# TMCO1在胃癌中高表达与患者不良预后相关并通过抑制调亡促进肿瘤恶性进展

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**摘要:目的** 探究跨膜和卷曲螺旋结构域1(TMCO1)在胃癌组织中的表达,明确其对胃癌患者预后的影响,并分析其机制。**方法** 基于癌症公共数据库和我院行胃癌根治术的患者临床资料,分析TMCO1在胃癌中的表达情况和对胃癌进展及其预后的影响。利用KEGG和GO分析其可能的生物学功能和作用机制。采用慢病毒载体构建TMCO1高表达和沉默的胃癌细胞株(HGC-27),并以空载处理的胃癌细胞作为对照,体外实验观察其对胃癌细胞凋亡、增殖、侵袭和迁移能力的影响。**结果** TMCO1在胃癌组织中表达升高($P$<0.05),高表达TMCO1与胃癌恶性进展参数呈正相关($P$<0.001),且TMCO1高表达组的5年生存率低于低表达组($P$<0.05)。富集分析结果显示,TMCO1可能通过Wnt信号影响胃癌细胞凋亡。CCK-8结果显示,上调胃癌细胞系TMCO1的表达促进肿瘤细胞的增殖($P$<0.05),下调反之($P$<0.05);流式细胞术结果显示,TMCO1高表达组的胃癌细胞凋亡率低于TMCO1沉默组($P$<0.05);划痕和Transwell实验结果显示,上调TMCO1的表达增加胃癌细胞的迁移($P$<0.05)和侵袭能力($P$<0.05)。免疫印迹结果显示,上调TMCO1高表达增加β-catenin的水平($P$<0.05),下调反之($P$<0.05)。**结论** TMCO1在胃癌组织中表达升高,促进胃癌患者的恶性进展并影响远期预后,其可能和激活Wnt/β-catenin信号抑制胃癌细胞凋亡有关。

**关键词:**胃癌;跨膜和卷曲螺旋结构域1;凋亡;预后;Wnt/β-catenin

## Elevated TMCO1 expression in gastric cancer is associated poor prognosis and promotes malignant phenotypes of tumor cells by inhibiting apoptosis

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**Abstract: Objective** To investigate the impact of high expression of transmembrane and coiled helix structural domain 1 (TMCO1) on prognosis of gastric cancer and the possible mechanisms. **Methods** TMCO1 expression in gastric cancer and its effect on gastric cancer progression and prognosis were analyzed using publicly available databases and clinical data of patients undergoing radical surgery in our hospital, and its possible biological functions were explored using KEGG and GO analyses. In gastric cancer HGC-27 cells, the effects of lentivirus-mediated TMCO1 overexpression and TMCO1 silencing on cell apoptosis, proliferation, invasion and migration were examined. **Results** TMCO1 expression was significantly elevated in gastric cancer tissues ($P$<0.05), and its high expression was positively correlated with cancer progression ($P$<0.001) and a lowered postoperative 5-year survival rate of the patients ($P$<0.05). Bioinformatic analyses suggested that TMCO1 may affect gastric cancer cell apoptosis via Wnt signaling. In HGC-27 cells, TMCO1 overexpression significantly promoted tumor cell proliferation, inhibited cell apoptosis, and enhanced cell migration and invasion, whereas TMCO1 silencing produced the opposite effects. Western blotting showed that β-catenin levels were significantly upregulated in TMCO1-overexpressing cells and downregulated in cells with TMCO1 silencing. **Conclusion** TMCO1 is overexpressed in gastric cancer tissues, and its high expression promotes gastric cancer progression and affects long-term prognosis of the patients possibly by activating the Wnt/β-catenin signaling pathway to inhibit apoptosis of gastric cancer cells.

**Keywords:** gastric cancer; transmembrane and coiled-coil domain 1; apoptosis; prognosis; Wnt/β-catenin

胃癌在全球的发病率排名第2,死亡率居于第5[1,2]。尽管一部分早期胃癌患者可以通过手术获得根治,新的放、化疗方案也在不断推陈出新[3-5],但我国胃癌患者的总体生存率仍然仅有35.2%[6]。胃癌细胞具有抑制凋亡而获得异常生存能力的特征,这是导致胃癌难以治疗和预后不良的重要原因之一[7,8]。虽然胃癌细胞拮抗凋亡的机制尚不明确,但当前靶向凋亡的生物制剂在临床上取得的初步疗效提示其进一步研究的价值[9,10]。膜卷曲螺旋结构域1(TMCO1)是一种内质网跨膜蛋白,其被证实在前列腺癌、卵巢癌等中高表达并导致预后不良[11-14],在乳腺癌的研究中发现TMCO1参与了肿瘤细胞拮抗

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药物诱导的癌细胞凋亡作用[15]。然而，TMCO1在胃癌中的表达及其作用尚未见报道，其是否参与调控胃癌细胞凋亡过程也有待研究。本文系统评估TMCO1在胃癌中的表达情况、对肿瘤恶性进展和预后生存的影响，并通过实验探讨TMCO1对胃癌细胞凋亡的影响及其可能的作用机制。

## 1 资料和方法

### 1.1 研究对象及分组

从我院病案系统中筛选2015年1月~2018年12月的胃癌患者共104例，对其展开回顾性研究。纳入标准：确诊为原发性胃癌；癌症未发生远处转移；成功实施胃癌R0根治性切除术；临床资料完整。排除标准：患者术后5年内死于胃癌以外的因素；合并有其他器官组织来源的恶性肿瘤。从病理科调取所有患者的蜡块，进行免疫组织化学实验，逐个测定癌组织中TMCO1的表达水平，将所有患者按照所得数据的中位数分组，大于中位数的52例为高表达组，低于中位数的52例为低表达组。本研究严格遵循医学伦理规范，研究方案已获得本院伦理委员会批准（伦理批号：2023KY028）。

### 1.2 资料采集

通过我院电子病例系统收集患者资料。基本信息：性别、年龄和联系电话等；病理信息：肿瘤大小、组织病理类型、T分期、N分期、临床分级、癌胚抗原CEA、糖类抗原19-9（CA19-9）等；术后生存情况：以电话随访和复查的方式，收集患者术后5年的生存状况以及死亡原因，截止时间为2024年1月。

### 1.3 癌症相关数据库分析TMCO1表达水平及其对生存期的影响

TIMER2.0数据库（timer. cistrome. org）分析TMCO1在人体常见肿瘤中的表达情况。采用GEPIA数据库（gepia.cancer-pku.cn）分析TMCO1在胃癌组织和癌旁组织中的表达情况。通过UALCAN数据库（https://ualcan.path.uab.edu/），分析TMCO1的表达量和胃癌恶性进展参数之间的关系。采用Kaplan-Meier Plotter数据库（kmplot.com）预测TMCO1的表达量对患者生存期的影响；通过cBioPortal数据库（www.cbioportal.org）检索TMCO1共表达基因，并将其整合至DAVID数据库（davidbioinformatics. nih. gov）进行KEGG和GO功能富集分析，以预测其潜在的生物学功能和作用机制，最后整理数据上传至Bioinformatics网站（www.bioinformatics.com.cn），构建可视化富集结果图谱。

### 1.4 免疫组化检测TMCO1和Ki67表达

从病理科调取患者的组织蜡块（包括术中切除的癌变组织和邻近癌旁的正常组织），将组织蜡块切成厚度3 μm的切片，温水摊片，载玻片捞出后65 ℃下烘烤3 h，随后依次进行脱蜡水化、抗原修复液修复、内源性过氧化物酶阻断、山羊血清封闭后，4 ℃孵育一抗［TMCO1（1∶200，武汉三鹰）；Ki67（1∶200，Abcam）］过夜[11]，室温孵育二抗（酶标山羊抗兔IgG聚合物，1∶2000）2 h、DAB显色、苏木精复染、分化、返蓝、封片。随后用玻片扫描仪（OLYMPUS SLIDEVIEW VS200）扫描成电子图像，并使用Image J软件对图像进行处理，测定目标蛋白的相对积分光密度（IOD）值[16]。

### 1.5 细胞培养和稳转细胞系的构建

提前配制RPMI 1640完全培养基，包含10%胎牛血清、100 U/mL青霉素和0.1 mg/mL链霉素，将人胃癌细胞系HGC-27置于培养基，并在37 ℃和5%浓度的$CO_2$环境下培养。待细胞生长至对数生长期，按$10^5$/孔铺至无菌6孔板中，再制备过表达TMCO1、沉默TMCO1以及空载对照的慢病毒浓缩液。加入慢病毒浓缩液处理细胞12 h，更换新鲜的完全培养基继续常规培养3 d。第4天滴加1 μg/mL的嘌呤霉素筛选，持续7 d；稀释存活细胞并接种至96孔板，继续加入1 μg/mL嘌呤霉素筛选，14 d后转入无菌6孔板进行扩增，待细胞稳定生长后，收集3组细胞：对照组（Vector）、TMCO1过表达组（OE-TMCO1）和TMCO1沉默组（sh-TMCO1）[17, 18]。

### 1.6 Western blotting检测

提取各组HGC-27细胞总蛋白，使用BCA法进行蛋白定量，各组蛋白经变性、SDS-PAGE凝胶电泳、转膜、5%脱脂牛奶封闭后，加入一抗：anti-TMCO1（1∶200，武汉三鹰）、anti-Ki67（1∶200，Abcam）、anti-PCNA（1∶1000，Abcam），以及内参蛋白anti-β-actin（1∶3000，Abcam）于4 ℃冰箱孵育过夜[19]；次日取出恢复室温，TBST洗膜后二抗（辣根酶标记山羊抗兔/鼠IgG聚合物，1∶3000，中杉金桥）室温孵育1 h，TBST洗膜后采用ECL化学法显影。使用Image J软件分析蛋白条带的灰度值，以上实验独立重复3次。

### 1.7 流式细胞实验

收集3组的HGC-27细胞，依据Annexin V-FITC/PI Apoptosis Detection Kit（Vazyme）的操作说明，进行细胞染色和流式细胞仪上机检测，Flow Jo软件分析早期凋亡（Annexin $V^+$/$PI^-$）的细胞比例[20, 21]。

### 1.8 CCK-8实验

将3组的HGC-27细胞按$1\times10^3$/孔接种于96孔板中，分别在接种后24、48、72和96 h，加入10 μL/孔的CCK-8试剂，放入培养箱中孵育2 h，使用酶标仪测定吸光度$A_{450\,nm}$。

### 1.9 细胞划痕和Transwell实验检测侵袭迁移

细胞划痕实验：取各组细胞，均匀的接种在无菌6孔板中（$5\times10^5$/孔），加入2 mL完全培养基后，37 ℃、

5%$CO_2$条件下培养，次日吸去培养基，用200 μm的无菌枪头划线，PBS轻柔洗去漂浮细胞，加入无血清培养基培养24 h。分别在初始时间（0 h）和24 h后拍照采集划痕区域的图像[22]。

Transwell实验：Transwell上室（Corning）加入200 μL含有2×10⁴细胞的无血清培养基，下室加入1000 μL含10%胎牛血清的培养基。培养箱培养48 h后，依次经过多聚甲醛固定、结晶紫染色后，置于显微镜下观察并拍照。侵袭实验：预先用Matrigel基质胶（Corning）包被上室膜，其余操作步骤与迁移实验相同[23]。

### 1.10 统计学分析

使用GraphPad Prism 9.0和SPSS 27软件进行统计分析。定性资料以$n$(%)表示，组间比较采用$\chi^2$检验；定量资料以均数±标准差表示，组间比较采用$t$检验或ANOVA检验；术后5年生存率采用Kaplan-Meier（K-M）生存曲线呈现，组间比较采用Log-rank检验；相关性分析采用Spearman检验；分析影响预后的危险影响因素采用单因素和多因素Cox回归分析；受试者工作特征曲线（ROC）用于评估TMCO1对患者术后5年生存率的预测价值[24]。$P$<0.05为差异具有统计学意义。

## 2 结果

### 2.1 胃癌组织中TMCO1高表达且与Ki67呈正相关

癌症数据库TIMER2.0分析显示，TMCO1在乳腺癌、胆管癌等多种人体常见恶性肿瘤中表达升高（$P$<0.001，图1A）；GEPIA数据库显示，在胃癌组织中，TMCO1表达高于癌旁组织（$P$<0.05，图1B）。免疫组化分析显示，TMCO1在胃癌组织中高表达（$P$<0.05，图1C、D），且细胞增殖核抗原Ki67的表达量与其呈正相关（$r$=0.626，$P$<0.001，图1E~H）。

### 2.2 TMCO1的表达量和胃癌恶性进展参数正相关

UALCAN数据库分析显示，TMCO1的表达量和胃癌恶性进展参数呈正相关（$P$<0.001，图2A、B）；患者临床数据显示，TMCO1高表达组中CEA≥5 μg/L、CA19-9≥37 kU/L、T stage3~4及N stage2~3期的比例高于低表达组（$P$<0.001，表1）。

### 2.3 TMCO1高表达降低胃癌患者术后5年生存期

Kaplan-Meier Plotter数据库预测显示，TMCO1高表达胃癌患者的生存期低于低表达组的患者（$P$<0.05，图3A）。临床数据分析显示，相比于低表达组，高表达组的生存率下降（$P$<0.0001，图3B）。ROC生存曲线评估TMCO1相对表达量的预测效能，以2.95为截点值，该生物标志物对患者死亡风险的识别敏感度为68.9%，特异度为93.3%；曲线下面积为0.816（图3C）。单因素和多因素Cox回归分析显示，TMCO1高表达、CEA≥5 μg/L、CA19-9≥37 kU/L、T stage3~4期及N stage2~3是影响患者术后5年生存率的独立危险因素（表2）。

### 2.4 TMCO1影响胃癌细胞的功能学途径和可能机制

KEGG和GO富集分析结果显示，TMCO1的功能可能影响细胞凋亡的过程，以及激活Wnt信号通路有关（图4A、B）。

### 2.5 高表达TMCO1抑制胃癌细胞凋亡促进增殖

免疫印迹检测结果显示，过表达组、沉默组和空白对照组的TMCO1表达差异具有统计学意义（$P$<0.05，图5A、D），表明稳转细胞株构建成功。CCK-8细胞增殖实验结果显示，TMCO1高表达促进胃癌细胞的增殖，沉默TMCO1则呈现出相反结果（$P$<0.05，图5B）。与Vector组相比，增殖相关蛋白Ki67和PCNA的表达量在TMCO1过表达组升高，而在TMCO1沉默组表达降低（$P$<0.05，图5C、D）。流式细胞仪分析凋亡细胞染色，发现过表达TMCO1组细胞的凋亡率较TMCO1-Vector组更低；而沉默TMCO1组的细胞凋亡率相对更高（$P$<0.001，图5F、G）。

### 2.6 TMCO1高表达促进胃癌细胞的侵袭和迁移能力

划痕和Transwell实验显示，过表达的TMCO1促进HGC-27细胞的迁移和侵袭，沉默TMCO1的表达则结果反之（$P$<0.05，图6A~E）。

### 2.7 高表达TMCO1可能通过激活Wnt/β-catenin信号通路调控细胞凋亡

免疫印迹检测结果显示，与Vector组相比，过表达TMCO1组的β-catenin的表达水平升高，沉默TMCO1组的表达量相对减少（$P$<0.05，图7A、B）。

## 3 讨论

本研究首先利用癌症公共数据库初步分析，结合本院胃癌患者临床资料发现TMCO1在胃癌组织中的表达量高于癌旁组织，并影响肿瘤的恶性进展，导致患者的不良预后；体外实验发现，高表达的TMCO1抑制胃癌细胞的凋亡，促进胃癌细胞的恶性增殖、侵袭和迁移，有望为未来胃癌的诊疗提供新的靶点。

TMCO1是新近发现的内质网钙离子通道的关键调控蛋白，参与维持内质网钙稳态的调节[25, 26]，而细胞凋亡、增殖等多种生物学过程都与这一过程密切相关[27, 28]。本研究通过GO富集分析发现，TMCO1与凋亡的进程有关。凋亡是癌症发生发展的重要环节，凋亡受抑制，从而导致肿瘤细胞的数量异常增多[7, 29]。研究表明TMCO1在乳腺癌中高表达，并调节乳腺癌细胞对促凋亡药物的反应[15]。然而TMCO1在胃癌领域的研究尚属未知，本文首次探索了TMCO1的表达在胃恶性肿瘤的作用；此外凋亡是癌症相关的重要生物学过程，在多种恶性肿瘤中报道颇多[30, 31]，然而在胃癌领域中缺乏研究。本研究通过构建慢病毒转染的方式构建过表达

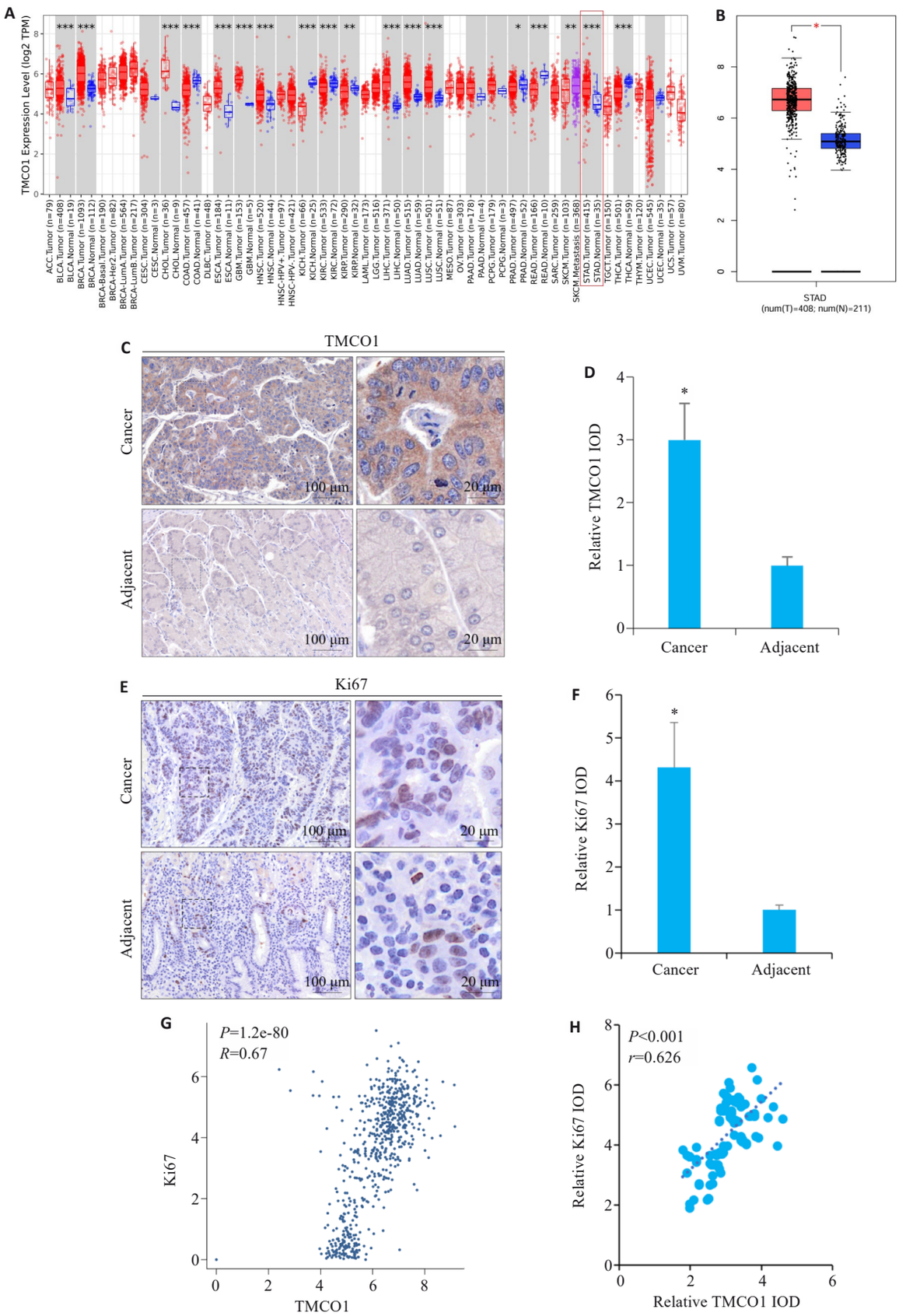


图1 TMCO1在胃癌组织中表达升高且和Ki67表达量正相关

Fig.1 TMCO1 expression is elevated in gastric cancer tissues in positive correlation with the expression of Ki67. **A**: Expression of TMCO1 in different human tumors (**P*<0.05, ***P*<0.01, ****P*<0.001 *vs* Normal). **B**: Expression levels of TMCO1 in gastric cancer (**P*<0.05). **C, D**: Immunohistochemistry for detecting TMCO1 expression in gastric cancer tissues and adjacent tissues and the relative IOD values (**P*<0.05 *vs* adjacent tissue). **E, F**: Immunohistochemistry for detecting Ki67 expression in gastric cancer and adjacent tissues and the relative IOD value (**P*<0.05 *vs* adjacent tissue). **G, H**: Correlation analysis of TMCO1 and Ki67 expressions.

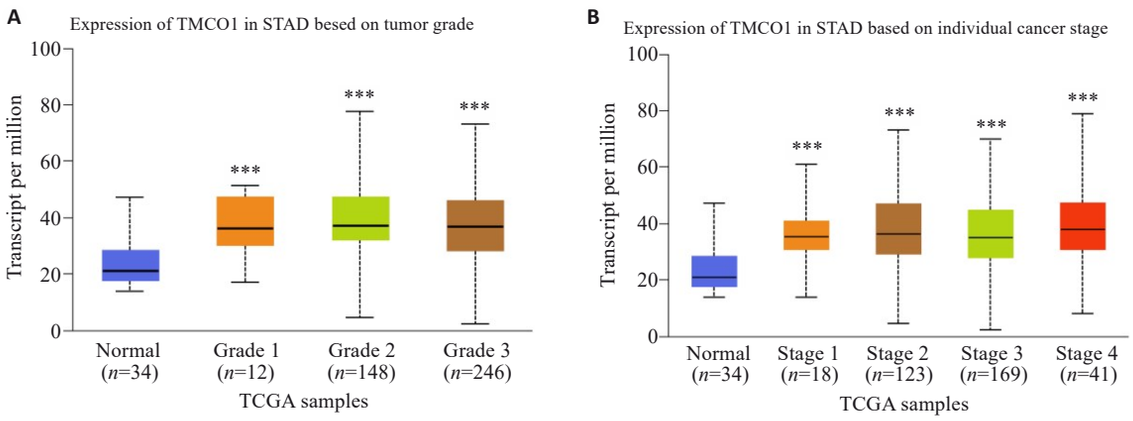


**图2 TMCO1的表达量和胃癌恶性进展参数的相关性**

Fig.2 Correlation between TMCO1 expression level and progression of gastric cancer based on tumor grade (**A**) and stage (**B**). ***$P$<0.001 *vs* Normal.

**表1 TMCO1的表达量与胃癌恶性进展参数的相关性分析**

Tab.1 Correlation between TMCO1 expression levels and parameters of gastric cancer progression

| Factor | $n$ | TMCO1 expression [$n$, (%)] | | $\chi^2$ | $P$ |
|---|---|---|---|---|---|
| | | Low ($n$=52) | High ($n$=52) | | |
| Gender | | | | | |
| Male | 72 | 40 (55.6%) | 32 (44.4%) | 2.889 | 0.089 |
| Female | 32 | 12 (37.5%) | 20 (62.5%) | | |
| Age (year) | | | | | |
| <60 | 49 | 28 (57.1%) | 21 (42.9%) | 1.891 | 0.169 |
| ≥60 | 55 | 24 (43.6%) | 31 (56.4%) | | |
| CEA (μg/L) | | | | | |
| <5 | 40 | 28 (70.0%) | 12 (30.0%) | 10.400 | 0.001 |
| ≥5 | 64 | 24 (37.5%) | 40 (62.5%) | | |
| CA19-9 (kU/L) | | | | | |
| <37 | 38 | 29 (76.3%) | 9 (23.7%) | 16.587 | <0.001 |
| ≥37 | 66 | 23 (34.8%) | 43 (65.2%) | | |
| Tumor size (cm) | | | | | |
| <5 | 46 | 26 (56.5%) | 20 (43.5%) | 1.403 | 0.236 |
| ≥5 | 58 | 26 (44.8%) | 32 (55.2%) | | |
| Cancer cell type | | | | | |
| Adenocarcinoma | 70 | 36 (51.4%) | 34 (48.6%) | 0.175 | 0.676 |
| Other | 34 | 16 (47.1%) | 18 (52.9%) | | |
| T stage | | | | | |
| 1-2 | 37 | 24 (64.9%) | 13 (35.1%) | 5.076 | 0.024 |
| 3-4 | 67 | 28 (41.8%) | 39 (58.2%) | | |
| N stage | | | | | |
| 0-1 | 42 | 27 (64.3%) | 15 (35.7%) | 5.751 | 0.016 |
| 2-3 | 62 | 25 (40.3%) | 37 (59.7%) | | |

和沉默的稳转细胞系，流式细胞术检测发现上调TMCO1可以抑制胃癌细胞的凋亡，免疫印迹和CCK-8实验发现上调TMCO1可以促进胃癌细胞的增殖，细胞划痕和Transwell实验结果表明TMCO1高表达的胃癌细胞的侵袭和迁移能力增强。

既往研究表明，Wnt信号通路是调控胃癌的重要分子途径之一[32-34]。本研究将TMCO1共表达的相关基因进行KEGG富集分析，结果显示TMCO1与Wnt信号通

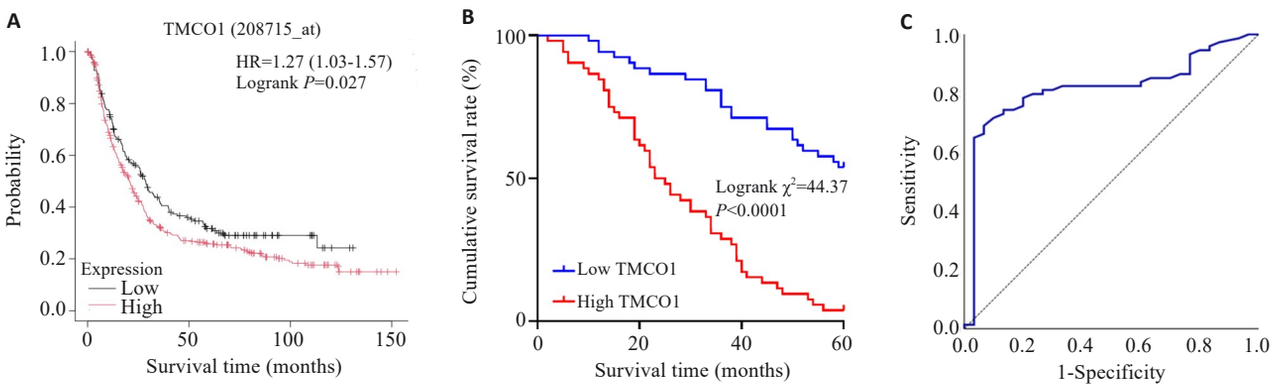


**图3 TMCO1高表达降低胃癌患者术后5年生存率**

Fig.3 High expression of TMCO1 is associated with decreased postoperative 5-year survival rate of gastric cancer patients. **A**: Kaplan-Meier (KM) online platform analysis. **B**: KM survival curves for analyzing clinical data of patients in our hospital. **C**: Predictive value of TMCO1 for 5-year survival after radical gastrectomy.

**表2 单因素和多因素Cox回归分析胃癌患者术后5年生存率的影响因素**

Tab.2 Univariate and multivariate Cox regression analysis of prognostic factors influencing 5-year survival of gastric cancer patients

| Factor | Univariate analysis | | Multivariate analysis | | |
|---|---|---|---|---|---|
| | Log-rank $\chi^2$ | *P* | *HR* | 95% *CI* | *P* |
| Gender (male *vs* female) | 0.041 | 0.840 | | | |
| Age (<60 years *vs* ≥60 years) | 3.189 | 0.074 | | | |
| TMCO1 expression (high *vs* low) | 44.369 | <0.001 | 3.449 | 1.966-6.053 | <0.001 |
| CEA (<5 μg/L *vs* ≥5 μg/L) | 29.662 | <0.001 | 2.513 | 1.394-4.530 | 0.002 |
| CA19-9 (<37 kU/L *vs* ≥37 kU/L) | 33.690 | <0.001 | 2.934 | 1.593-5.405 | <0.001 |
| Tumor size (<5 cm *vs* ≥5 cm) | 3.084 | 0.079 | | | |
| Cancer cell type (adenocarcinoma *vs* other) | 1.336 | 0.248 | | | |
| T stage (T1-T2 *vs* T3-T4) | 20.352 | <0.001 | 2.217 | 1.265-3.887 | 0.005 |
| N stage (N0-N1 *vs* N2-N3) | 26.017 | <0.001 | 2.202 | 1.218-3.981 | 0.009 |

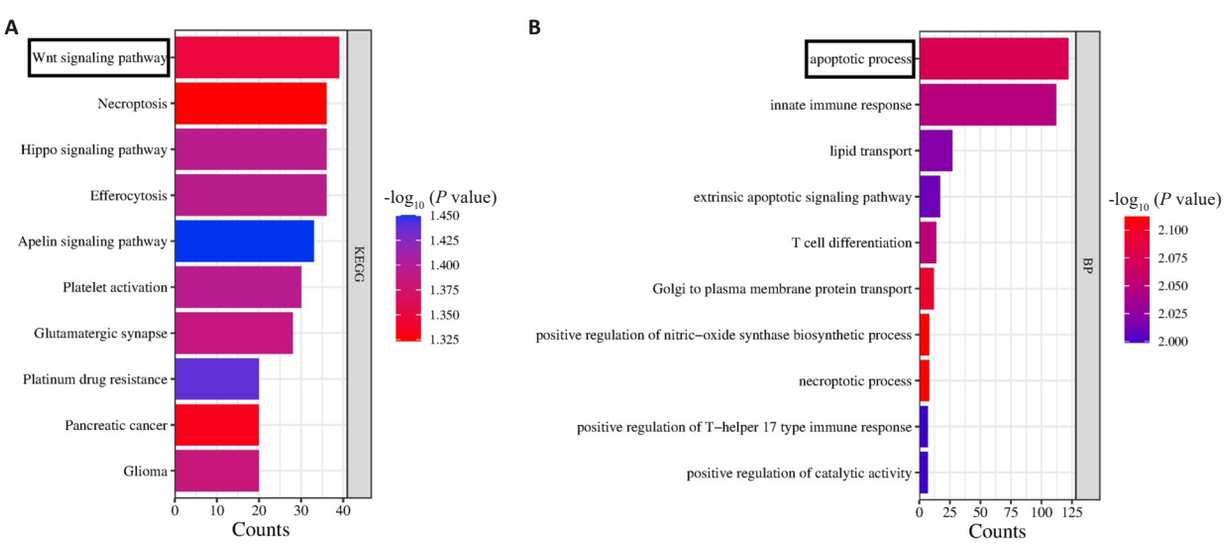


**图4 TMCO1的KEGG和GO富集分析**

Fig. 4 KEGG and GO enrichment analysis of TMCO1. **A**: KEGG enrichment analysis shows that TMCO1 is related to the Wnt signaling pathway. **B**: GO enrichment analysis shows that TMCO1 is associated with cell apoptosis process.

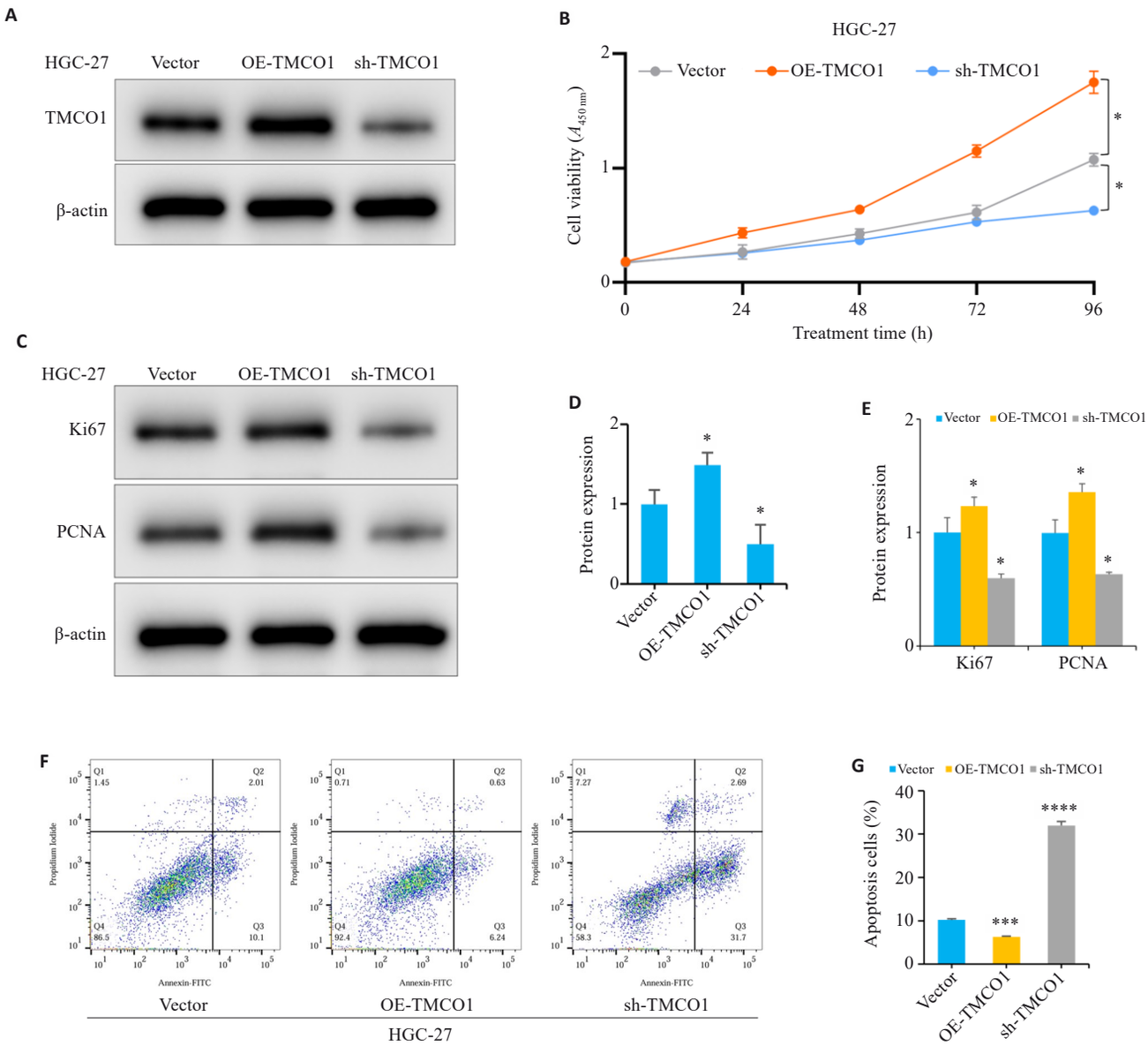


**图5 高表达TMCO1抑制胃癌细胞凋亡并促进细胞增殖**

Fig.5 High expression of TMCO1 inhibits apoptosis and promotes proliferation of gastric cancer cells. **A, D**: Validation of TMCO1 overexpression and TMCO1 silencing in HGC-27 cells. **B**: CCK8 assay for assessing HGC-27 cell proliferation. **C, E**: TMCO1 regulates the expression of proliferation-associated proteins Ki67 and PCNA in HGC-27 cells. **F, G**: Flow cytometry for analyzing apoptosis of HGC-27 cells. **P*<0.05, ****P*<0.001, *****P*<0.0001 *vs* Vector group.

路有关;随后通过免疫印迹法检测蛋白表达量,发现上调TMCO1可以激活Wnt/β-catenin通路的信号传导,β-catenin蛋白的表达量升高,而沉默TMCO1则呈现相反的结果。以上研究结果提示Wnt/β-catenin信号通路的激活可能参与了TMCO1抑制胃癌细胞凋亡的进程。

本研究通过使用TIMER、GEPIA等癌症相关数据库联合患者临床资料综合分析,并通过K-M数据库和追踪患者的随访信息深入调查,得出结论:胃癌组织中TMCO1高表达促进了胃癌患者肿瘤的恶性进展,导致患者的预后不良。体外实验结果解释了其分子生物学的机制:TMCO1高表达抑制胃癌细胞的凋亡,可能通过激活Wnt/β-catenin信号通路调控。

本研究仍存在以下不足:纳入的临床样本量不足;本研究仅探讨了TMCO1与Wnt信号通路之间的关系,然而与肿瘤相关的信号通路还有很多[35, 36],但TMCO1是否可能通过其他的途径共同影响胃癌进展还有待进一步研究;基于本机构的数据,我们发现肿瘤大小的差异无统计学意义,而与其他恶性进展参数(如TNM分期)的相关性仍支持总体结论[37, 38]。这提示TMCO1的表达量在肿瘤大小中的作用有限,或受当前样本量限制,未来需要更深入的研究。

综上,本研究发现TMCO1在胃癌组织中表达量高于正常组织,其影响患者的肿瘤恶性进展提示和预后不良密切相关,机制上可能通过激活Wnt/β-catenin信号通路,参与调控胃癌细胞凋亡的进程。

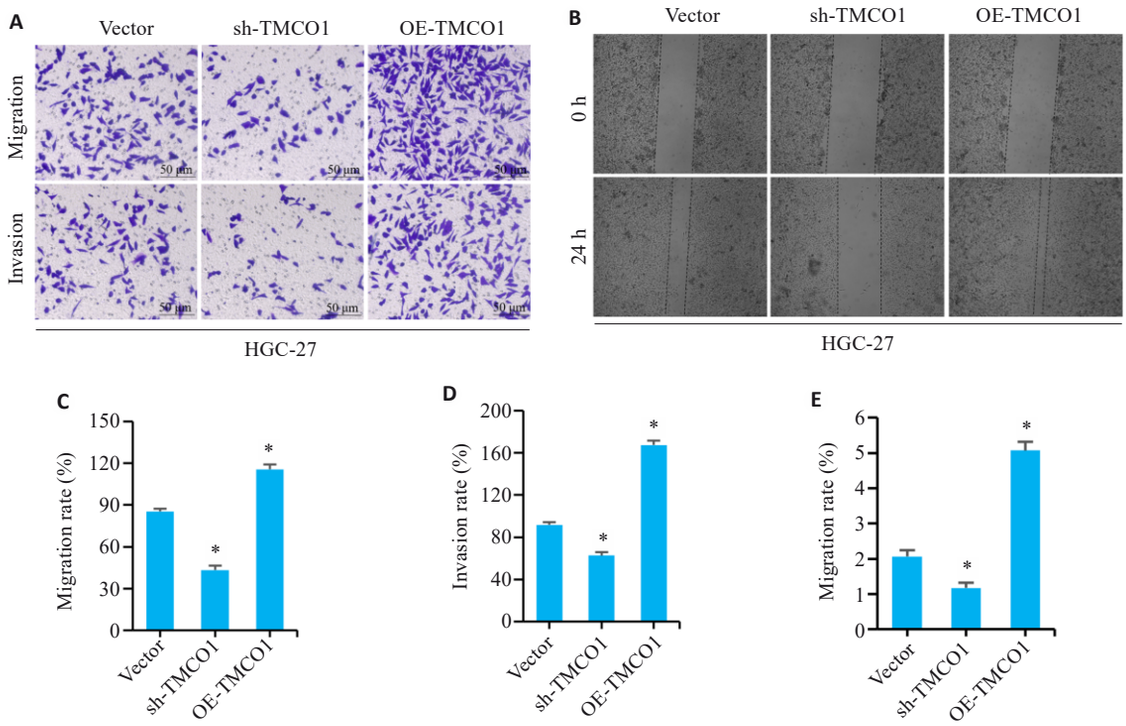


**图6 TMCO1促进胃癌细胞的迁移和侵袭能力**

Fig.6 TMCO1 overexpression promotes migration and invasion of gastric cancer cells. **A, C, D**: Cell migration and invasion of HGC-27 cells. **B, E**: Wound-healing assay. **P*<0.05 *vs* Vector group.

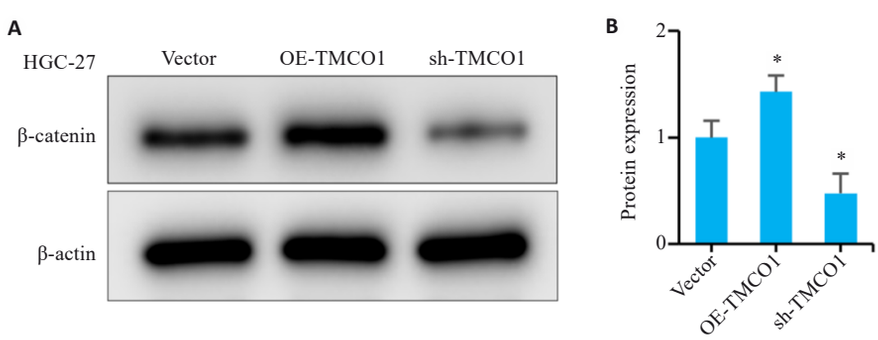


**图7 TMCO1调控胃癌细胞中的Wnt/β-catenin信号通路**

Fig.7 TMCO1 regulates the Wnt signaling pathway in gastric cancer cells. **A**: Detection of β-catenin protein expression in HGC-27 cells with TMCO1 overexpression or silencing by Western blotting. **B**: Analysis of the IOD values of β-catenin in HGC-27 cells. **P*<0.05 *vs* Vector group.

**Declaration of interests:** The authors declare no competing interests.

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