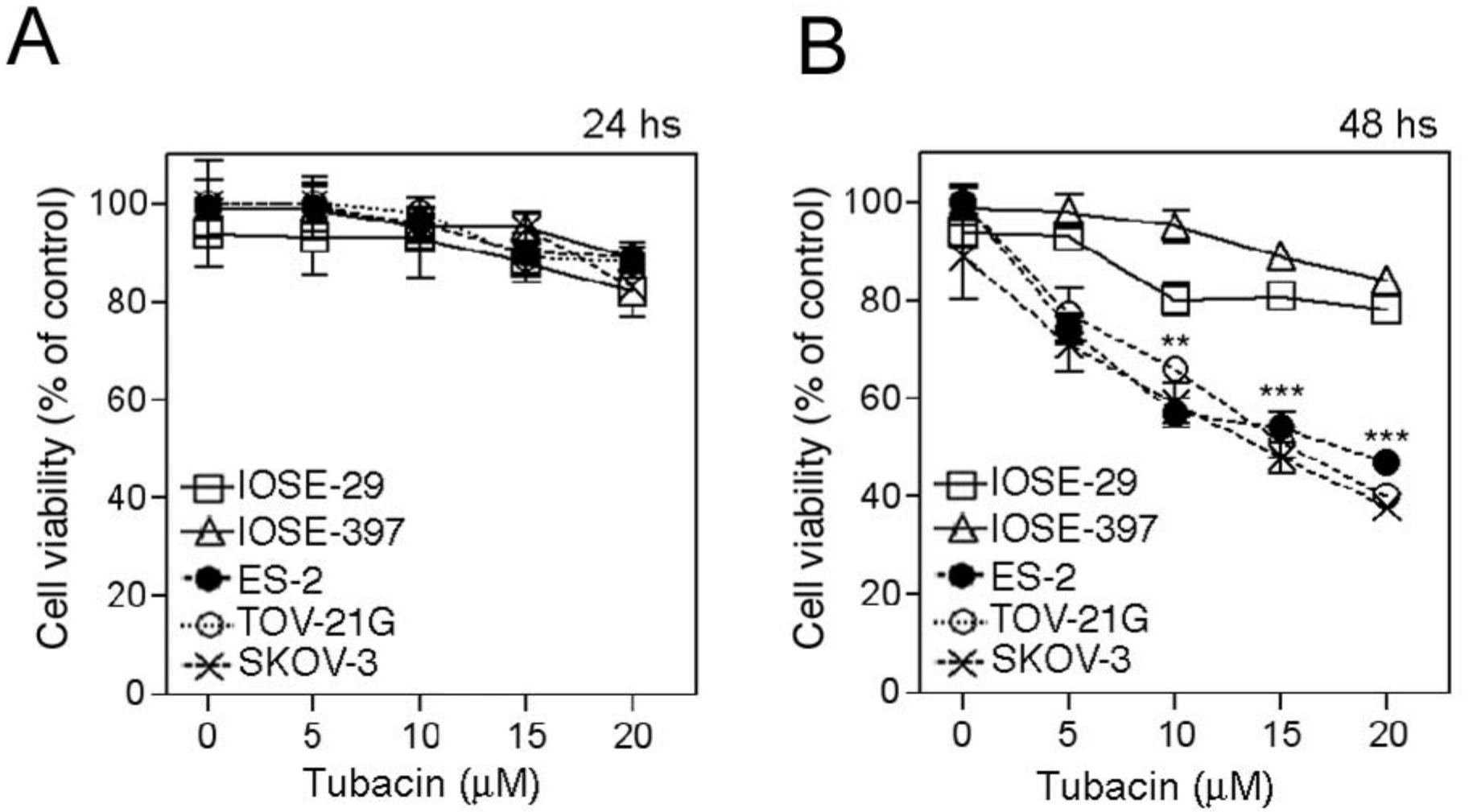
Supplemental Figure 1. Tubacin treatment impacts the viability ovarian cancer cells while sparing IOSEs. A, B. Cell viability of ovarian cancer cell lines (SKOV-3, ES-2, TOV-21G) and immortalized ovarian surface epithelial cells (IOSE-29 and IOSE-397) in the presence of the HDAC6 inhibitor Tubacin. Cell viability was measured by XTT assay after culturing the cells for 24 hours (A) or 48 hours (B) in the presence of Tubacin at the concentrations indicated. Error bars indicate ± SD.

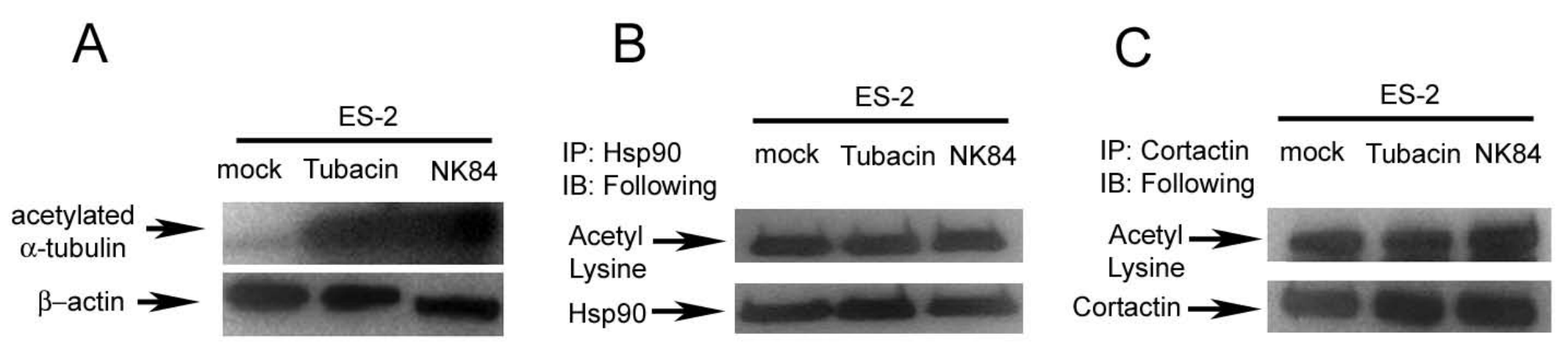
Supplemental Figure 2. NK84 specifically inhibits the α-tubulin deacetylase (TDAC) domain of HDAC6. A. Immunoblot analysis of the levels of acetylated α-tubulin in Tubacin (10 μM) or NK84 (10μM) treated ES-2 ovarian cancer cells. Equal protein loading was verified by using an antibody directed against β-actin. **B.** Cell lysates from ES-2 cells treated or not with Tubacin (10 μM) or NK84 (10μM) were immunoprecipitated with anti-Hsp90 antibody. Immunoprecipitates were immunoblotted for acetylated lysine and Hsp90. **C.** Cell lysates from ES-2 cells treated or not with Tubacin (10 μM) or NK84 (10μM) were immunoprecipitated with anti-cortactin antibody. Immunoprecipitates were immunoblotted for acetylated lysine and cortactin.

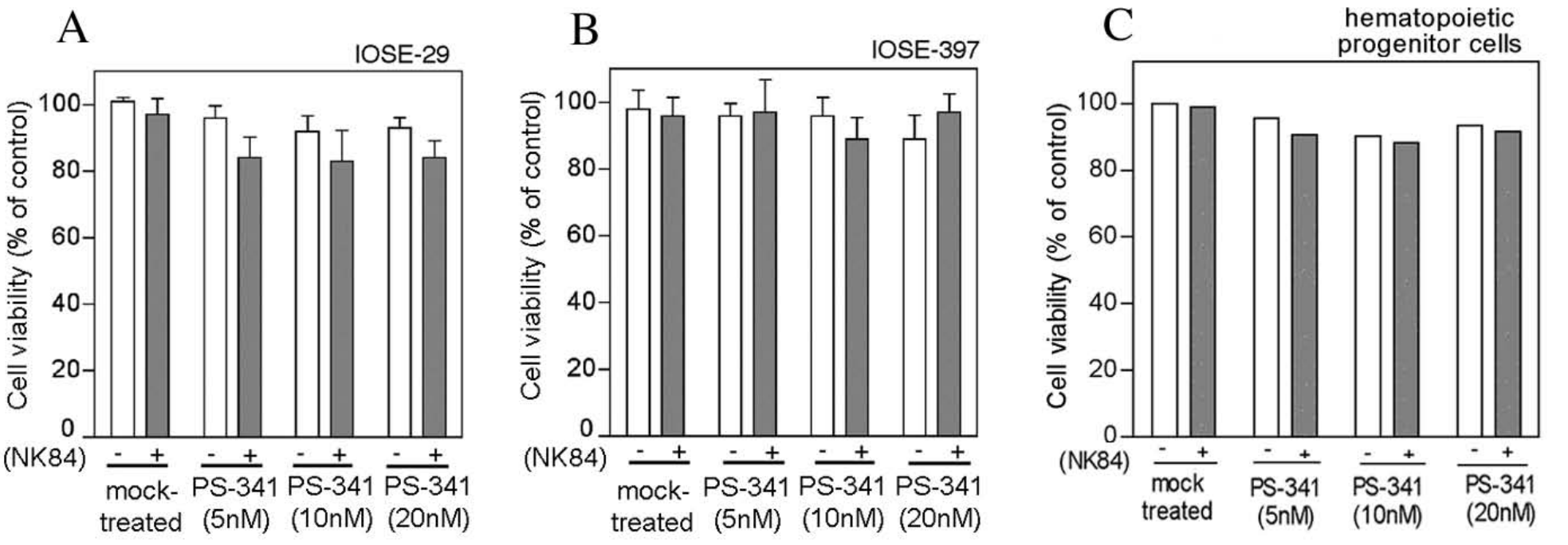
Supplemental Figure 3. Inhibition of HDAC6 and proteasome does not affect the cell viability of IOSEs and hematopoietic precursor cells. A, B. Cell viability of IOSE-29 and IOSE-397 in absence (-) or in presence (+) of 5μM NK84 HDAC6 inhibitor and PS-341 at the indicated concentration over a 24 hours period. Cell viability was measured by XTT assay and percentage of viable cells is relative to mock-treated controls. Error bars indicate ± SD. **C.** Cell viability of CD34+ hematopoietic progenitor cells in absence (-) or in presence (+) of 5μM NK84 HDAC6 inhibitor and PS-341 at the indicated concentration over a 24 hours period. Cell viability

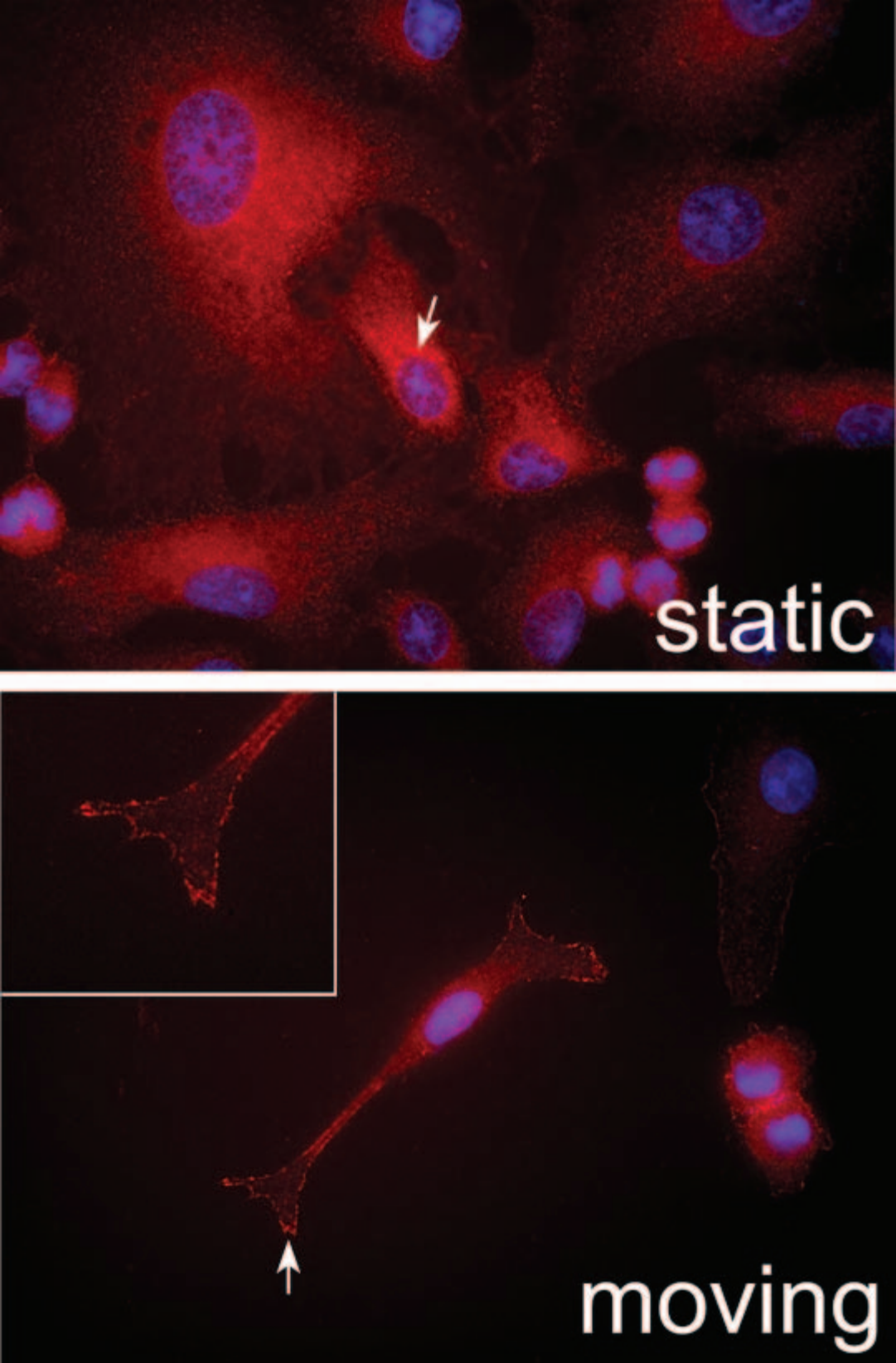
was measured by XTT assay and percentage of viable cells is relative to mock-treated controls. Error bars indicate ± SD.

Supplemental Figure 4. Immunofluorescent localization of HDAC6 at the leading edges of moving ovarian cancer cells. Localization of endogenous HDAC6 (red) and DNA (blue) in confluent, static (*top panel*) versus scratch wounded and moving (*bottom panel*) SKOV-3 ovarian cancer cell cultures. Arrows point toward the region of more intense HDAC6 staining for each culturing condition.


A
24 hs
Cell viability (% of control)
100
80
60
40
20
0
IOSE-29
IOSE-397
ES-2
TOV-21G
SKOV-3
0
5
10
15
20
Tubacin (μM)
B
48 hs
Cell viability (% of control)
100
80
60
40
20
0
**
***
***
IOSE-29
IOSE-397
ES-2
TOV-21G
SKOV-3
0
5
10
15
20
Tubacin (μM)


A
ES-2
mock
Tubacin
NK84
acetylated
α-tubulin
β–actin
B
ES-2
IP: Hsp90
IB: Following
mock
Tubacin
NK84
Acetyl
Lysine
Hsp90
C
ES-2
IP: Cortactin
IB: Following
mock
Tubacin
NK84
Acetyl
Lysine
Cortactin


A
IOSE-29
Cell viability (% of control)
100
80
60
40
20
0
(NK84)
- +
- +
- +
- +
mock-treated
PS-341 (5nM)
PS-341 (10nM)
PS-341 (20nM)
B
IOSE-397
Cell viability (% of control)
100
80
60
40
20
0
(NK84)
- +
- +
- +
- +
mock-treated
PS-341 (5nM)
PS-341 (10nM)
PS-341 (20nM)
C
hematopoietic progenitor cells
Cell viability (% of control)
100
80
60
40
20
0
(NK84)
- +
- +
- +
- +
mock treated
PS-341 (5nM)
PS-341 (10nM)
PS-341 (20nM)


static
moving