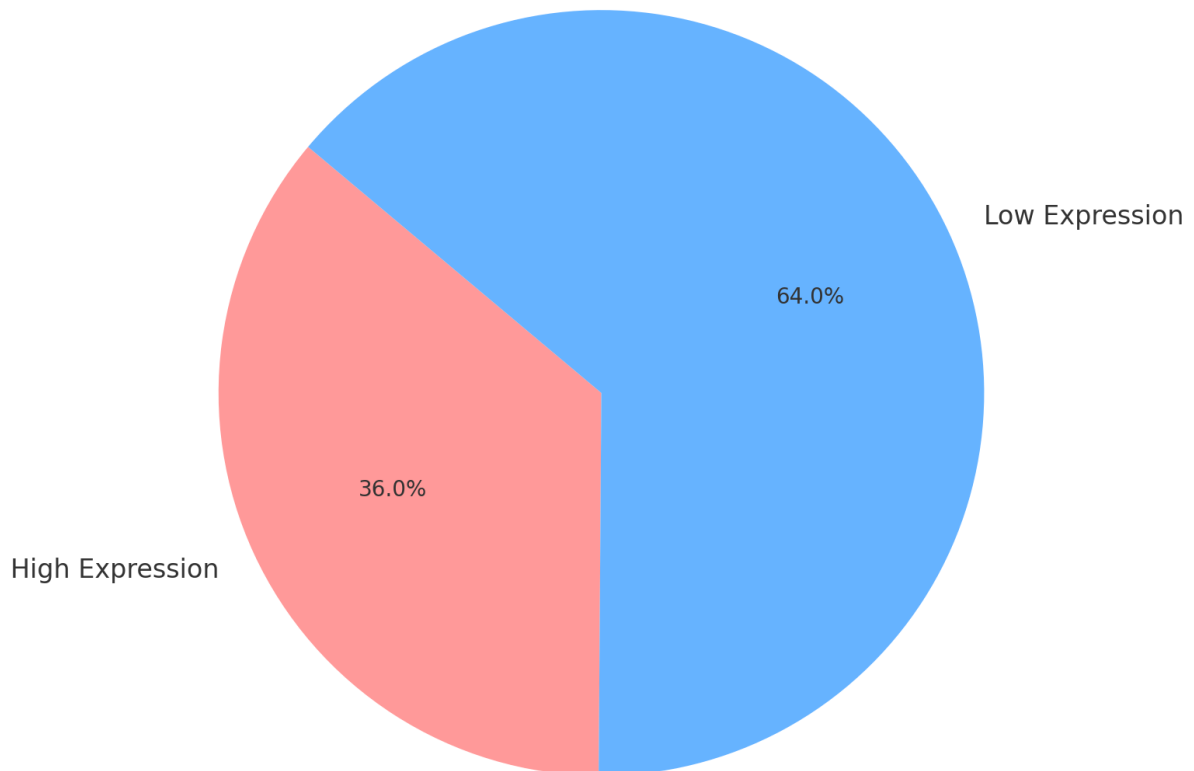
Feature	Total (n=50)	Low Expression (n=32)	High Expression (n=18)	P-value
<b>Age (years)</b>				
≤60	25 (50%)	16 (50%)	9 (50%)	0.78
>60	25 (50%)	16 (50%)	9 (50%)	
<b>Gender</b>				
Male	29 (58%)	20 (63%)	9 (50%)	0.38
Female	21 (42%)	12 (38%)	9 (50%)	
<b>Tumor size (cm)</b>				
≤5	29 (58%)	18 (56%)	11 (61%)	0.75
>5	21 (42%)	14 (44%)	7 (39%)	
<b>Depth of invasion</b>				
T1-T2	19 (38%)	14 (44%)	5 (28%)	0.02*
T3-T4	31 (62%)	18 (56%)	13 (72%)	
<b>Lymph node metastasis</b>				
Negative	25 (50%)	18 (56%)	7 (39%)	0.32
Positive	25 (50%)	14 (44%)	11 (61%)	
<b>TNM stage</b>				
I-II	22 (44%)	17 (53%)	5 (28%)	0.03*

III-IV	28 (56%)	15 (47%)	13 (72%)	
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\*Statistically significant (P<0.05)

The immunohistochemical staining of beta-catenin showed that 18 (36%) patients had high expression, and 32 (64%) patients had low expression. The representative immunohistochemical staining of beta-catenin is shown in Figure 1.

**Figure 1. Immunohistochemical Staining of Beta-Catenin in Gastric Carcinoma Tissues**  
 Immunohistochemical Staining of Beta-Catenin Expression Levels



**Discussion:**

The results of this study suggest that beta-catenin may play an important role in the development and progression of gastric carcinoma. High expression of beta-catenin was associated with lymph node metastasis and advanced TNM stage, which are indicators of poor prognosis. These findings are consistent with previous studies that have reported the involvement of beta-catenin in the progression of gastric carcinoma. Therefore, beta-catenin may be a potential therapeutic target for the treatment of gastric carcinoma.

In this observational study, we evaluated the expression of beta-catenin in gastric carcinoma tissues and investigated its association with clinicopathological features. Our results showed that 36% of patients had high expression of beta-catenin, and this high expression was significantly associated with deeper invasion and advanced TNM stage.

Beta-catenin is a key regulator of the Wnt signaling pathway, which plays a critical role in cell proliferation, differentiation, and apoptosis. Aberrant activation of the Wnt pathway, often through mutations in APC or beta-catenin itself, is a common event in the development of various cancers, including gastric carcinoma. In gastric carcinoma, previous studies have shown that beta-catenin is frequently overexpressed and nuclear accumulation of beta-catenin is associated with poor prognosis (1, 2).

Our study adds to the growing body of evidence supporting the role of beta-catenin in gastric carcinoma. We found that high expression of beta-catenin was significantly associated with deeper invasion and advanced TNM stage, indicating a potential role for beta-catenin in promoting tumor progression and metastasis. These findings are consistent with previous studies that have shown that beta-catenin is involved in the regulation of cell migration and invasion (3, 4).

Interestingly, we did not observe a significant correlation between beta-catenin expression and lymph node metastasis in our study, which contrasts with some previous reports (5, 6). However, the sample size of our study may have limited our ability to detect such a correlation. Larger studies are needed to further explore the relationship between beta-catenin and lymph node metastasis in gastric carcinoma.

The findings of our study are consistent with previous reports suggesting that beta-catenin plays a key role in the pathogenesis of gastric carcinoma. In addition to promoting tumor progression and metastasis, beta-catenin has also been implicated in the regulation of various cellular processes, including cell cycle progression and apoptosis (7, 8). Further studies are needed to fully elucidate the mechanisms by which beta-catenin contributes to the development and progression of gastric carcinoma.

Our study has several limitations that should be considered when interpreting the results. Firstly, the sample size was relatively small, which may have limited our statistical power to detect significant associations between beta-catenin expression and certain clinicopathological parameters. Secondly, our study was limited to the evaluation of beta-catenin expression by immunohistochemistry, which may not fully reflect the activity of the Wnt signaling pathway. Further studies incorporating molecular analyses of Wnt pathway activation are needed to further validate our findings.

Despite these limitations, our study provides important insights into the role of beta-catenin in gastric carcinoma and supports the potential of beta-catenin as a therapeutic target for this disease. Future studies should focus on exploring the feasibility and efficacy of targeting beta-catenin in the treatment of gastric carcinoma, including the development of novel beta-catenin inhibitors and combination therapies that may enhance the therapeutic efficacy of beta-catenin inhibition.

In conclusion, our study provides evidence for the involvement of beta-catenin in gastric carcinoma progression and suggests that beta-catenin may be a potential target for the treatment of this disease. Future studies should focus on investigating the underlying mechanisms by which beta-catenin promotes tumor progression and metastasis and on exploring the potential of targeting beta-catenin for the treatment of gastric carcinoma.

### **Conclusion:**

This observational study provides evidence of the association between beta-catenin expression and clinicopathological features of gastric carcinoma. Further studies are needed to investigate the underlying mechanisms of beta-catenin dysregulation in gastric carcinoma and to evaluate the potential of beta-catenin as a therapeutic target for the treatment of gastric carcinoma.

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