LINC00680通过靶向miR-195-5p调控IL-17诱导的肺癌细胞增殖、迁移和侵袭

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摘要 该文主要讨论LINC00680靶向调控miR-195-5p对IL-17诱导的肺癌细胞增殖、迁移和侵袭的影响。将肺癌细胞H1299分为Control组、IL-17组、IL-17+si-NC组、IL-17+si-LINC00680组、IL-17+si-LINC00680+anti-miR-NC组、IL-17+si-LINC00680+anti-miR-195-5p组。采用qRT-PCR检测LINC00680和miR-195-5p的表达; 克隆形成实验、MTT检测细胞增殖情况; Transwell实验检测细胞迁移和侵袭能力; Western blot检测Ki67、E-cadherin、N-cadherin蛋白表达水平; 荧光素报告实验验证LINC00680和miR-195-5p靶向关系。与Control组比较, IL-17组LINC00680相对表达量、克隆细胞数、细胞活力、迁移细胞数、侵袭细胞数、Ki67和N-cadherin蛋白合成产物明显增加, E-cadherin蛋白、miR-195-5p相对表达量明显减少。与IL-17+si-NC组比较, IL-17+si-LINC00680组LINC00680相对表达量、克隆细胞数、细胞活力、迁移细胞数、侵袭细胞数、Ki67和N-cadherin蛋白表达量明显减少, E-cadherin蛋白、miR-195-5p相对表达量明显增多。LINC00680可以靶向调控miR-195-5p表达, 抑制miR-195-5p可以逆转下调LINC00680对IL-17诱导的肺癌细胞增殖、迁移和侵袭的作用。LINC00680通过调控miR-195-5p促进IL-17诱导的肺癌细胞增殖、迁移和侵袭。

关键词 LINC00680; miR-195-5p; 肺癌细胞; 增殖; 迁移; 侵袭

LINC00680 Regulates IL-17-Induced Proliferation, Migration and Invasion of Lung Cancer Cells by Targeting miR-195-5p

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Abstract This article mainly discusses the effects of LINC00680 targeted regulation of miR-195-5p on the IL-17-induced proliferation, migration and invasion of lung cancer cells. In this study, lung cancer cells H1299 were divided into Control group, IL-17 group, IL-17+si-NC group, IL-17+si-LINC00680 group, IL-17+si-LINC00680+anti-miR-NC group, IL-17+si-LINC00680+anti-miR-195-5p group. Expression levels of LINC00680 and miR-195-5p were determined by qRT-PCR; cell proliferation was determined by clone formation experiments and MTT; cell migration and invasion abilities were measured by Transwell assay; Ki67, E-cadherin and N-cadherin protein expression levels were determined by Western blot; and the targeting relationship between LINC00680 and

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miR-195-5p was verified by dual-luciferase reporter system. Compared with the Control group, the relative expression of LINC00680, the number of clones, the cell viability, the number of migrating cells, the number of invasive cells, the expression of Ki67 and N-cadherin protein in the IL-17 group were significantly increased, and the expression of E-cadherin protein and the relative expression of miR-195-5p were significantly decreased. Compared with the IL-17+si-NC group, the relative expression of LINC00680, the number of cloned cells, cell viability, the number of migrating cells, the number of invasive cells, and the expression of Ki67 and N-cadherin protein in the IL-17+si-LINC00680 group were significantly decreased, but the expression of E-cadherin protein and the relative expression of miR-195-5p increased significantly. LINC00680 can targetedly regulate miR-195-5p expression, and the inhibition of miR-195-5p can reverse the effect of down-regulation of LINC00680 on IL-17-induced proliferation, migration and invasion of lung cancer cells. LINC00680 promotes the IL-17-induced lung cancer cells proliferation, migration and invasion through the regulation of miR-195-5p.

Keywords LINC00680; miR-195-5p; lung cancer cells; proliferation; migration; invasion

肺癌作为常见的恶性肿瘤之一, 发病率、死亡率居高不下, 早期时症状不明显, 导致许多患者确诊时已错过了最佳治疗时机[1]。在肺癌患者中局部淋巴结、肺部、骨髓等是常见的转移部位, 这也是肺癌患者死亡率较高的主要原因[2-3]。近年来, 靶向治疗在肺癌的治疗中已经取得了明显的进展, 因此, 寻找新的靶点对治疗肺癌具有重要的意义[4]。长链非编码RNA(lncRNA)与肺癌发生、发展密切相关[5], 如LINC00680在非小细胞癌患者组织中表达上调, 与恶性程度及预后不良有关, LINC00680通过调控miR-410-3p/HMGB1轴促进非小细胞肺癌细胞增殖和抑制其凋亡[6], 但是对肺癌细胞迁移、侵袭的作用尚不清楚。IL-17是多功能细胞分泌因子, 在肿瘤细胞中可促进细胞恶性转移, 是肿瘤微环境的组成部分[7]。miR-195-5p在肺癌、乳腺癌等癌细胞中表达下调[8-9], IL-17可降低子宫内膜癌细胞HEC-1-B中的miR-195-5p的表达水平, 过表达miR-195-5p可抑制IL-17诱导的人子宫内膜癌细胞HEC-1-B增殖、迁移和侵袭[10]。在线生物信息学软件预测显示, LINC00680与miR-195-5p之间存在结合位点, 鉴于此, 本研究以肺癌细胞H1299为研究对象, 主要研究LINC00680靶向调控miR-195-5p对IL-17诱导的肺癌细胞增殖、迁移及侵袭的影响。

1 一般材料与方法

1.1 细胞及主要试剂

肺癌细胞H1299购于美国ATCC; 胎牛血清、DMEM培养基购于美国Hyclone公司; IL-17、MTT试剂盒购于上海碧云天生物技术有限公司; si-NC、si-LINC00680、anti-miR-NC、anti-miR-195-5p、引物(LINC00680、miR-195-5p、U6、GAPDH)由上海吉玛公司进行设计并完全合成; Lipofectamine 2000试剂盒、Trizol试剂盒购于深圳子科生物科技有限公司; 逆转录试剂盒、SYBR Premix Ex Taq试剂盒购于北京天根生化科技有限公司; Transwell、基质胶购于美国Corning公司; Ki67抗体、E-cadherin抗体、N-cadherin抗体、GAPDH抗体购于美国Abcam公司; 双荧光素酶检测试剂盒购于北京全式金生物技术有限公司。

1.2 细胞培养和分组

肺癌细胞H1299在37 °C水浴锅内复苏, 复苏完成后转移至提前配制好的DMEM培养基(包含10%胎牛血清和青霉素–链霉素)内, 并将培养基放于37 °C、5% CO_2培养箱中进行培育, 每2天换1次细胞液, 当细胞生长融合率达到85%以上时, 用胰酶消化传代培养细胞。

取对数期肺癌细胞H1299, 用100 μg/L的IL-17处理细胞记为IL-17组, 正常培养细胞作为Control组; 将si-NC、si-LINC00680、si-LINC00680和anti-miR-NC、si-LINC00680和anti-miR-195-5p转染至细胞内, 用100 μg/L的IL-17处理细胞, 记为IL-17+si-NC组、IL-17+si-LINC00680组、IL-17+si-LINC00680+anti-miR-NC组、IL-17+si-LINC00680+anti-miR-195-5p组。细胞转染按照Lipofectamine 2000试剂盒说明书进行。

1.3 qRT-PCR检测LINC00680和miR-195-5p的表达情况

收集上述1.2方法培养48 h的各组肺癌细胞

H1299根据Trizol法提取细胞总RNA, 置于分光光度计内检测RNA浓度和纯度, 逆转录合成cDNA, 根据SYBR Premix Ex Taq试剂盒说明书进行PCR反应。LINC00680以*GAPDH*作为内参基因, miR-195-5p以*U6*作为内参基因, 用$2^{-\Delta\Delta Ct}$公式计算LINC00680和miR-195-5p的表达量。LINC00680 Forward: 5′-CGA TGT TTA AGC GTC CGG G-3′, Reverse: 5′-GGA AAG ATG ATG CGC TGT GT-3′; *GAPDH* Forward: 5′-TGGT TGA GCA CAG GGT ACT T-3′, Reverse: 5′-CCA AGG AGT AAG ACC CCT GG-3′。miR-195-5p Forward: 5′-GAA TTC GCC TCA AGA GAA CAA AGT GGA G-3′, Reverse: 5′-AGA TCT CCC ATG GGG GCT CAG CCC CT-3′; *U6* Forward: 5′-GCT TCG GCA GCA CAT ATA CTA AAA T-3′, Reverse:5′-CGC TTC ACG AAT TTG CGT GTC AT-3′。

1.4 克隆形成实验、MTT检测细胞增殖

克隆形成实验: 按照1.2方法处理肺癌细胞H1299, 制成1×10^3个/mL细胞悬液, 并接种至6孔板中, 将细胞放置培养箱内14天, 肉眼可见细胞出现克隆时停止培养, 加入多聚甲醛室温固定30 min, 结晶紫染色20 min, 倒去培养液清洗细胞, 晾干后将其置于倒置显微镜下拍照, 观察细胞克隆形成数。

MTT: 按照1.2方法处理肺癌细胞H1299并接种至96孔中, 然后培养48 h, 每孔内加入20 μL的MTT试剂, 继续培养4 h后弃培养液, 每孔内加入150 μL的DMSO试剂, 用酶标仪检测波长为490 nm处的细胞吸光度(*D*)值并记录, 通过公式转换为细胞活力值。细胞活力(%)=(实验组*D*值–空白组*D*值)/(对照组*D*值–空白组*D*值)×100%。

1.5 Transwell实验评估细胞转移能力及侵袭能力

检测研究细胞迁移的实验方法: 将收集的1.2方法培养的肺癌细胞H1299用不含血清的完全培养基重悬细胞, 密度为5×10^4个/mL; Transwell小室内放入100 μL无血清培养基, 在培养板内放入500 μL具有血清并适合细胞生长繁殖的培养基, 培养24 h擦去没有迁移的细胞, 使用多聚甲醛室温固定30 min后, 再使用结晶紫对研究细胞进行染色, 上色时间为20 min; 倒去培养液后用显微镜进行观察, 选取视野亮度较好的部分记录细胞转移数量。检测研究细胞发生侵袭的实验方法: 将基质胶用不含血清的完全培养基稀释, 取80 μL均匀铺在Transwell上室内, 风干, 后续实验与上述迁移实验方法相同。

1.6 Western blot检测Ki67、E-cadherin、N-cadherin蛋白表达水平

选取每组培养48 h的细胞, 并用缓冲液对研究细胞进行冲洗, 冲洗完成后向各组培养基中加入RIPA裂解细胞, 同时遵循BCA法对各组蛋白进行检 测定量, 经过12%的SDS-PAGE凝胶处理上样蛋白(50 μg), 浓缩胶(80 V反应40 min)、分离胶(120 V反应1.5 h), 电泳结束后转膜, 脱脂奶粉室温封闭培养2 h, 加入Ki67(1:800)、E-cadherin(1:600)、N-cadherin(1:800)、GAPDH(1:1 000)抗体4 °C过夜孵育, 加入带标记的二抗(1:2 000) 37 °C孵育2 h, 避光加入发光试剂, 显影、拍照。以GAPDH为内参, 采用ImageJ软件扫描分析蛋白密度值。

1.7 双荧光素酶实验检验LINC00680和miR-195-5p靶向关系

通过LncBase预测显示LINC00680和miR-195-5p之间存在结合位点, 将LINC00680基因片段克隆至pmirGLO载体上, 并进行定点突变, 构建LINC00680 WT、LINC00680 MUT荧光素酶质粒, 再分别与miR-NC或miR-195-5p共转染至肺癌细胞H1299中, 培养48 h, 严格按照使用说明书进行, 并利用双荧光素酶试剂盒检验荧光素酶存活情况。

1.8 统计学分析

使用SPSS 20.0软件对实验数据进行处理及统计分析, 数据结果以均数±标准差($\bar{x}\pm s$)显示, 多组间比较应用单因素方差进行数据处理和分析, 两组间比较使用*t*检验。以$P<0.05$为差异具有显著统计学意义。

2 结果

2.1 IL-17诱导肺癌细胞内LINC00680表达情况

与Control组相比, IL-17组LINC00680相对表达量显著增加($P<0.05$)(表1); 与IL-17+si-NC组相比, IL-17+si-LINC00680组LINC00680相对表达量显著减少($P<0.05$)(表1)。

2.2 下调LINC00680对IL-17诱导肺癌细胞增殖的影响

与Control组相比, IL-17组克隆细胞数、细胞活力、Ki67蛋白表达水平显著升高($P<0.05$)(图1和表2); 与IL-17+si-NC组相比, IL-17+si-LINC00680组克隆细胞数、细胞活力、Ki67蛋白表达水平显著降低($P<0.05$)(图1和表2)。

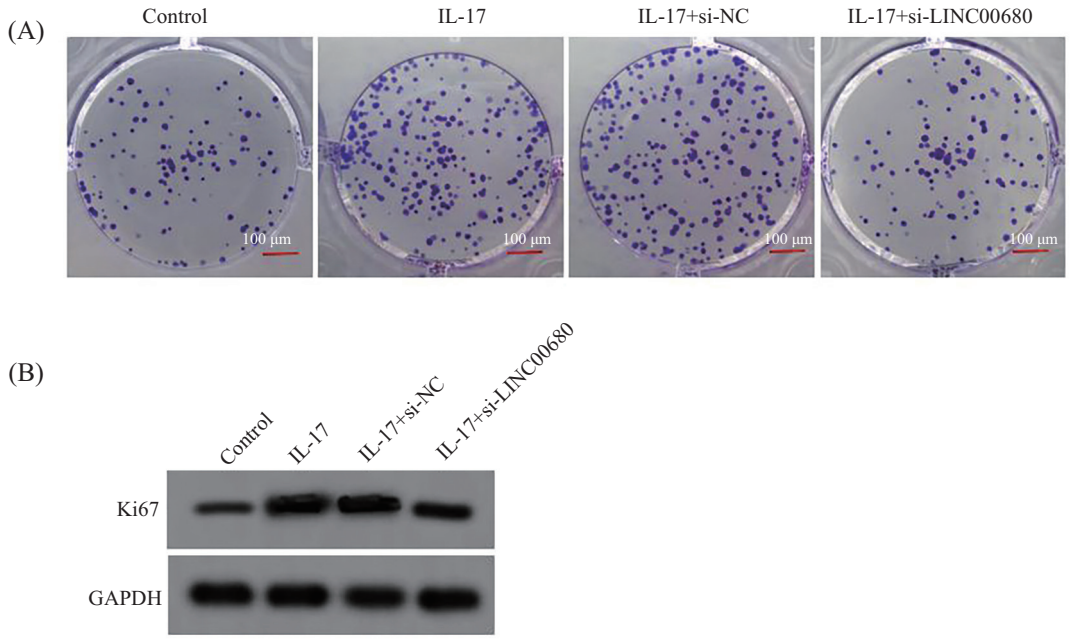
表1 IL-17诱导肺癌细胞后LINC00680相对表达量

Table 1 The relative expression of LINC00680 of lung cancer cells induced by IL-17

分组 Group	LINC00680
Control	0.99±0.08
IL-17	3.24±0.26*
IL-17+si-NC	3.20±0.31
IL-17+si-LINC00680	1.85±0.14#
F	228.399
P	0.000

*P<0.05, 与Control组相比; #P<0.05, 与IL-17+si-NC组相比。

*P<0.05 compared with Control group; #P<0.05 compared with IL-17+si-NC group.



A: 克隆形成实验检测细胞增殖; B: Western blot检测Ki67蛋白表达。

A: cell proliferation was determined by clone formation assay; B: the Ki67 protein expression level was determined by Western blot.

图1 下调LINC00680对IL-17诱导肺癌细胞增殖的影响

Fig.1 The effect of down-regulation of LINC00680 on the proliferation of lung cancer cells induced by IL-17

表2 下调LINC00680对IL-17诱导肺癌细胞增殖的影响

Table 2 The effect of down-regulation of LINC00680 on the proliferation of lung cancer cells induced by IL-17

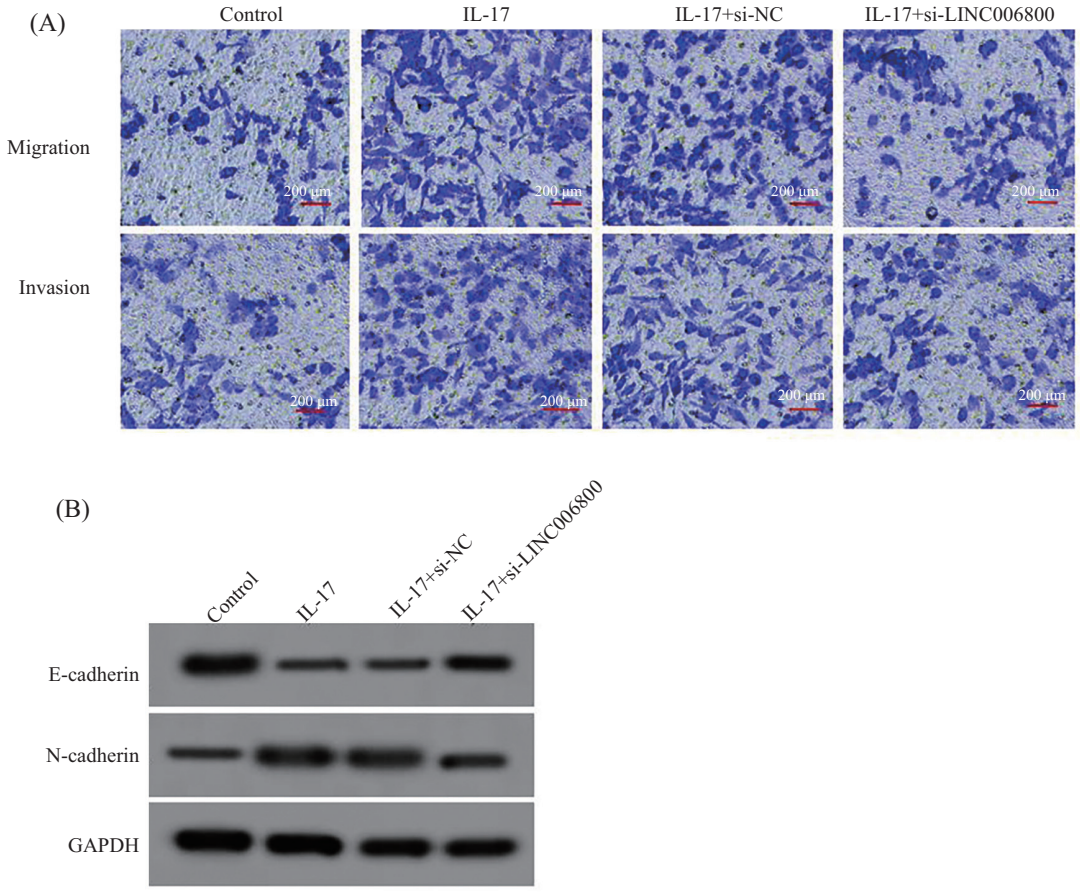
分组 Group	克隆细胞数 Number of cloned cells	细胞活力/% Cell viability /%	Ki67
Control	62.47±5.48	99.73±8.72	0.32±0.04
IL-17	126.98±10.36*	168.12±13.21*	0.82±0.07*
IL-17+si-NC	122.46±11.24	172.33±12.65	0.79±0.07
IL-17+si-LINC00680	85.14±6.35#	132.52±9.85#	0.53±0.04#
F	112.868	82.112	154.062
P	0.000	0.000	0.001

*P<0.05, 与Control组相比; #P<0.05, 与IL-17+si-NC组相比。

*P<0.05 compared with Control group; #P<0.05 compared with IL-17+si-NC group.

2.3 下调LINC00680对IL-17诱导肺癌细胞迁移和侵袭的影响

与Control组相比, IL-17组在迁移细胞数、侵袭细胞数、N-cadherin蛋白表达水平等方面明显增加(P<0.05), 而IL-17组E-cadherin蛋白表达水平对比Control组则显著降低(P<0.05) (图2和表3); 与IL-17+si-NC组相比, IL-17+si-LINC00680组迁移细胞数、侵袭细胞数及N-cadherin蛋白表达水平降低(P<0.05), E-cadherin蛋白表达水平明显升高(P<0.05)(图2和表3)。



A: Transwell实验检测细胞迁移、侵袭; B: Western blot检测E-cadherin、N-cadherin蛋白表达水平。

A: cell migration and invasion were detected by the Transwell assay; B: protein expression levels of E-cadherin and N-cadherin were determined by Western blot.

图2 下调LINC00680对IL-17诱导肺癌细胞迁移和侵袭的影响

Fig.2 The effect of down-regulation of LINC00680 on the migration and invasion of lung cancer cells induced by IL-17

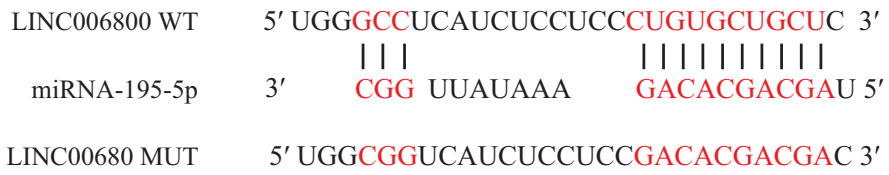
表3 下调LINC00680对IL-17诱导肺癌细胞迁移和侵袭的影响

Table 3 The effect of down-regulation of LINC00680 on the migration and invasion of lung cancer cells induced by IL-17

分组 Group	迁移细胞数 Number of migrating cells	侵袭细胞数 Number of invasion cells	E-cadherin	N-cadherin
Control	46.85±5.21	38.94±3.36	0.63±0.05	0.20±0.01
IL-17	92.36±7.62*	80.26±6.98*	0.31±0.04*	0.53±0.04*
IL-17+si-NC	89.83±8.05	82.47±7.34	0.29±0.03	0.55±0.05
IL-17+si-LINC00680	61.60±6.00#	54.33±4.52#	0.43±0.05#	0.36±0.04#
F	94.922	118.560	116.960	166.759
P	0.000	0.000	0.000	0.000

*P<0.05, 与Control组相比; #P<0.05, 与IL-17+si-NC组相比。

*P<0.05 compared with Control group; #P<0.05 compared with IL-17+si-NC group.



红色碱基代表结合位点。

Red bases represent the binding sites.

图3 LINC00680和miR-195-5p之间存在互补的核苷酸序列

Fig.3 Complementary nucleotide sequences were present in both LINC00680 and miR-195-5p

表4 双荧光素酶报告实验

Table 4 Double luciferase report experiment

分组 Group	LINC00680 WT	LINC00680 MUT
miR-NC	1.00±0.10	0.99±0.07
miR-195-5p	0.38±0.04*	1.02±0.11
t	17.720	0.690
P	0.000	0.500

*P<0.05, 与miR-NC组相比。

*P<0.05 compared with miR-NC group.

表5 LINC00680靶向负调控miR-195-5p的表达

Table 5 LINC00680 negatively regulates the expression of miR-195-5p

分组 Group	miR-195-5p
Control	1.00±0.08
IL-17	0.32±0.03*
IL-17+si-NC	0.33±0.04
IL-17+si-LINC00680	0.76±0.06$^{\#}$
F	323.400
P	0.000

*P<0.05, 与Control组相比; $^{\#}P$<0.05, 与IL-17+si-NC组相比。

*P<0.05 compared with Control group; $^{\#}P$<0.05 compared with IL-17+si-NC group.

2.4 LINC00680靶向miR-195-5p的表达

通过LncBase预测显示LINC00680和miR-195-5p之间存在互补的结合位点(图3)。与miR-NC组比较, miR-195-5p可明显降低LINC00680 WT荧光素酶活性, 而对LINC00680 MUT荧光素酶活性没有显著影响(表4)。

2.5 LINC00680靶向负调控miR-195-5p的表达

与Control组相比, IL-17组miR-195-5p相对表达水平显著降低(P<0.05); 与IL-17+si-NC组相比, IL-17+si-LINC00680组miR-195-5p相对表达水平显著升高(P<0.05, 表5)。与anti-miR-NC组相比, anti-miR-195-5p组中miR-195-5p相对表达水平显著降低(P<0.05, 表6)。

2.6 抑制miR-195-5p可以部分回复LINC00680下调对IL-17诱导的肺癌细胞增殖、迁移和侵袭的影响

与IL-17+si-LINC00680+anti-miR-NC组相比, IL-17+si-LINC00680+anti-miR-195-5p组的miR-195-5p相对表达水平下降效果更加明显, IL-17+si-LINC00680+anti-miR-195-5p组克隆细胞数、细胞活性、迁移细胞数、侵袭细胞数、Ki-67和N-cadherin蛋白表达水平则明显上升(P<0.05), E-cadherin蛋白表达明显下降(P<0.05)(图4和表7)。

3 讨论

IL-17家族成员因子由6个成员组成, 主要包

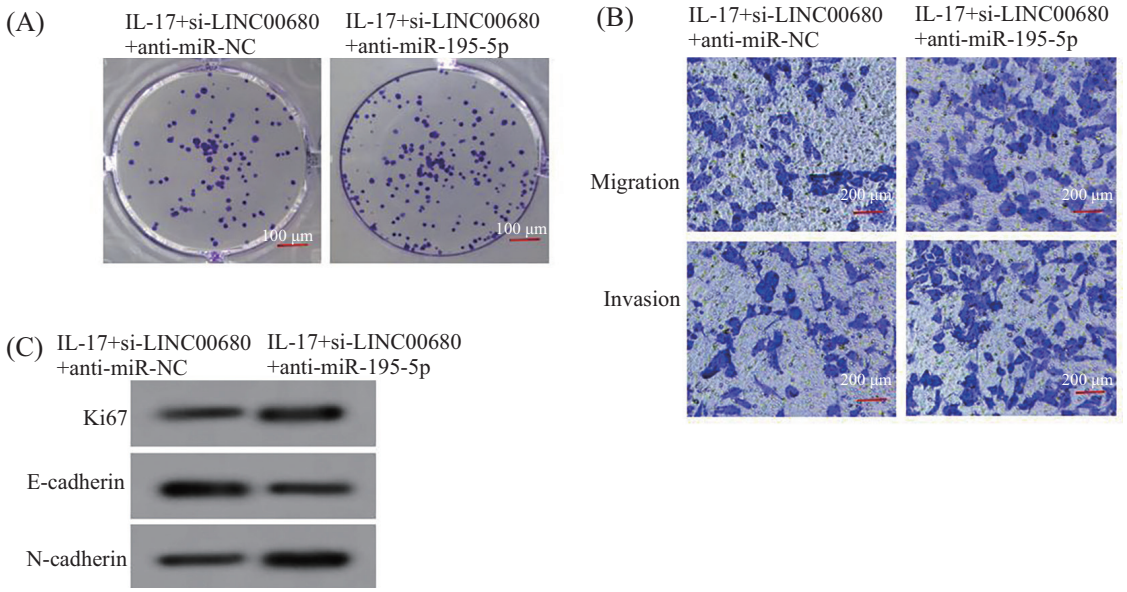
表6 转染miR-195-5p后对肺癌细胞miR-195-5p表达的影响

Table 6 Effect of miR-195-5p transfection on the expression of miR-195-5p in lung cancer cells

分组 Group	miR-195-5p
anti-miR-NC	1.01±0.07
anti-miR-195-5p	0.40±0.05*
t	21.273
P	0.000

*P<0.05, 与anti-miR-NC组相比。

*P<0.05 compared with anti-miR-NC group.



A: 克隆形成实验检测细胞增殖; B: Transwell实验检测细胞迁移、侵袭; C: Western blot检测PCNA、E-cadherin、N-cadherin蛋白表达。

A: cell proliferation was determined by the clone formation assay; B: cell migration and invasion were detected by the Transwell assay; C: the protein expression levels of PCNA, E-cadherin, and N-cadherin were determined by Western blot.

图4 抑制miR-195-5p可以部分回复下调LINC00680对IL-17诱导肺癌细胞增殖、迁移和侵袭的影响

Fig.4 Inhibition of miR-195-5p could partially respond to the effect of downregulation of LINC00680 on IL-17-induced proliferation, migration, and invasion of lung cancer cells

表7 抑制miR-195-5p可以部分回复下调LINC00680对IL-17诱导肺癌细胞增殖、迁移和侵袭的影响

Table 7 Inhibition of miR-195-5p could partially respond to the effect of LINC00680 downregulation on IL-17-induced proliferation, migration, and invasion of lung cancer cells

分组 Group	miR-195-5p	克隆细胞数 Number of cloned cells	细胞活力/% Cell viability /%	Ki-67	迁移细胞数 Number of migrating cells	侵袭细胞数 Number of invasion cells	E-cadherin	N-cadherin
IL-17+si-LINC00680+anti-miR-NC	0.98±0.06	81.21±6.42	128.42±8.82	0.50±0.05	59.27±4.39	50.42±4.08	0.45±0.04	0.34±0.04
IL-17+si-LINC00680+anti-miR-195-5p	0.41±0.04*	100.33±7.17*	153.64±10.27*	0.63±0.04*	71.33±6.83*	68.72±6.89*	0.35±0.03*	0.49±0.05*
t	23.173	5.960	5.589	6.091	4.456	6.856	6.000	7.028
P	0.000	0.000	0.000	0.002	0.000	0.000	0.000	0.000

*P<0.05, 与IL-17+si-LINC00680+anti-miR-NC组相比。

*P<0.05 compared with IL-17+si-LINC00680+anti-miR-NC group.

括IL-17A(又称IL-17)、IL-17B、IL-17C、IL-17D、IL-17E(又称IL-25)、IL-17F, 其中IL-17是最早被发现的, 由Th17细胞分泌而来的[11-12], 可促进非小细胞肺癌淋巴管内皮细胞增殖、迁移和成管形成[13]。吴振华等[14]研究结果发现, IL-17受体(IL-17RA)可促进肺腺癌A549细胞增殖、迁移和侵袭。本研究结果显示, IL-17处理肺癌细胞H1299后, 细胞克隆细胞数、细胞活力均增加, 迁移、侵袭数明显增多, Ki67、N-cadherin蛋白反应活性显著增强, E-cadherin蛋白表达水平则明显降低, 通过本研究结果可显示出, IL-17能促进肺癌细胞H1299增殖、迁移及侵袭, 这与上述研究结果相似。

lncRNA是当前医学研究的热点, 在肺癌组织中异常表达, 如lncRNA LUCAT1、lncRNA JPX等在肺癌组织中表达上调, 与肺癌患者肿瘤大小、晚期分期有关, 参与肺癌细胞增殖、迁移、侵袭等过程[15-16]。LINC00680是一种促癌基因, 在肝癌、软组织肉瘤患者组织中表达上调, 与不良预后密切相关[17-18]。肺腺癌组织和细胞内LINC00680表达上调, 体外实验发现抑制LINC00680可抑制A549细胞增殖, 并阻滞周期发展[19]。本研究结果显示, IL-17处理肺癌细胞H1299后LINC00680表达上调, 下调LINC00680可降低IL-17诱导的肺癌细胞H1299克隆细胞数、细胞活力, 减少细胞迁移数量及侵袭数量, 下调Ki67、N-cadherin蛋白表达, 上调E-cadherin蛋白表达, 提示下调LINC00680可抑制IL-17诱导的肺癌细胞增殖、迁移和侵袭。

LncBase预测显示miR-195-5p是LINC00680靶基因之一, 本研究通过双荧光素酶报告实验和qRT-PCR实验证明了LINC00680靶向调控miR-195-5p的表达。有研究结果显示, IL-17诱导的胃癌细胞AGS中miR-195-5p表达下调, 过表达miR-195-5p可有效抑制IL-17诱导的胃癌细胞AGS增殖、迁移和侵袭[20]。多数研究表明, miR-195-5p在肺癌细胞中表达水平降低, 且其可参与肺癌细胞增殖、凋亡、迁移及侵袭等过程[21-22]。但是关于miR-195-5p和IL-17在肺癌细胞中的关系尚不清楚, 本研究发现, IL-17处理肺癌细胞H1299后miR-195-5p表达下调, 提示IL-17能调控miR-195-5p的表达从而影响肺癌的发展。下调LINC00680可增加miR-195-5p表达量, 表明LINC00680靶向负调控miR-195-5p的表达。进一步实验结果显示, 抑制miR-195-5p可以逆转下调LINC00680对IL-17诱导肺癌细胞增殖、迁移和侵袭的作用, 这说明LINC00680通过靶向调控miR-195-5p影响IL-17诱导的肺癌细胞增殖和转移。

综上所述, IL-17处理肺癌细胞H1299后LINC-00680高表达, 下调LINC00680能抑制IL-17诱导的肺癌细胞H1299增殖、迁移、侵袭, 其作用机制可能与miR-195-5p有关, LINC00680与miR-195-5p靶向调控作用为肺癌研究提供了新的靶点, 并为肺癌诊断和治疗提供了新的证据。本实验研究也有不足之处, 关于miR-195-5p和IL-17对肺癌细胞具体作用机制需要进行下一步实验探究。

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