分类号：　　密　　级：
学　号：20222014008　　单位代码：10759

石河子大学

硕 士 学 位 论 文

去氧拉巴醌调控 FOXM1/MYBL2/CDCA3 轴抗食管鳞癌的分子机制研究

学位申请人	祁婧茹
指导教师	王艳明副教授
申请学位类别	医学硕士
专业名称	基础医学
研究方向	病理学与病理生理学
所在学院	医学院

中国·新疆·石河子
2025年5月

分类号：　　密　　级：
学　号：20222014008　　单位代码：10759

石河子大学

硕 士 学 位 论 文

去氧拉巴醌调控 FOXM1/MYBL2/CDCA3 轴抗食管鳞癌的分子机制研究

学 位 申 请 人	祁婧茹
指 导 教 师	王艳明副教授
申 请 学 位 类 别	医学硕士
专 业 名 称	基础医学
研 究 方 向	病理学与病理生理学
所 在 学 院	医学院

中国·新疆·石河子

2025年5月

The Molecular Mechanism of Deoxylapachol in Regulating the FOXM1/MYBL2/CDCA3 Pathway in Esophageal Squamous Cell Carcinoma

A Dissertation Submitted to

Shihezi University

In Partial Fulfillment of the Requirements

for the Degree of

Master of Medicine

By

Jingru Qi

（Pathology and pathophysiology）

Dissertation Supervisor：Assoc. Prof. Yanming Wang

May, 2025

石河子大学学位论文独创性声明及使用授权声明

学位论文独创性声明

本人所呈交的学位论文是在我导师的指导下进行的研究工作及取得的研究成果。据我所知，除文中已经注明引用的内容外，本论文不包含其他个人已经发表或撰写过的研究成果。对本文的研究做出重要贡献的个人和集体，均已在文中作了明确的说明并表示谢意。

研究生签名：邓婧茹　　时间：2015 年 5 月 27 日

使用授权声明

本人完全了解石河子大学有关保留、使用学位论文的规定，学校有权保留学位论文并向国家主管部门或指定机构送交论文的电子版和纸质版。有权将学位论文在学校图书馆保存并允许被查阅。有权自行或许可他人将学位论文编入有关数据库提供检索服务。有权将学位论文的标题和摘要汇编出版。保密的学位论文在解密后适用本规定。

研究生签名：邓婧茹　　时间：2015 年 5 月 27 日

导师签名：[illegible]　　时间：2015 年 5 月 27 日

摘要

目的：食管鳞状细胞癌（ESCC）是全球范围内预后极差的恶性肿瘤，因早期症状隐匿且缺乏有效筛查手段，约 70%患者确诊时已进展至晚期，患者五年生存率不足 15%。当前食管癌的临床治疗取得一定进展，但仍存在靶向性弱、毒副作用大等问题，导致临床化疗药物的应用受限。去氧拉巴醌（Deoxylapachol，DEO）是新疆特色中草药毛菊苣中的蒽醌类化合物。目前关于 DEO 的药用价值报道较少，其对 ESCC 抗肿瘤药效作用及分子机制尚未见报道。前期通过网络药理学及体外分子实验筛选毛菊苣中抗食管癌单体，发现 DEO 能显著抑制食管癌细胞生长。本研究继续通过生信分析、分子对接等技术，从体内及体外层面阐明 DEO 抗食管鳞癌的药效作用及机制，为从新疆特色植物药毛菊苣中筛选抗食管癌作用的小分子化合物提供思路，也为新疆维药毛菊苣单体 DEO 的临床应用提供基础研究资料及理论依据。

方法：1. 体内动物层面探究 DEO 抗食管癌的药效作用。首先通过皮下注射人食管鳞癌细胞 KYSE-150 构建 BALB/c 裸鼠移植瘤模型。在成功构建移植瘤模型后，将裸鼠随机分为对照组（Control）、模型组（Model）、DEO-低剂量组（4mg/kg）、DEO-高剂量组（6mg/kg）和紫杉醇（PTX）阳性对照组（6mg/kg），连续腹腔注射给药 10 天，记录小鼠体重变化，观察荷瘤小鼠肿瘤大小、测量瘤体重量及体积，初步评估 DEO 对食管鳞癌的抑瘤作用。H&E 染色观察肿瘤组织、心脏、肝脏、肾脏组织形态。免疫组化检测 Ki67 表达；Western blot 检测肿瘤组织中增殖蛋白 PCNA、基质金属蛋白酶（MMP2、MMP9）蛋白表达。2. 体外细胞层面探究 DEO 对人食管鳞癌 KYSE-150 和 KYSE-410 细胞增殖、迁移、侵袭、凋亡、周期的影响。CCK-8 法检测细胞活力。平板克隆、EdU 掺入实验检测细胞增殖能力；划痕愈合及 Transwell 实验检测细胞迁移能力；TUNEL 染色检测细胞凋亡水平。荧光探针 JC-1 检测线粒体膜电位变化；Hoechst 33342 染色和 Annexin V-FITC/PI 法检测细胞凋亡形态及凋亡率。DCFH-DA 荧光探针检测活性氧含量；流式细胞术检测细胞周期分布情况；Western blot 检测增殖（PCNA）、迁移（MMP2，MMP9）、侵袭（β-catenin，Vimentin）、凋亡（Bax，Bcl-2，Cleaved Caspase-3）、周期相关蛋白（Cyclin A1，CDK2）表达。3. 生物信息学技术联合体内外实验阐明 DEO 治疗食管鳞癌作用机制：通过 GEO 数据集与转录组学筛选 DEO 抗食管癌的关键差异基因及信号通路，构建核心差异基因互作网络，分析差异基因 GO 富集分析及 KEGG 通路富集分析；实时荧光定量 PCR（Quantitative Real-time PCR）检测 DEO 对食管鳞癌细胞关键信号轴 FOXM1/MYBL2/CDCA3 mRNA 表达；分子对接评估 DEO 与信号轴 FOXM1、MYBL2、CDCA3 结合能；Western blot 实验检测 DEO 对食管鳞癌细胞信号轴 FOXM1/MYBL2/CDCA3 及核心通路 WNT/β-catenin 蛋白影响。体内层面，Western blot 检测 DEO 对荷瘤小鼠肿瘤组织 FOXM1/MYBL2/CDCA3 关键信号轴蛋白表达；最后利用慢病毒构建 shFOXM1-KYSE-150 及 shFOXM1-KYSE-410 细胞系，并通过 qRT-PCR 和 Western blot 验证 shFOXM1-KYSE-150 及 shFOXM1-KYSE-410 细胞中 FOXM1 基因及蛋白表达水

平。平板克隆实验和 Transwell 实验检测 DEO 对 shFOXM1-KYSE-150 及 shFOXM1-KYSE-410 增殖及迁移能力；Western blot 检测 DEO 对 shFOXM1-KYSE-150 及 shFOXM1-KYSE-410 增殖（PCNA）、迁移（MMP2，MMP9）、侵袭（β-catenin，Vimentin）、关键信号轴 FOXM1/MYBL2/CDCA3 及核心通路 WNT/β-catenin 蛋白影响。

结果：1. 体外动物层面证明 DEO 具有抗食管癌的药效作用。与模型组相比，DEO 治疗组的荷瘤小鼠肿瘤体积明显减小，瘤体重量明显减轻（$P<0.05$）。H&E 染色结果显示，与 Model 组比，DEO 给药组的肿瘤组织中固缩深染细胞增多，细胞形态更加不规则。与正常组相比，DEO 单纯给药组对裸鼠的心脏、肝脏、肾脏组织形态无明显变化。免疫组织化学染色结果显示，与模型组相比，DEO 显著降低增殖相关标志物 Ki67 蛋白表达。Western blot 结果显示，与模型组相比，DEO 明显抑制荷瘤小鼠肿瘤组织增殖（PCNA）及迁移蛋白（MMP2、MMP9）表达（$P<0.05$ 或 $P<0.01$）。以上结果说明 DEO 具有显著的抗食管癌药效作用。2. 体外层面实验证明 DEO 抑制食管鳞癌细胞增殖、迁移侵袭能力，诱导凋亡，并促进 S 期阻滞。CCK-8 结果显示，与对照组相比，DEO 呈浓度依赖性抑制食管鳞癌细胞活力（$P<0.05$ 或 $P<0.01$），KYSE-150、KYSE-410 的 IC_{50} 分别 29.32 μM、41.73 μM。平板克隆、EdU 及 Western blot 结果表明，与对照组相比，DEO 减少食管鳞癌细胞集落数及集落面积，抑制蛋白 PCNA 表达（$P<0.05$ 或 $P<0.01$）。根据 EdU 实验结果，DEO 显著减少 EdU 掺入比例（$P<0.05$ 或 $P<0.01$）。以上结果说明 DEO 对食管鳞癌细胞增殖有显著抑制作用。划痕愈合实验、Transwell、Western blot 结果表明，DEO 显著减少 KYSE-150 和 KYSE-410 细胞迁移面积，抑制迁移标志蛋白（MMP2、MMP9）及侵袭标志蛋白（β-catenin、Vimentin）表达（$P<0.05$ 或 $P<0.01$）。荧光探针 JC-1 染色实验结果显示，与对照组相比，随着 DEO 浓度增加，细胞内线粒体膜电位水平逐渐下降（$P<0.05$ 或 $P<0.01$）。荧光探针 DCFH-DA 实验结果显示，DEO 浓度依赖性增加 KYSE-150 和 KYSE-410 细胞活性氧含量（$P<0.05$ 或 $P<0.01$）。TUNEL 染色结果显示，与正常组相比，DEO 浓度依赖性增加 KYSE-150 和 KYSE-410 细胞荧光表达量（$P<0.05$ 或 $P<0.01$）。Annexin V-FITC/PI 结果显示，与正常组相比，DEO 显著增加 KYSE-150 和 KYSE-410 细胞凋亡数量（$P<0.05$ 或 $P<0.01$）。Western blot 结果表明 DEO 明显上调促凋亡 Bax 蛋白表达，下调抗凋亡 Bcl-2，Cleaved Caspase-3 蛋白表达（$P<0.05$ 或 $P<0.01$）。以上结果说明 DEO 能浓度依赖性促进 ROS 增加，诱导线粒体膜电位水平下降，促进食管鳞癌细胞凋亡。流式细胞术及 Western blot 结果显示，与正常组相比，DEO 诱导 KYSE-150 和 KYSE-410 细胞 S 期占比增多，显著抑制 CyclinA1、CDK2 蛋白表达，说明 DEO 能诱导食管鳞癌细胞阻滞在 S 期（$P<0.05$ 或 $P<0.01$）。3. 生信分析联合体内外实验阐明 DEO 能通过调控 FOXM1/MYBL2/CDCA3 信号轴抑制 WNT/β-catenin 信号通路，发挥抗食管鳞癌作用。GEO 数据集结合转录组生信分析，筛选 DEO 抗食管癌的关键差异基因 18945 个，其中上调 2765 个基因，下调 1662 个基因。根据基因表达差异倍数及基因互作网络 degree 值，确定 FOXM1 为抗食管癌的关键基因，FOXM1/MYBL2/CDCA 为关键信号轴。分子对接结果显示，DEO 与 FOXM1、MYBL2、CDCA3 结合能良好。qRT-PCR 及 Western blot 结果显示，DEO 显著抑制食管鳞癌细胞基因 mRNA 及蛋白表达。免疫组织化学结果显示，与模型组相比，DEO 明显抑制荷瘤小鼠肿瘤组织

FOXM1/MYBL2/CDCA3 信号轴核心蛋白表达（$P < 0.05$）。Western blot 验证 DEO 可以显著下调 WNT/β-catenin 通路相关蛋白表达（$P < 0.05$ 或 $P < 0.01$）。慢病毒构建 shFOXM1-KYSE-150 及 shFOXM1-KYSE-410 细胞系，qRT-PCR 和 Western blot 结果，DEO 显著抑制 shFOXM1-KYSE-150 及 shFOXM1-KYSE-410 细胞内 FOXM1 基因及蛋白表达（$P<0.01$）。平板克隆实验和 Transwell 实验检测结果显示，DEO 抑制 shFOXM1-KYSE-150 及 shFOXM1-KYSE-410 细胞增殖能力，减少迁移面积（$P < 0.05$）。Western blot 检测结果发现，DEO 可以抑制 shFOXM1-KYSE-150 及 shFOXM1-KYSE-410 内增殖（PCNA）、迁移（MMP2，MMP9）、侵袭（β-catenin，Vimentin）、信号轴 FOXM1/MYBL2/CDCA3 及信号通路 WNT/β-catenin 关键蛋白的表达（$P < 0.05$ 或 $P < 0.01$）。

结论：DEO 能通过调控 FOXM1/MYBL2/CDCA3 信号轴，下调 WNT/β-catenin 信号通路，抑制食管鳞癌增殖、迁移、侵袭能力，降低线粒体膜电位并增加细胞内 ROS 含量，诱导凋亡，阻滞食管癌细胞周期至 S 期，进而发挥抗食管癌的作用。

关键词：食管鳞癌；去氧拉巴醌；转录组学；FOXM1/MYBL2/CDCA3 轴；WNT/β-catenin 信号通路

Abstract

Objective: Esophageal squamous cell carcinoma (ESCC) is a malignancy cancer. Due to the insidious early symptoms and lack of effective screening tools, approximately 70% of patients have advanced disease by the time they are diagnosed, and the five-year survival rate is less than 15%. Currently, problems such as poor targeting and high toxicity and side effects still limit the use of clinical chemotherapeutic drugs. Deoxylapachol (DEO) is an anthraquinone compound extracted from Xinjiang's speciality herb, hairy chicory. There are few reports on the medicinal value of DEO, and its antitumour pharmacodynamic effects and molecular mechanisms in ESCC have not been reported. Previous screening of anti-esophageal cancer monomers in hairy chicory by network pharmacology and in vitro molecular experiments showed that DEO significantly inhibited the growth of esophageal cancer cells. In this study, we further elucidated the pharmacodynamic effects and mechanisms of DEO against esophageal squamous cell carcinoma in vivo and in vitro using bioconfidence analysis, molecular docking and other techniques to provide ideas for the screening of small molecule compounds with anti-esophageal carcinoma effects in Xinjiang's characteristic herbal medicine, *Cichorium glandulosum*, and to provide basic research information and theoretical basis for the clinical application of the Xinjiang Uyghur *Cichorium glandulosum* monomer, DEO.

Methods: 1. Exploring the pharmacological effects of DEO on esophageal cancer at the animal level in vivo. F he transplantation tumour model of BALC/c nude mice was first constructed by subcutaneous injection of human oesophageal squamous carcinoma cells KYSE-150. After successfully constructing a transplant tumor model, nude mice were randomly divided into a Control group (Control), a model group (Model), a low dose group (4mg/kg), a high dose group (6mg/kg), and a paclitaxel (PTX) positive Control group (6mg/kg). The mice were continuously injected intraperitoneally for 10 days, and their weight changes were recorded. The tumor size, weight, and volume of the tumor bearing mice were observed, and the anti-tumor effect of DEO on ESCC was preliminarily evaluated. Observe the morphology of tumor tissue, heart, liver, and kidney tissue using H&E staining. Ki67 expression was detected by immunohistochemistry. Western blot was used to detect the protein expression of proliferating protein PCNA and matrix metalloproteinases (MMP2, MMP9) in tumor tissues. 2. Exploring the effects of DEO on the proliferation, migration, invasion, apoptosis, and cell cycle of human esophageal squamous cell carcinoma KYSE-150 and KYSE-410 cells at the cellular level in vitro. CCK-8 method is used to detect cell viability. Plate cloning and EdU incorporation experiments were used to detect cell proliferation ability. Scratch healing and Transwell assay to detect cell migration ability. TUNEL staining was used to detect the level of cell apoptosis. Annexin V-FITC/PI method were used to detect the morphology and apoptosis rate of cells. JC-1 fluorescent probe detects mitochondrial membrane potential changes. Flow cytometry is used to detect the distribution of cell

cycle. DCFH-DA fluorescent probe detects reactive oxygen species content. Western blot was used to detect the expression of proliferation (PCNA), migration (MMP2, MMP9), invasion (β-catenin, Vimentin), apoptosis (Bax, Bcl-2, Cleaved Caspase-3), and cycle related proteins (Cyclin A1, CDK2). 3. Bioinformatics technology combined with in vitro and in vivo experiments elucidated the mechanism of action of DEO in the treatment of esophageal squamous cell carcinoma: key differentially expressed genes and signaling pathways of DEO in anti-esophageal cancer were screened using GEO dataset and transcriptomics, and a core differentially expressed gene interaction network was constructed. Real time quantitative PCR was used to detect the expression of FOXM1/MYBL2/CDCA3 mRNA, a key signaling axis, in esophageal squamous cell carcinoma cells by DEO; Molecular docking evaluation of the binding energy between DEO and signal axes FOXM1, MYBL2, CDCA3. Western blot experiment was used to detect the effect of DEO on the signal axis FOXM1/MYBL2/CDCA3 and core pathway WNT/β-catenin protein in esophageal squamous cell carcinoma cells. At the in vivo level, Western blot was used to detect the expression of key signaling axis proteins FOXM1/MYBL2/CDCA3 in tumor tissues of tumor bearing mice by DEO. Finally, shFOXM1-KYSE-150 and shFOXM1-KYSE-410 cell lines were constructed using lentivirus, and the expression of FOXM1 gene and protein in shFOXM1-KYSE-150 and shFOXM1-KYSE-410 cells was validated by qRT PCR and Western blot. Plate cloning experiment and Transwell experiment were used to detect the proliferation and migration ability of DEO on shFOXM1-KYSE-150 and shFOXM1-KYSE-410. Western blot was used to detect the effects of DEO on the proliferation (PCNA), migration (MMP2, MMP9), invasion (β-catenin, Vimentin), key signaling axis FOXM1/MYBL2/CDCA3, and core pathway WNT/β-catenin proteins of shFOXM1-KYSE-150 and shFOXM1-KYSE-410.

Results: 1. In vitro animal studies have shown that DEO has pharmacological effects against esophageal cancer. Compared with the model group, the tumor volume and weight of the tumor bearing mice in the DEO treatment group were reduced by DEO ($P < 0.05$). The H&E staining showed that compared with the model group, the number of cells in the tumor tissue of the DEO treated group decreased, the number of cells with deep staining increased, and the cell morphology became more irregular. Compared with the normal group, DEO and PTX were no significant changes in the morphology of the heart, liver, and kidney tissues in mice treated solely with DEO. The immunohistochemical staining results showed that compared with the model group, DEO significantly reduced the expression of Ki67. Western blot results showed that compared with the model group, DEO significantly inhibited tumor tissue proliferation (PCNA) and migration protein (MMP2, MMP9) expression in the tumor bearing group ($P <0.05$ or $P <0.01$). The above results indicate that DEO has significant anti esophageal cancer pharmacological effects. 2. In vitro experiments have shown that DEO inhibits the proliferation, migration, and invasion of ESCC cells, induces apoptosis, and promotes S phase arrest. The CCK-8 results showed that compared with the Control group, DEO exhibited concentration dependent inhibition of esophageal squamous cell carcinoma cell viability ($P < 0.05$ or $P < 0.01$), with IC50

values of 29.32 μM and 41.73 μM for KYSE-150 and KYSE-410, respectively. The results of plate cloning, EdU, and Western blot showed that compared with the Control group, DEO reduced the number of ESCC cell colonies, and inhibited the expression of protein PCNA ($P < 0.05$ or $P < 0.01$). According to the EdU experiment results, compared with the Control group, DEO significantly reduced the proportion of EdU incorporation ($P < 0.05$ or $P < 0.01$). The above results indicate that DEO has a significant inhibitory effect on the proliferation of esophageal squamous cell carcinoma cells. The scratch healing experiment, Transwell, and Western blot results showed that compared with the Control group, DEO significantly reduced the migration area of KYSE-150 and KYSE-410 cells, inhibited the expression of migration marker proteins (MMP2, MMP9) and invasion marker proteins (β-catenin, Vimentin) ($P < 0.05$ or $P < 0.01$). The fluorescence probe JC-1 staining results showed that, the intracellular mitochondrial membrane potential level gradually decreased with the increase of DEO compared with the Control group ($P < 0.05$). The experimental results of fluorescence probe DCFH-DA showed that the concentration of DEO increased the reactive oxygen species content in KYSE-150 and KYSE-410 cells ($P < 0.05$). The results of TUNEL staining and Hoechst 33342 staining showed that compared with the normal group, the concentration dependent increase in fluorescence expression of KYSE-150 and KYSE-410 cells was observed in the DEO group ($P < 0.05$ or $P < 0.01$). The Annexin V-FITC/PI results showed that compared with the normal group, DEO significantly increased the number of apoptotic KYSE-150 and KYSE-410 cells ($P < 0.05$ or $P < 0.01$). Western blot results showed that DEO significantly upregulated the expression of pro apoptotic Bax protein and downregulated the expression of antiapoptotic Bcl-2 and Cleaved Caspase-3 proteins ($P < 0.05$ or $P < 0.01$). The above results indicate that DEO can promote ROS increase in a concentration dependent manner, induce a decrease in mitochondrial membrane potential levels, and promote apoptosis of esophageal squamous cell carcinoma cells. Flow cytometry and Western blot results showed that compared with the normal group, DEO induced a significant increase in the proportion of KYSE-150 and KYSE-410 cells in the S phase, and significantly inhibited the expression of CyclinA1 and CDK2 proteins, indicating that DEO can induce esophageal squamous cell carcinoma cells to be blocked in the S phase ($P < 0.05$ or $P < 0.01$). 3. Bioinformatics analysis combined with in vitro and in vivo experiments demonstrated that DEO can inhibit the WNT/β-catenin signaling pathway by targeting the FOXM1/MYBL2/CDCA3 signaling axis, exerting anti esophageal squamous cell carcinoma effects. GEO dataset combined with transcriptome bioinformatics analysis, screened 18945 key-differentially expressed genes for DEO anti esophageal cancer, including 2765 upregulated genes and 1662 downregulated genes. Based on the differential fold of gene expression and the degree of gene interaction network, FOXM1 was identified as a key gene for anti-esophageal cancer, with FOXM1/MYBL2/CDCA as the key signaling axis. The molecular docking results showed that DEO can bind well to FOXM1, MYBL2, and CDCA3. QRT-PCR and Western blot results showed that compared with the Control group, DEO significantly inhibited gene mRNA and protein expression in esophageal squamous cell

carcinoma cells. The immunohistochemical results showed that compared with the model group, DEO significantly inhibited the expression of FOXM1/MYBL2/CDCA3 signaling axis proteins in tumor tissues of the tumor bearing group ($P < 0.05$). Western blot analysis showed that DEO downregulated four proteins expression in WNT/β-catenin pathway ($P < 0.05$ or $P < 0.01$). Lentiviral vectors were used to construct shFOXM1-KYSE-150 and shFOXM1-KYSE-410 cell lines. qRT-PCR and Western blot analyses revealed that DEO markedly inhibited FOXM1 gene and protein expression in both cell lines ($P < 0.05$ or $P < 0.01$). Plate clone and Transwell assays demonstrated that DEO suppressed the proliferation of these cells and reduced their migration ($P < 0.05$). Western blot analysis also indicated that DEO could suppress the expression of proteins related to proliferation (PCNA), migration (MMP2, MMP9), invasion (β-catenin, Vimentin), the FOXM1/MYBL2/CDCA3 signalling axis and key proteins in the WNT/β-catenin pathway in these cells ($P < 0.05$ or $P < 0.01$).

Conclusion: DEO can suppress esophageal squamous cell carcinoma proliferation, migration, and invasion by modulating the FOXM1/MYBL2/CDCA3 axis and downregulating the WNT/β-catenin pathway. It also reduces mitochondrial membrane potential, increases intracellular ROS, induces apoptosis, and arrests the cell cycle in the S phase, thus exerting anti-esophageal cancer effects.

Key words: Esophageal squamous cell carcinoma; Deoxylaboquinone; Transcriptomics; FOXM1/MYBL2/CDCA3 axis; WNT/β-catenin signaling pathway

目录

中英文缩略词

（LIST OF ABBREVIATIONS）

英文缩写	英文全名	中文译名
Acr	Acrylamide	丙烯酰胺
AP	Ammonium persulfate	过硫酸铵
Bax	BCL 2-associated	BCL 2-Associated X 的蛋白质
BCA	bicinchoninicacid	二喹啉甲酸
Bcl-2	B-cell lymphoma	B 淋巴细胞瘤-2 基因
BP	Biological process	生物学过程
BSA	Albumin from bovine serum	牛血清白蛋白
CCK-8	Cell counting kit-8	细胞计数试剂盒
CDCA3	Cell Division Cycle Associated 3	细胞分裂周期相关 3
CDK4	Cyclin=dependent kinase 4	周期蛋白依赖性激酶 4
ddH2O	Distillation-Distillation H2o	双蒸水
DEO	Deoxylapachol	去氧拉巴醌
DMSO	Dimethyl sulfoxide	二甲基亚砜
EdU	5-ethynyl-2’-deoxyuridine	5-溴-2-脱氧尿嘧啶
EP	Eppendorf micro test tube	微量离心管
ESCC	Esophageal squamous cell carcinoma	食管鳞癌
FBS	Fatal bovine serun	胎牛血清
FDR	False bovine serum	错误发现率