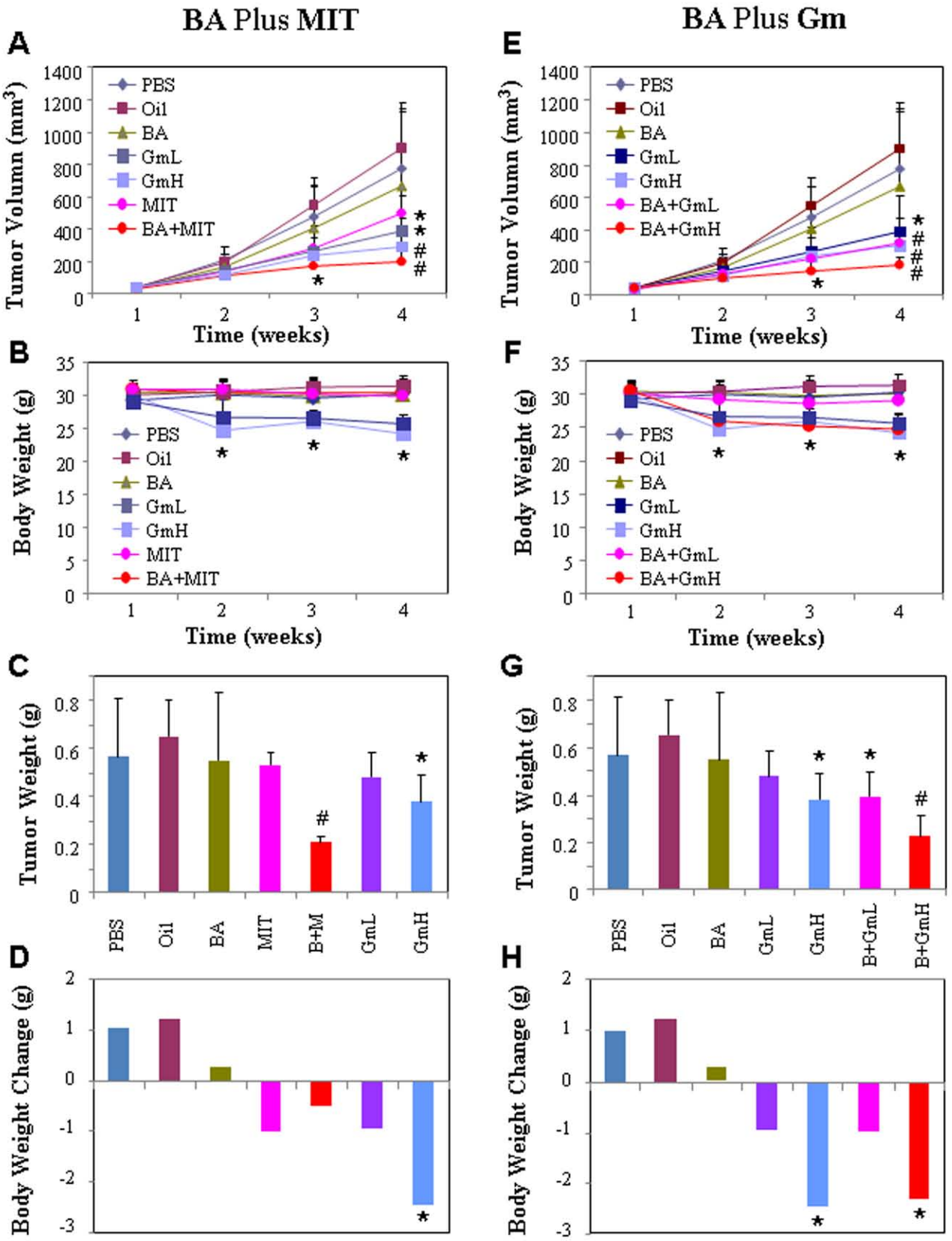
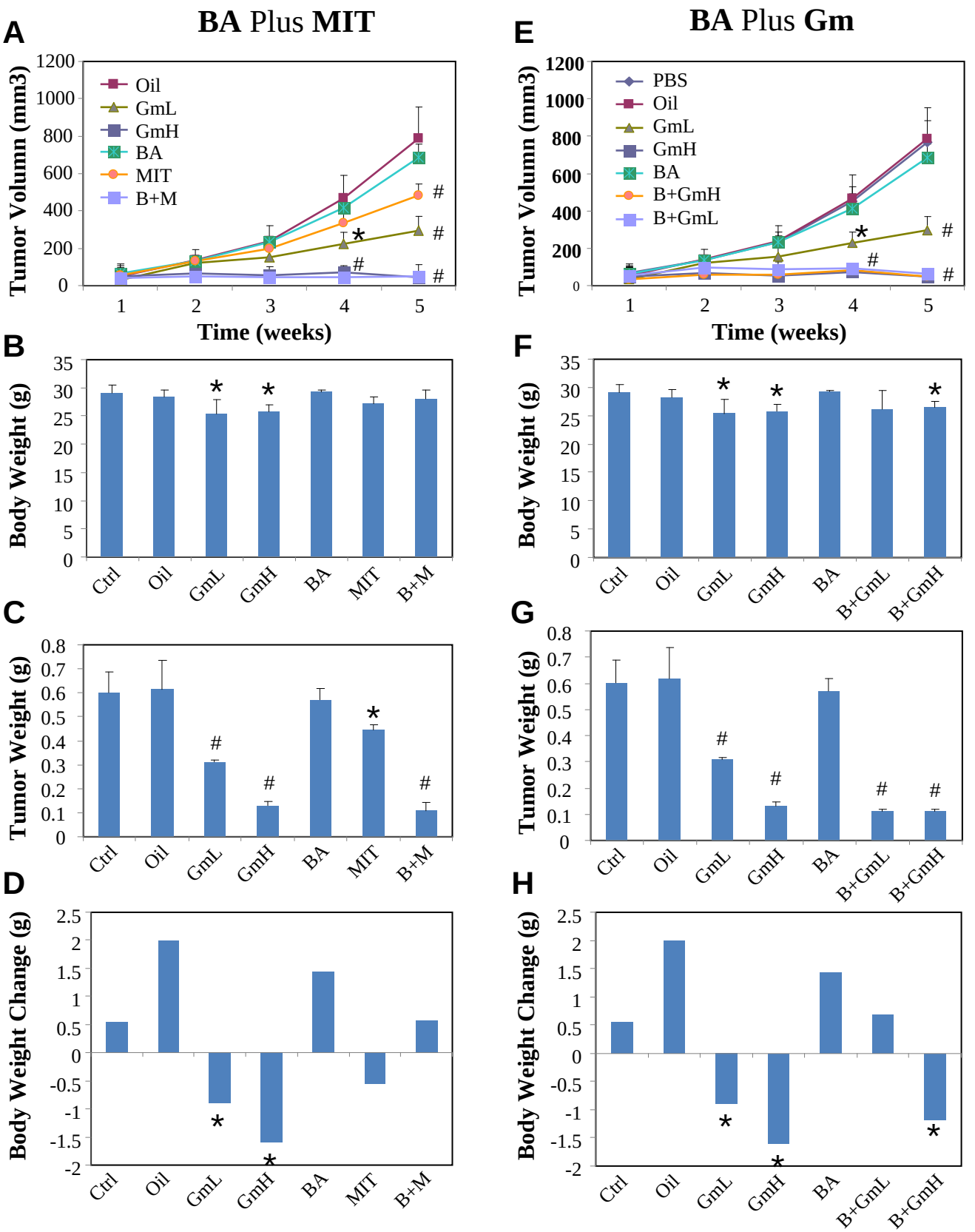
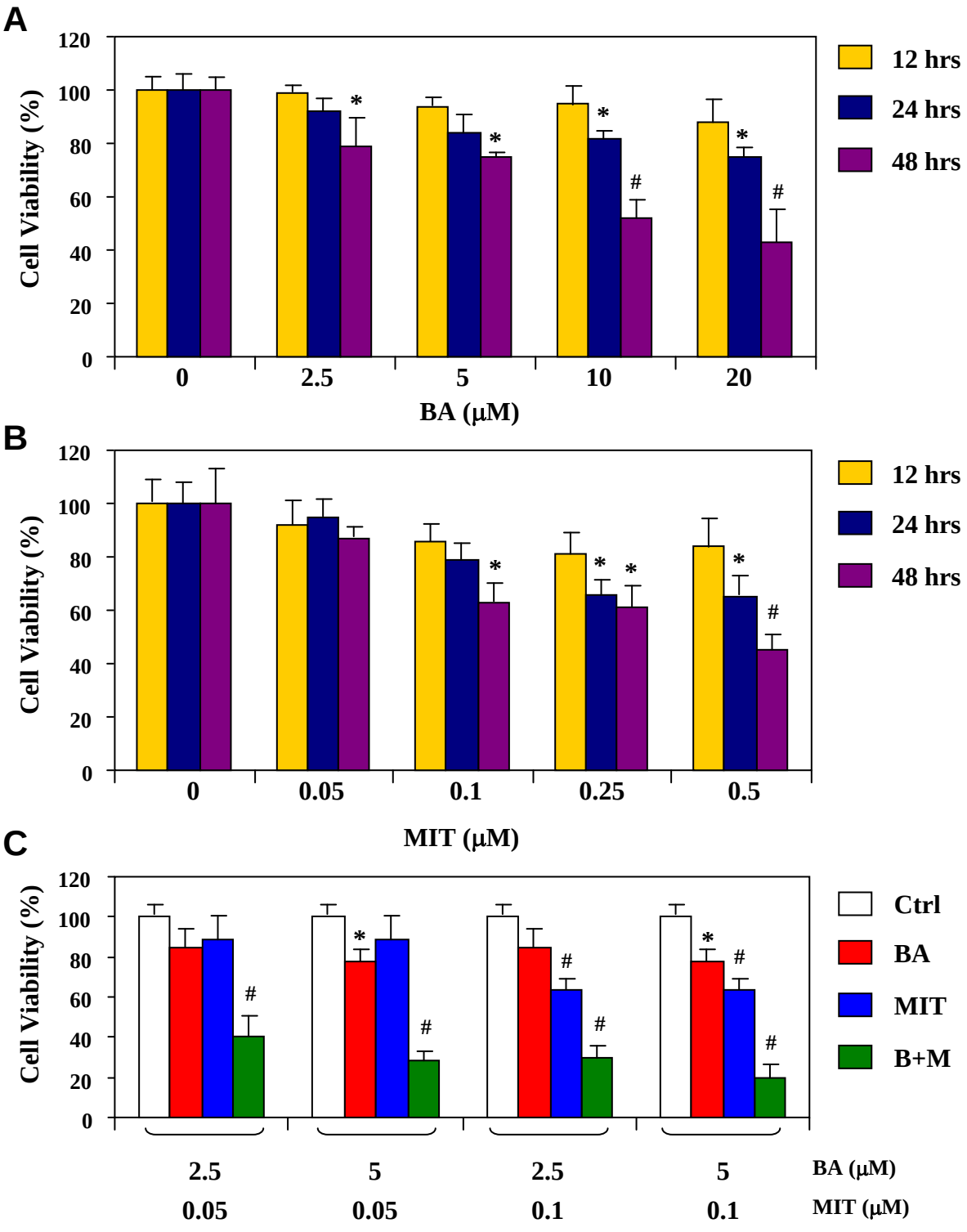
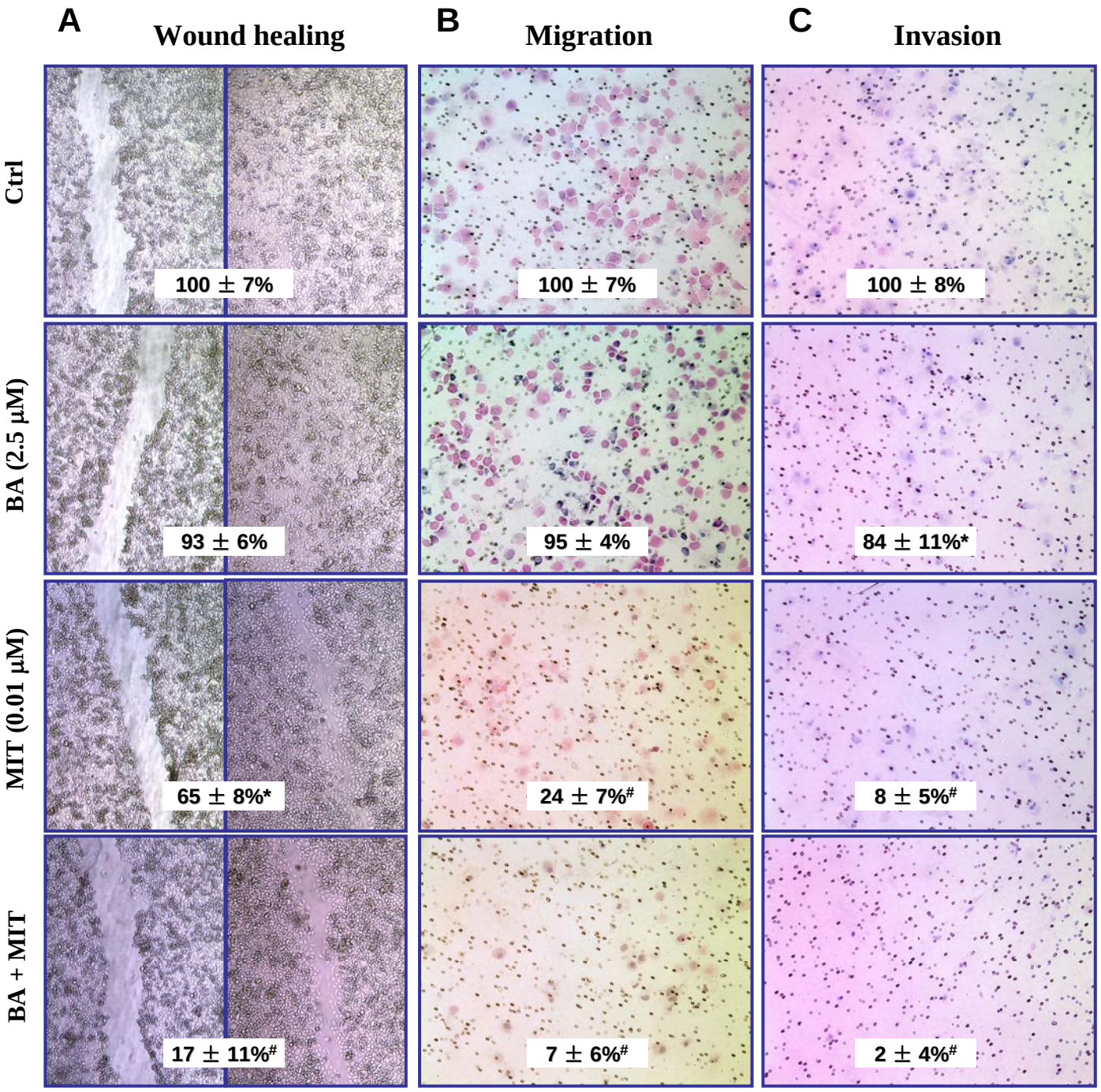
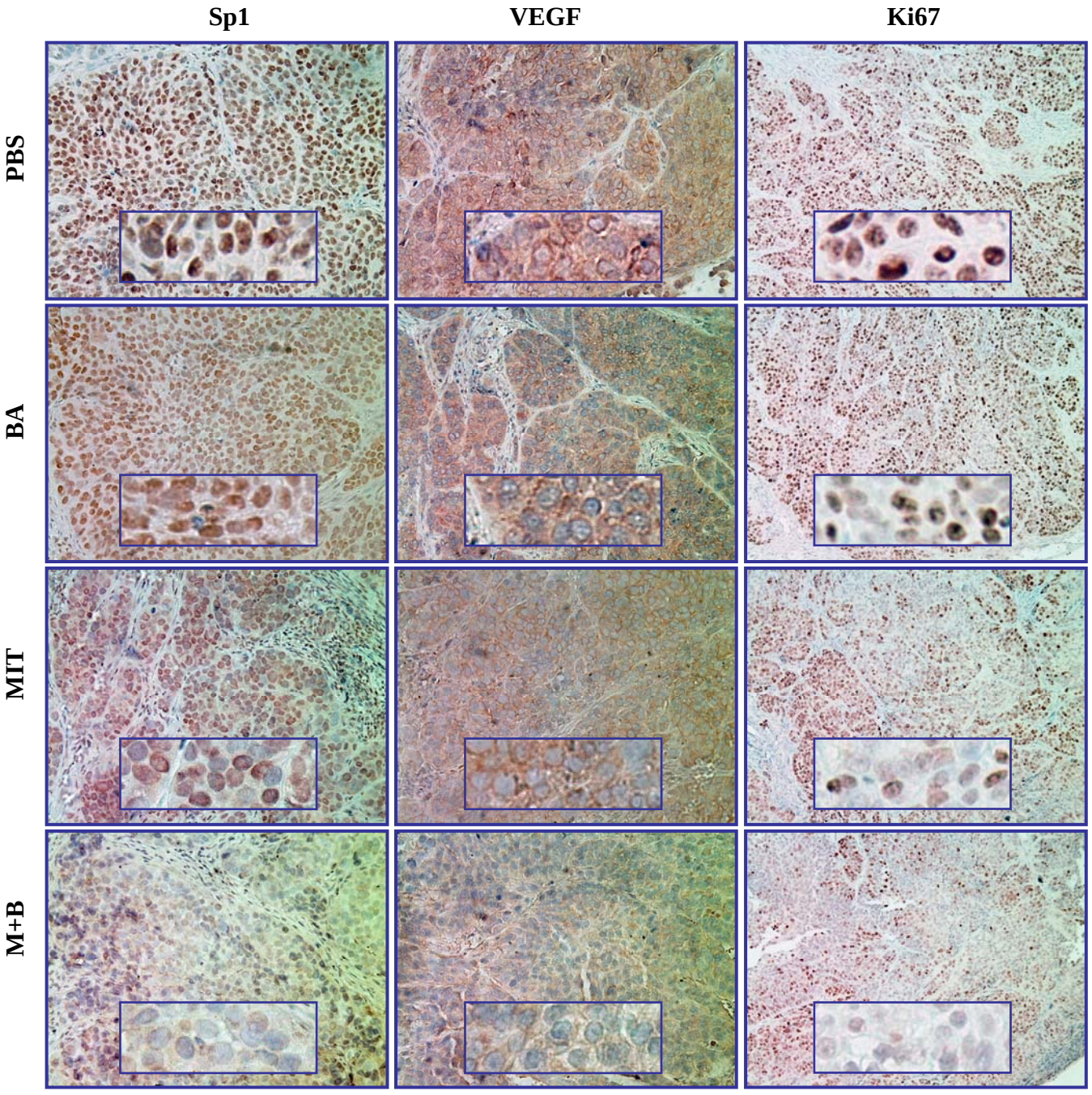
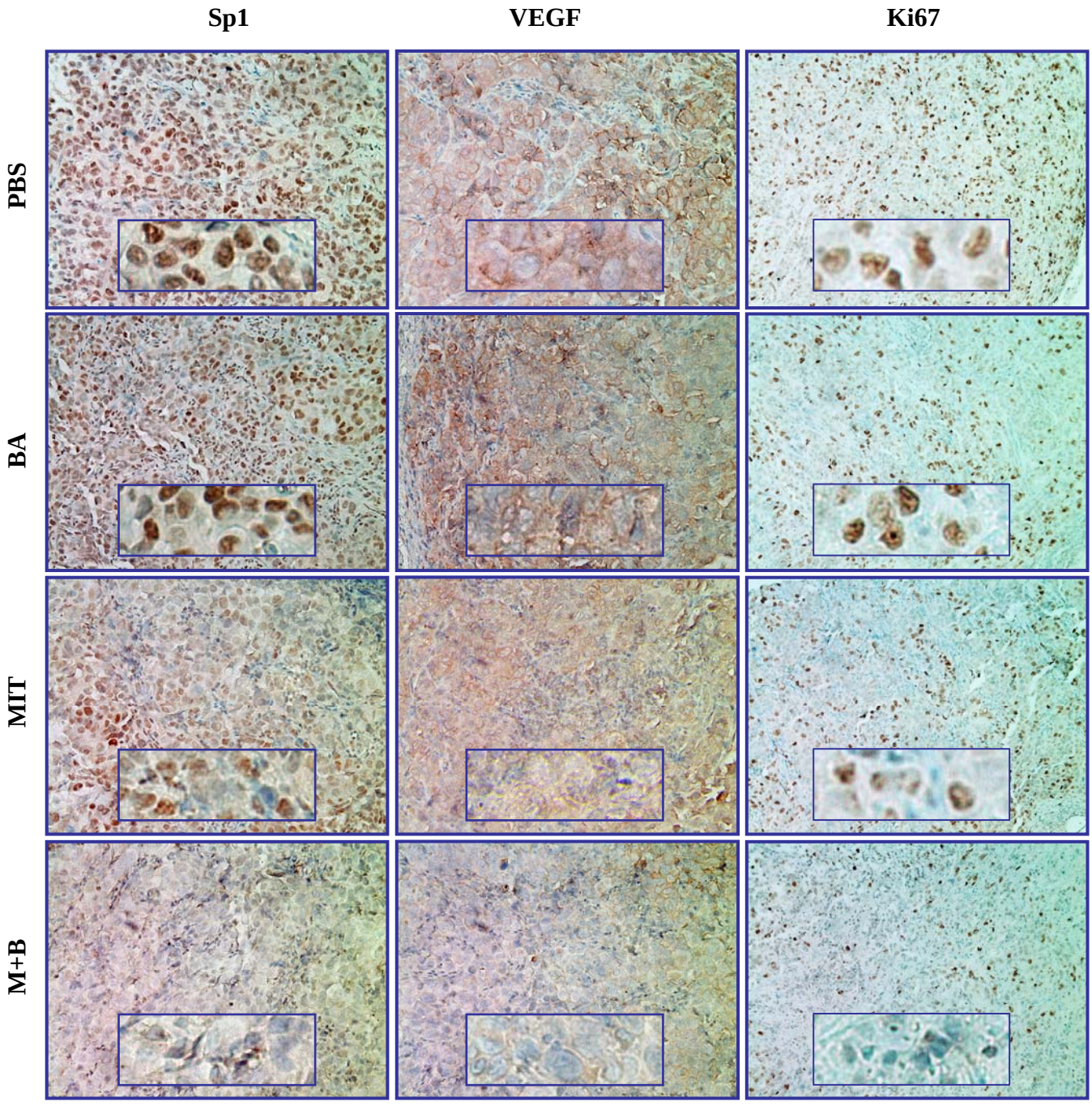

BA Plus MIT
BA Plus Gm
A
E
Tumor Volumn (mm3)
Time (weeks)
PBS
Oil
BA
GmL
GmH
MIT
BA+MIT
BA+GmL
BA+GmH
B
F
Body Weight (g)
C
G
Tumor Weight (g)
PBS
Oil
BA
MIT
B+M
GmL
GmH
B+GmL
B+GmH
D
H
Body Weight Change (g)


A
BA Plus MIT
Tumor Volumn (mm3)
Oil
GmL
GmH
BA
MIT
B+M
Time (weeks)
B
Body Weight (g)
Ctrl
Oil
GmL
GmH
BA
MIT
B+M
C
Tumor Weight (g)
D
Body Weight Change (g)
E
BA Plus Gm
PBS
B+GmH
B+GmL
F
G
H


A
Cell Viability (%)
120
100
80
60
40
20
0
0
2.5
5
10
20
BA (μM)
12 hrs
24 hrs
48 hrs
B
Cell Viability (%)
0
0.05
0.1
0.25
0.5
MIT (μM)
12 hrs
24 hrs
48 hrs
C
Cell Viability (%)
Ctrl
BA
MIT
B+M
2.5
0.05
5
0.05
2.5
0.1
5
0.1
BA (μM)
MIT (μM)


A
Wound healing
B
Migration
C
Invasion
Ctrl
BA (2.5 μM)
MIT (0.01 μM)
BA + MIT
100 ± 7%
100 ± 7%
100 ± 8%
93 ± 6%
95 ± 4%
84 ± 11%*
65 ± 8%*
24 ± 7%#
8 ± 5%#
17 ± 11%#
7 ± 6%#
2 ± 4%#


Sp1
VEGF
Ki67
PBS
BA
MIT
M+B


Sp1
VEGF
Ki67
PBS
BA
MIT
M+B

Supplementary Figure Legends

Figure S1. Antitumor effects of BA, MIT and gemcitabine and their combinations in xenograft models of FG human pancreatic cancer. *Ectopic model*: FG cells were injected into the subcutis of nude mice (n=5). When tumors reached around 4 mm in diameter, the animals received MIT (0.05 mg/kg) via oral gavages three times a week, BA (10 mg/kg), and gemcitabine (GmL: 75 mg/kg; GmH: 150 mg/kg) via intraperitoneal injection twice a week, or in combinations (***A***, ***B***, ***E***, & ***F***). Tumor volumes were measured every week until the mice were killed 45 days after tumor cell injection (***A*** & ***E***); the mice were weighed once every week (***B*** & ***F***). *Orthotopic models*: FG cells were injected into the pancreases of nude mice (n=5). Ten days after tumor injections, the animals received MIT (0.05 mg/kg) via intraperitoneal injection twice a week, BA (10 mg/kg) via oral gavages three times a week, and gemcitabine (GmL: 75 mg/kg; GmH: 150 mg/kg) via intraperitoneal injection twice a week, or in combinations (***C***, ***D***, ***G***, & ***H***). The mice were killed 45 days after tumor cell injection; both tumor weights (***C*** & ***G***) and the mice body weight changes (***D*** & ***H***) were determined. $*P<0.05$ and $^{\#}P<0.01$ as compared to respective controls: "PBS", "Oil"; or both (two tailed student t test).

Note that Panels ***A***, ***B***, ***E*** and ***F*** were from one experiment (ectopic model) and those groups "*PBS*", "*Oil*", "*BA*", "*GmL*" and "*GmH*" in the Panels ***A***, ***B***, ***E*** and ***F*** were the same.

Note that Panels ***C***, ***D***, ***G*** and ***H*** were from another experiment (orthotopic model) and those groups "*PBS*", "*Oil*", "*BA*", "*GmL*" and "*GmH*" in the Panels ***C***, ***D***, ***G*** and ***H*** were the same.

Figure S2. Antitumor effects of BA, MIT and gemcitabine and their combinations in xenograft models of PANC-1 human pancreatic cancer. *Ectopic model*: PANC-1 cells were injected into the subcutis of nude mice (n=5). When tumors reached around 4 mm in diameter, the animals received MIT (0.05 mg/kg) via intraperitoneal injection twice a week, BA (10 mg/kg) via oral gavages three times a week, and gemcitabine (GmL: 75 mg/kg; GmH: 150 mg/kg) via intraperitoneal injection twice a week, or in combinations (***A***, ***B***, ***E***, & ***F***). Tumor volumes were measured every week until the mice were killed 45 days after tumor cell injection (***A*** & ***E***); the mice were weighed at the time of termination of experiments (***B*** & ***F***). *Orthotopic models*: PANC-1 cells were injected into the pancreases of nude mice (n=5). Ten days

after tumor injections, the animals received MIT (0.05 mg/kg) via intraperitoneal injection twice a week, BA (10 mg/kg) via oral gavages three times a week, and gemcitabine (GmL: 75 mg/kg; GmH: 150 mg/kg) via intraperitoneal injection twice a week, or in combinations (***C***, ***D***, ***G***, & ***H***). The mice were killed 45 days after tumor cell injection; both tumor weights (***C*** & ***G***) and the mice body weight changes (***D*** & ***H***) were determined. **P*<0.05 and $^{\#}P$<0.01 as compared to respective controls: "PBS", "Oil"; or both (two tailed student t test).

Note that Panels ***A***, ***B***, ***E*** and ***F*** were from one experiment (ectopic model) and the data in those groups "*PBS*", "*Oil*", "*BA*", "*GmL*" and "*GmH*" in the Panels ***A***, ***B***, ***E*** and ***F*** were the same.

Note that Panels ***C***, ***D***, ***G*** and ***H*** were from another experiment (orthotopic model) and the data in those groups "*PBS*", "*Oil*", "*BA*", "*GmL*" and "*GmH*" in the Panels ***C***, ***D***, ***G*** and ***H*** were the same.

Figure S3. Synergistic effect of treatment with BA and MIT on inhibition of pancreatic cancer cell proliferation. ***A,*** PANC-1 pancreatic cancer cells were treated with BA (1 - 20 μM) for 12 - 48 h. Inhibition of cell proliferation was assessed using an MTT assay. ***B,*** PANC-1 cells were treated with MIT (0.05 – 0.5 μM) for 12 - 48 h. Inhibition of cell proliferation was assessed using an MTT assay. ***C***, PANC-1 cells were treated with 2.5 or 5 μM BA, 0.05 or 0.1 μM MIT or both BA and MIT for 48 h. Inhibition of cell proliferation was assessed using an MTT assay. This was one representative experiment of two with similar results. *p<0.05 and $^{\#}$p<0.01 (two-tailed student *t* test).

Figure S4. Synergistic effect of treatment with BA and MIT on inhibition of pancreatic cancer cell migration and invasion. ***A,*** PANC-1 cells were pretreated with 2.5 μM BA, 0.01 μM MIT, or both for 24 h, the cultures were wounded by scratching and maintained for additional 24 h. Cell cultures were photographed and cell migration was assessed by measuring gap sizes (inserted number represented percent area of gap ± SD). ***B***, PANC-1 cells were pretreated with 2.5 μM BA, 0.01 μM MIT, or both for 24 h, the cells were placed in the upper chambers and maintained for additional 24 h. Representative tumor cells migrated to the lower surface of the filters were photographed, while the numbers of tumor cells that

penetrated through filter were counted in 15 random fields identified within the lower surface of the filters and expressed as % of control (*inserted numbers*). Data represents mean ± SD of triplicates. ***C***, PANC-1 cells were pretreated with 2.5 μM BA, 0.01 μM MIT, or both for 24 h, the cells were placed in the upper chambers with Matrigel-coated filters and maintained for additional 24 h. Representative tumor cell invaded through Matrigel were photographed, while the numbers of invasive cells that penetrated through Matrigel-coated filter were counted in 15 random fields identified within the lower surface of the filters and expressed as % of control (*inserted numbers*). Data represents mean ± SD of triplicates. *$p<0.05$ and $^{\#}p<0.01$ (two-tailed student *t* test).

Figure S5. Effects of BA and MIT treatments on Sp1 and VEGF expression and cell proliferation in FG pancreatic tumors. The FG tumors from mice receiving dissolvent (PBS), BA treatment (described in Fig. 1A), MIT, or both were collected and processed for gene expression analyses by immunostaining of VEGF and Sp1 expression and cell proliferation (Ki67).

Figure S6. Effects of BA and MIT treatments on Sp1 and VEGF expression and cell proliferation in PANC-1 pancreatic tumors. The PANC-1 tumors from mice receiving dissolvent (PBS), BA treatment (described in Fig. 1A), MIT, or both were collected and processed for gene expression analyses by immunostaining of VEGF and Sp1 expression and cell proliferation (Ki67).