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Erratum
Multi-organ metastasis as destination for breast cancer cells guided by biomechanical architecture: Am J Cancer Res. 2021; 11(6): 2537-2567

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In the published article, errors were identified in **Figures 7C** and **12F**, which were inadvertently misused. We have adjusted the order of the result panels in **Figure 7** and revised the corresponding explanations in the text. In **Figure 12F**, the pathological image in the second row, second column has been replaced with the correct version. These corrections do not affect the interpretation of the data or the overall conclusions of the study. We sincerely apologize for any confusion or concern caused by these errors. The corrected versions of **Figures 7** and **12** are provided below.

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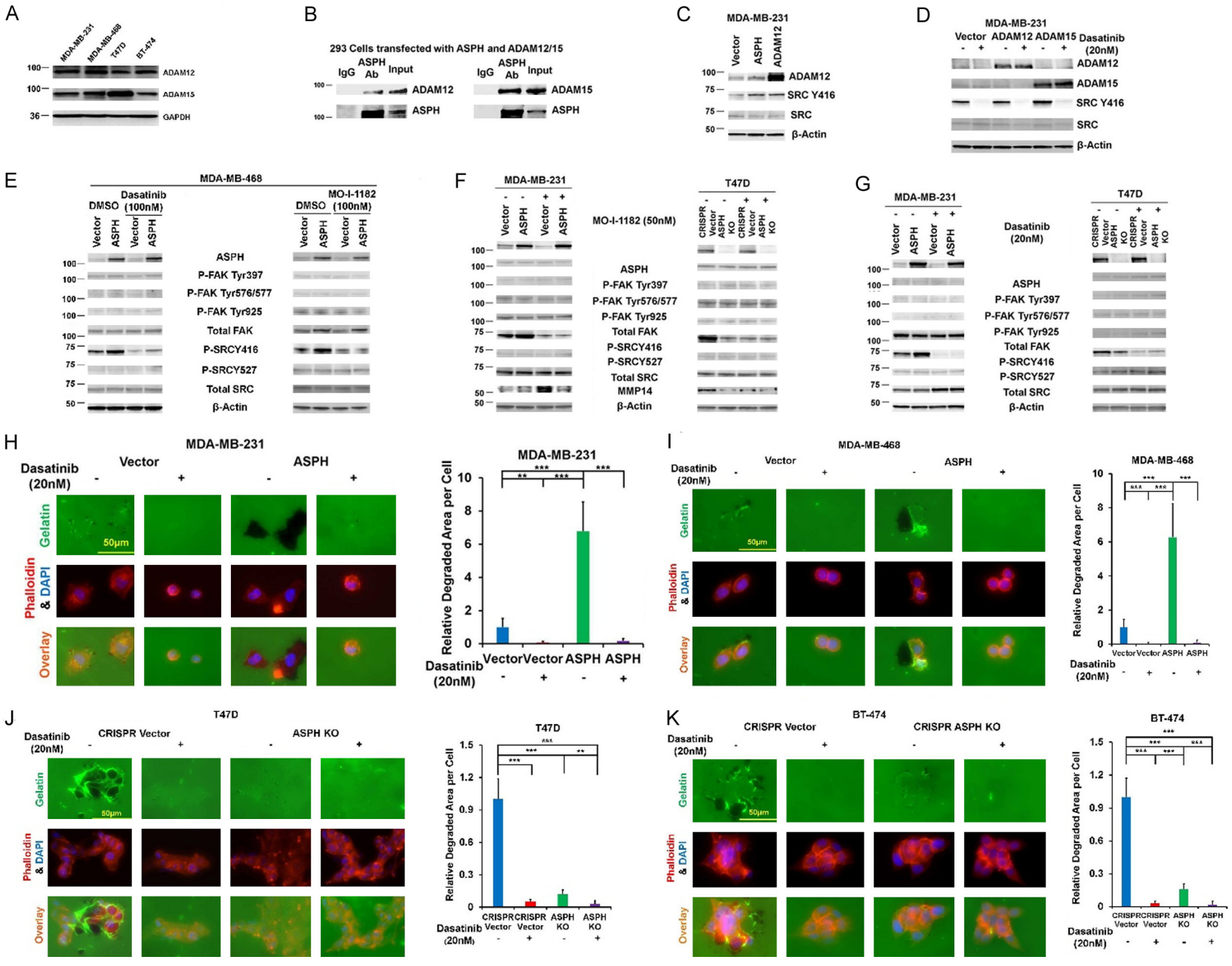
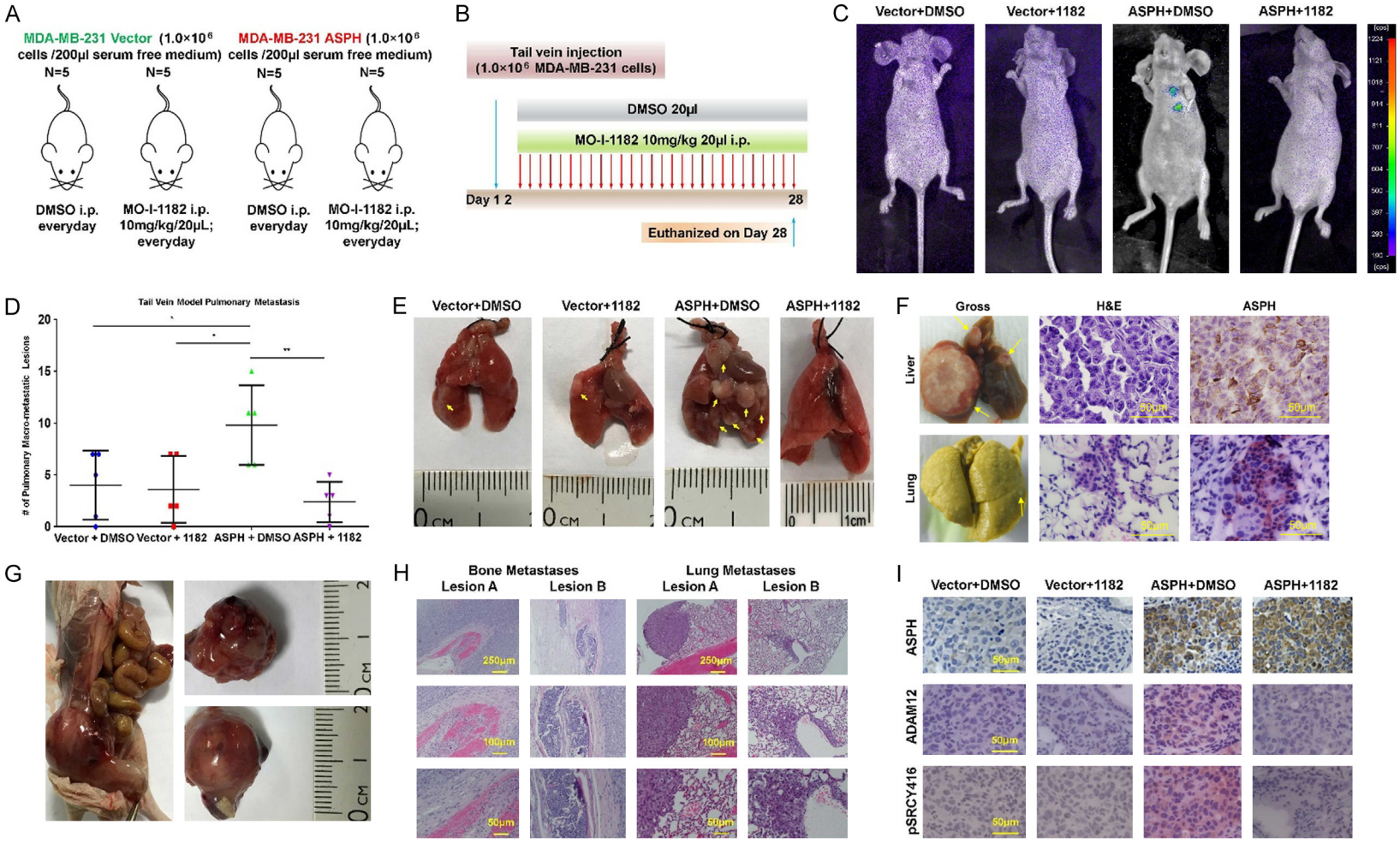

A
MDA-MB-231
MDA-MB-468
T47D
BT-474
ADAM12
ADAM15
GAPDH
B
293 Cells transfected with ASPH and ADAM12/15
IgG
ASPH Ab
Input
ADAM12
ASPH
ADAM15
C
MDA-MB-231
Vector
ASPH
ADAM12
SRC Y416
SRC
β-Actin
D
MDA-MB-231
Vector
ADAM12
ADAM15
Dasatinib (20nM)
SRC Y416
E
MDA-MB-468
DMSO
Dasatinib (100nM)
MO-I-1182 (100nM)
ASPH
P-FAK Tyr397
P-FAK Tyr576/577
P-FAK Tyr925
Total FAK
P-SRCY416
P-SRCY527
Total SRC
β-Actin
F
MDA-MB-231
T47D
MO-I-1182 (50nM)
CRISPR Vector
ASPH KO
MMP14
G
MDA-MB-231
T47D
Dasatinib (20nM)
H
MDA-MB-231
Dasatinib (20nM)
Vector
ASPH
Gelatin
Phalloidin & DAPI
Overlay
50μm
Relative Degraded Area per Cell
I
MDA-MB-468
J
T47D
CRISPR Vector
ASPH KO
K
BT-474
CRISPR ASPH KO

Figure 7. ASPH activates SRC signaling pathway. A. Expression profiling of ADAM12/ADAM15 in breast cancer cell lines. B. As confirmed by co-IP and Western blot, ASPH physically interacted with ADAM12/ADAM15. C. ASPH or ADAM12 overexpression activated SRC. D. ADAM12/ADAM15 induced SRC activation was blocked by Dasatinib (BCR-ABL/SRC family tyrosine kinase inhibitor) in MDA-MB-231 cells. E-G. ASPH significantly enhanced activation of SRC signaling, which was reversed by the SMI and SRC inhibitor Dasatinib. H-K. ECM degradation in response to Dasatinib. *P < 0.05; **P < 0.01; ***P < 0.001.



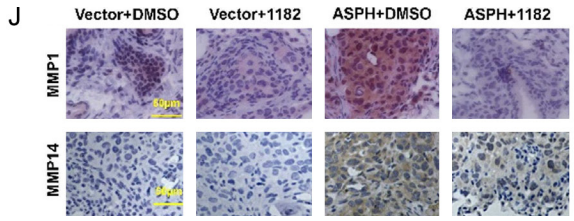


Figure 12. Compared to empty vector, WT-ASPH significantly enhanced metastatic capability of MDA-MB-231 cells, which was efficiently reversed by the SMI in experimental pulmonary metastatic (tail vein injection) murine model. (A) Experimental design and (B) Therapeutic protocol for tail vein injection model (n = 5/group). (C) Using fluorescent imaging system to detect potential pulmonary metastasis in mice from different groups of tail vein injection model. (D) The number of macro-metastases in the lungs derived from mice in tail vein injection model. *P < 0.05; **P < 0.01. (E) Gross appearance of the lungs derived from representative mice in tail vein injection model. Metastatic lesions were highlighted with yellow arrows. (F) Gross appearance and histopathologic characteristics of (Upper) hepatic and (Bottom) pulmonary metastatic lesions of a representative mouse in ASPH+DMSO group of tail vein injection model. Noted the metastatic lesions also maintain high expression of ASPH. This animal was euthanized at 7th weeks. (G) Gross appearance of bone (spine) metastasis derived from a representative mouse in tail vein injection model. The mouse was tail vein injected with ASPH overexpressing MDA-MB-231 cells and treated with DMSO. (H) Histologic characteristics of bone and lung lesions in this specific mouse in (P). (I, J) Expression profiling of key components in SRC signaling pathway (p-SRC Y416, SRC regulator ADAM12 and downstream MMPs) was substantially downregulated by SMI.