Supplementary Information for

ATAD5-BAZ1B interaction modulates PCNA ubiquitination during DNA repair

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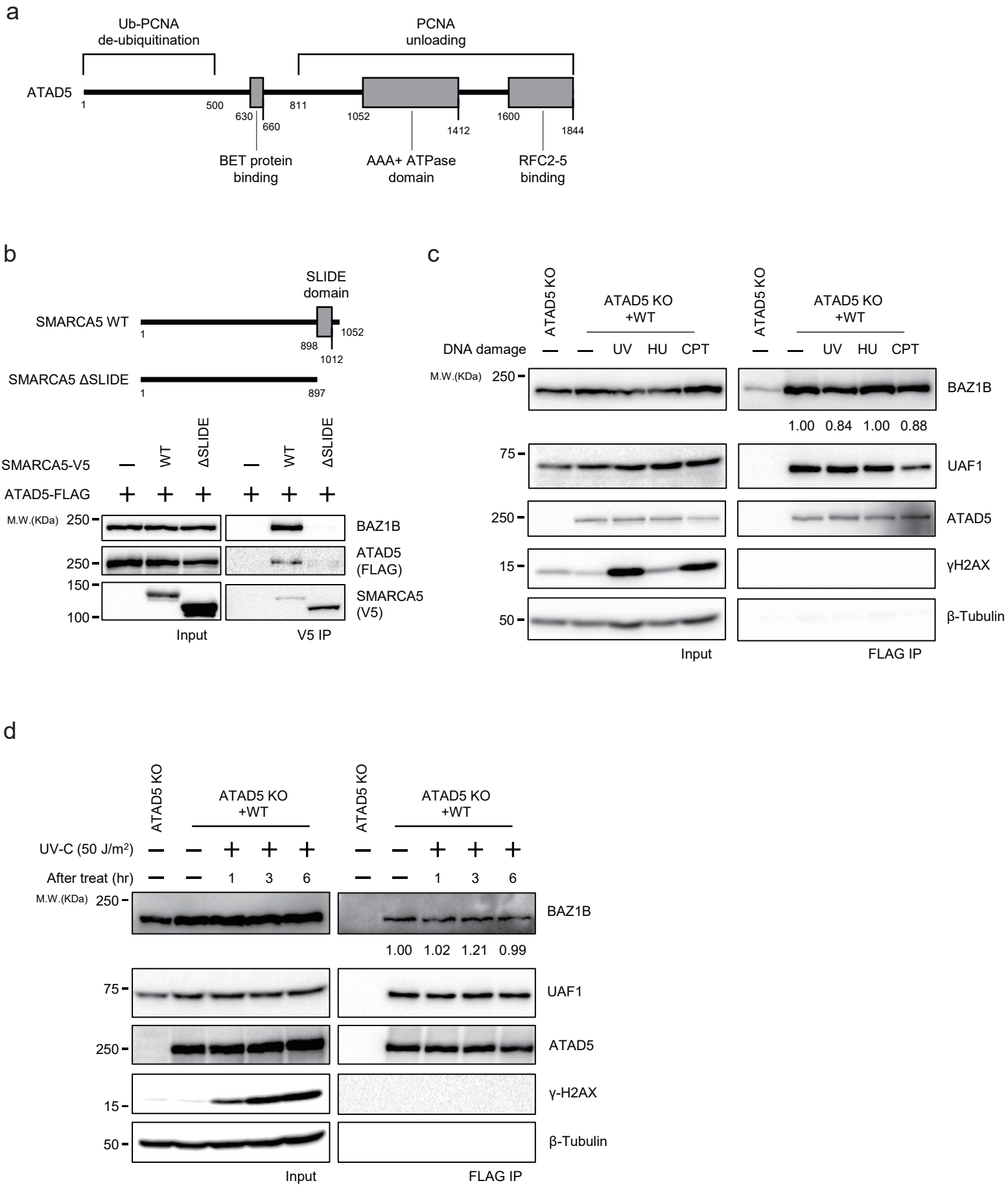
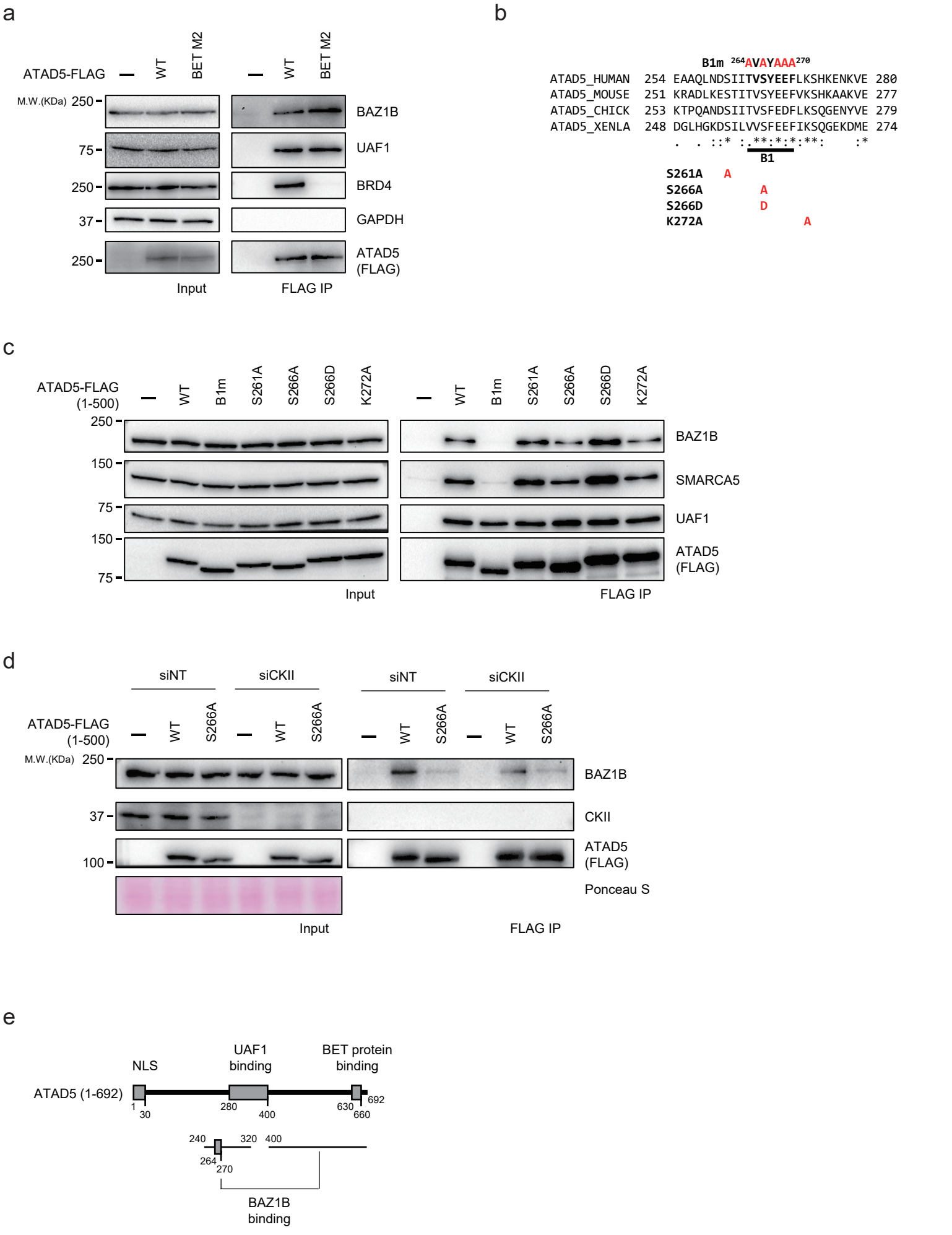
Supplementary Figures S1 to S8

Supplementary Table S1


a
Ub-PCNA
de-ubiquitination
PCNA
unloading
ATAD5
1
500
630
660
811
1052
1412
1600
1844
BET protein
binding
AAA+ ATPase
domain
RFC2-5
binding
b
SLIDE
domain
SMARCA5 WT
1
1052
898
1012
SMARCA5 ΔSLIDE
1
897
SMARCA5-V5
WT
ΔSLIDE
WT
ΔSLIDE
ATAD5-FLAG
M.W.(KDa)
250
BAZ1B
250
ATAD5
(FLAG)
150
100
SMARCA5
(V5)
Input
V5 IP
c
ATAD5 KO
ATAD5 KO
+WT
ATAD5 KO
ATAD5 KO
+WT
DNA damage
UV
HU
CPT
UV
HU
CPT
M.W.(KDa)
250
BAZ1B
1.00
0.84
1.00
0.88
75
UAF1
250
ATAD5
15
γH2AX
50
β-Tubulin
Input
FLAG IP
d
ATAD5 KO
ATAD5 KO
+WT
ATAD5 KO
ATAD5 KO
+WT
UV-C (50 J/m2)
After treat (hr)
1
3
6
1
3
6
M.W.(KDa)
250
BAZ1B
1.00
1.02
1.21
0.99
75
UAF1
250
ATAD5
15
γ-H2AX
50
β-Tubulin
Input
FLAG IP

Supplementary Figure 1. BAZ1B interacts with ATAD5 (related to Figure 1).
(a) Functional motifs of ATAD5. The N-terminal domain of ATAD5 is involved in Ub-PCNA de-ubiquitination, while the C-terminal domain forms PCNA unloading complex with RFC2-5. **(b)** SMARCA5 interacts with ATAD5 through BAZ1B. The upper panel shows the location of human SMARCA5 SLIDE domain, which is essential for interaction with BAZ1B. V5-tagged wild-type or ΔSLIDE SMARCA5 was transiently co-expressed with FLAG-tagged ATAD5 in HEK293T cells. Anti-V5 immunoprecipitation was performed, and the bound proteins were analyzed by immunoblotting. **(c)** UV, Hydroxyurea (HU) or Camptothecin (CPT) does not alter the interaction between ATAD5 and BAZ1B. ATAD5 knock-out (KO) cells expressing wild-type ATAD5 (ATAD5 KO+WT) were treated with UV-C (50 J/m^2), HU (2 mM for 6 hours), or CPT (2 μM for 17 hours), followed by anti-FLAG immunoprecipitation. UV-treated cells were incubated in fresh media for 6 hours. Numbers below the BAZ1B blot indicate the relative amount of BAZ1B. **(d)** ATAD5 KO+WT cells were treated with UV-C (50 J/m^2) and then incubated in fresh media for the indicated times. At each time point, cells were harvested, and anti-FLAG immunoprecipitation was performed. Numbers below the BAZ1B blot indicate the relative amount of BAZ1B. Source data are provided as a Source Data file.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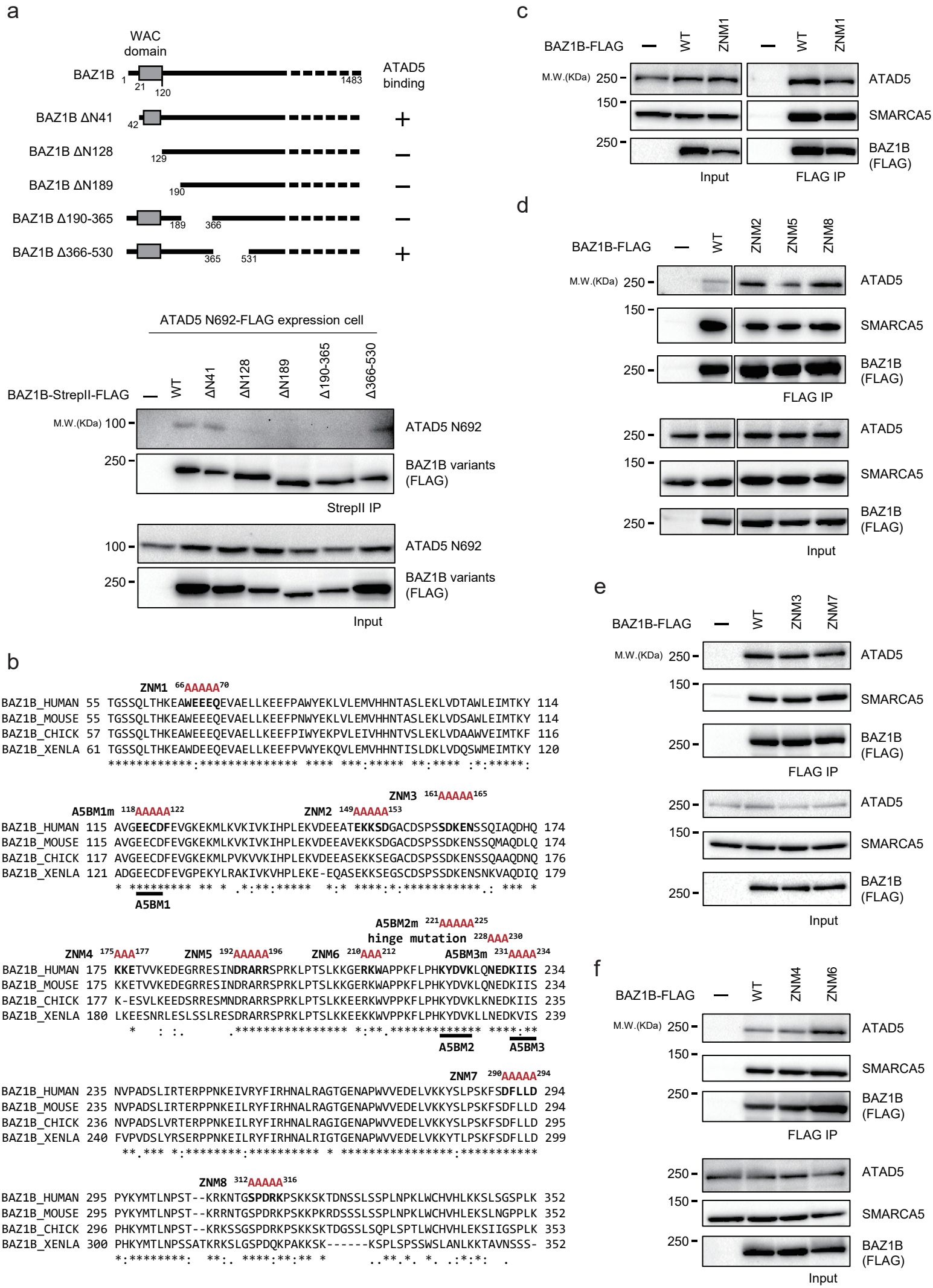
WT
BET M2
M.W.(KDa)
250
75
250
37
250
BAZ1B
UAF1
BRD4
GAPDH
ATAD5 (FLAG)
Input
FLAG IP
b
B1m 264AVAYAAA270
ATAD5_HUMAN 254 EAAQLNDSIITVSYEEFLKSHKENKVE 280
ATAD5_MOUSE 251 KRADLKESTITVSYEEFVKSHKAAKVE 277
ATAD5_CHICK 253 KTPQANDSIITVSFEDFLKSQGENYVE 279
ATAD5_XENLA 248 DGLHGKDSILVVSFEEFIKSQGEKDME 274
B1
S261A A
S266A A
S266D D
K272A A
c
ATAD5-FLAG (1-500)
WT
B1m
S261A
S266A
S266D
K272A
250
150
75
150
75
BAZ1B
SMARCA5
UAF1
ATAD5 (FLAG)
Input
FLAG IP
d
siNT
siCKII
ATAD5-FLAG (1-500)
WT
S266A
M.W.(KDa)
250
37
100
BAZ1B
CKII
ATAD5 (FLAG)
Ponceau S
Input
FLAG IP
e
NLS
UAF1 binding
BET protein binding
ATAD5 (1-692)
1
30
280
400
630
660
692
240
320
400
264
270
BAZ1B binding

Supplementary Figure 2. ATAD5 (264-270) is crucial for BAZ1B binding (related to Figure 2).

(a) BAZ1B binds to ATAD5 independently of BRD4. FLAG-tagged wild-type or BET M2 ATAD5 (a BET-binding mutant) was transiently expressed, followed by anti-FLAG immunoprecipitation. **(b)** The ATAD5 BAZ1B-binding motif is conserved across species. The locations of the B1m mutation and other single amino acid mutations are indicated. **(c)** ATAD5 B1m (^{264}TVSYEEF^{270} to AVAYAAA) fails to interact with endogenous BAZ1B and SMARCA5. Anti-FLAG-immunoprecipitation was performed with the indicated cells expressing wild-type or mutant FLAG-ATAD5 (1-500). **(d)** Depletion of CKII does not affect the interaction between ATAD5 (1-500) and BAZ1B. Wild-type or S266A mutant ATAD5 (1-500) was transiently expressed in 293T cells, in which CKII was depleted by siRNA. ATAD5 (1-500) was immunoprecipitated using anti-FLAG beads and co-isolated proteins were analyzed by immunoblotting. **(e)** Diagram illustrating the positions of BAZ1B binding motifs within the ATAD5 de-ubiquitination domain. Source data are provided as a Source Data file.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WAC domain
BAZ1B
1
21
120
1483
ATAD5 binding
BAZ1B ΔN41
42
+
BAZ1B ΔN128
129
−
BAZ1B ΔN189
190
−
BAZ1B Δ190-365
189
366
−
BAZ1B Δ366-530
365
531
+
ATAD5 N692-FLAG expression cell
BAZ1B-StrepII-FLAG
−
WT
ΔN41
ΔN128
ΔN189
Δ190-365
Δ366-530
M.W.(KDa)
100
250
ATAD5 N692
BAZ1B variants (FLAG)
StrepII IP
100
250
ATAD5 N692
BAZ1B variants (FLAG)
Input
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BAZ1B_MOUSE 55 TGSSQLTHKEAWEEEQEVAELLKEEFPNWYEKLVLEMVHHNTASLEKLVDSAWLEIMTKY 114
BAZ1B_CHICK 57 TGSSQLTHKEAWEEEQEVAELLKEEFPIWYEKPVLEIVHHNTVSLEKLVDAAWVEIMTKF 116
BAZ1B_XENLA 61 TGSSQLTHKEAWDEEQEVAELLKEEFPVWYEKQVLEMVHHNTISLDKLVDQSWMEIMTKY 120
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A5BM1m 118AAAAA122
ZNM2 149AAAAA153
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BAZ1B_MOUSE 115 AVGEECDFEVGKEKMLKVKIVKIHPLEKVDEEAVEKKSDGACDSPSSDKENSSQMAQDLQ 174
BAZ1B_CHICK 117 AVGEECDFEVGKEKMLPVKVVKIHPLEKVDEEASEKKSEGACDSPSSDKENSSQAAQDNQ 176
BAZ1B_XENLA 121 ADGEECDFEVGPEKYLRAKIVKVHPLEKE-EQASEKKSEGSCDSPSSDKENSNKVAQDIQ 179
A5BM1
A5BM2m 221AAAAA225
hinge mutation 228AAA230
ZNM4 175AAA177
ZNM5 192AAAAA196
ZNM6 210AAA212
A5BM3m 231AAAA234
BAZ1B_HUMAN 175 KKETVVKEDEGRRESINDRARRSPRKLPTSLKKGERKWAPPKFLPHKYDVKLQNEDKIIS 234
BAZ1B_MOUSE 175 KKETVVKEDEGRRESINDRARRSPRKLPTSLKKGERKWAPPKFLPHKYDVKLQNEDKIIS 234
BAZ1B_CHICK 177 K-ESVLKEEDSRRESMNDRARRSPRKLPTSLKKEERKWVPPKFLPHKYDVKLKNEDKIIS 235
BAZ1B_XENLA 180 LKEESNRLESLSSLRESDRARRSPRKLPTSLKKEEKKWVPPKFLPHKYDVKLLNEDKVIS 239
A5BM2
A5BM3
ZNM7 290AAAAA294
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BAZ1B_MOUSE 235 NVPADSLIRTERPPNKEILRYFIRHNALRAGTGENAPWVVEDELVKKYSLPSKFSDFLLD 294
BAZ1B_CHICK 236 NVPADSLVRTERPPNKEILRYFIRHNALRAGIGENAPWVVEDELVKKYSLPSKFSDFLLD 295
BAZ1B_XENLA 240 FVPVDSLYRSERPPNKEILRYFIRHNALRIGTGENAPWVVEDELVKKYTLPSKFSDFLLD 299
ZNM8 312AAAAA316
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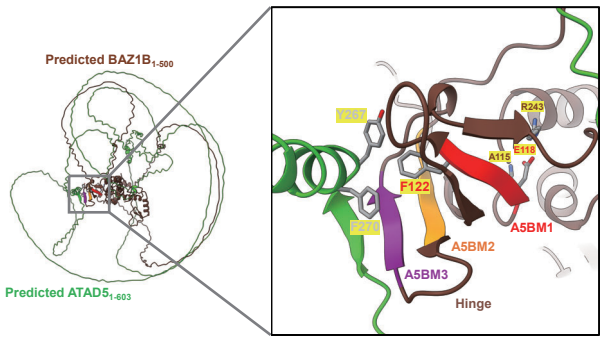
BAZ1B_MOUSE 295 PYKYMTLNPST--KRRNTGSPDRKPSKKPKRDSSSLSSPLNPKLWCHVHLEKSLNGPPLK 352
BAZ1B_CHICK 296 PHKYMTLNPST--KRKSSGSPDRKPSKKSKTDGSSLSQPLSPTLWCHVHLEKSIIGSPLK 353
BAZ1B_XENLA 300 PHKYMTLNPSSATKRKSLGSPDQKPAKKSK------KSPLSPSSWSLANLKKTAVNSSS- 352
c
BAZ1B-FLAG
−
WT
ZNM1
−
WT
ZNM1
M.W.(KDa)
250
150
250
ATAD5
SMARCA5
BAZ1B (FLAG)
Input
FLAG IP
d
BAZ1B-FLAG
−
WT
ZNM2
ZNM5
ZNM8
M.W.(KDa)
250
150
250
ATAD5
SMARCA5
BAZ1B (FLAG)
FLAG IP
250
150
250
ATAD5
SMARCA5
BAZ1B (FLAG)
Input
e
BAZ1B-FLAG
−
WT
ZNM3
ZNM7
M.W.(KDa)
250
150
250
ATAD5
SMARCA5
BAZ1B (FLAG)
FLAG IP
250
150
250
ATAD5
SMARCA5
BAZ1B (FLAG)
Input
f
BAZ1B-FLAG
−
WT
ZNM4
ZNM6
M.W.(KDa)
250
150
250
ATAD5
SMARCA5
BAZ1B (FLAG)
FLAG IP
250
150
250
ATAD5
SMARCA5
BAZ1B (FLAG)
Input

g



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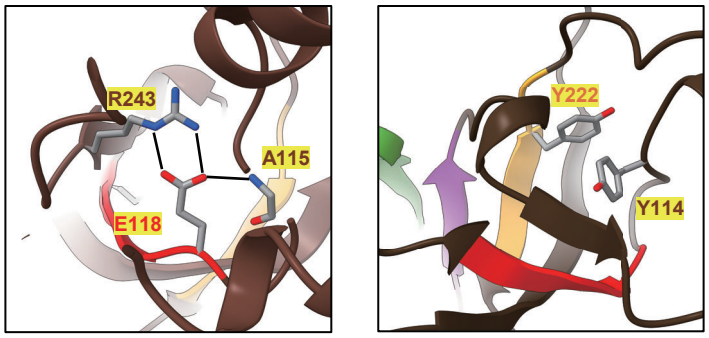

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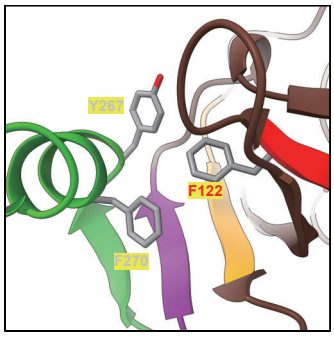

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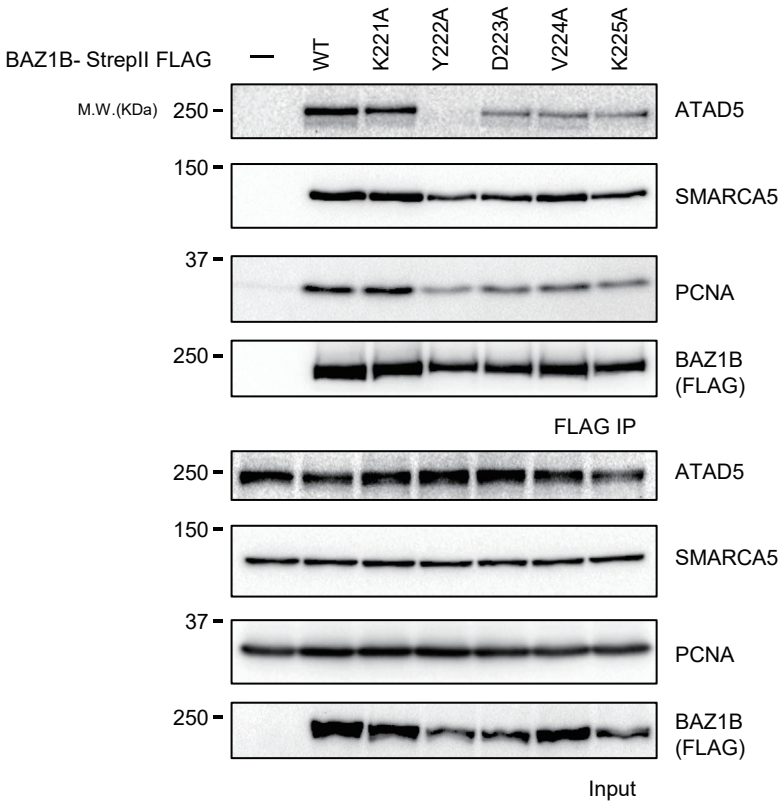

i

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ATAD5_MOUSE  251 KRADLKESTITVSYEEFVKSHKAAKVE 277
ATAD5_CHICK  253 KTPQANDSIITVSFEDFLKSQGENYVE 279
ATAD5_XENLA  248 DGLHGKDSILVVSFEEFIKSQGEKDME 274
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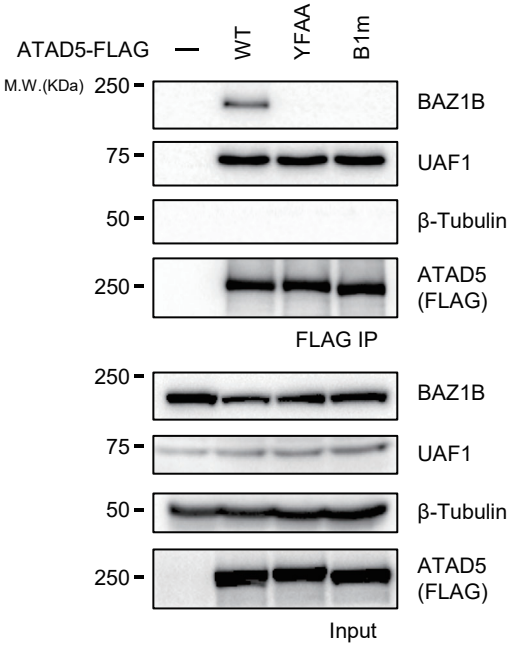

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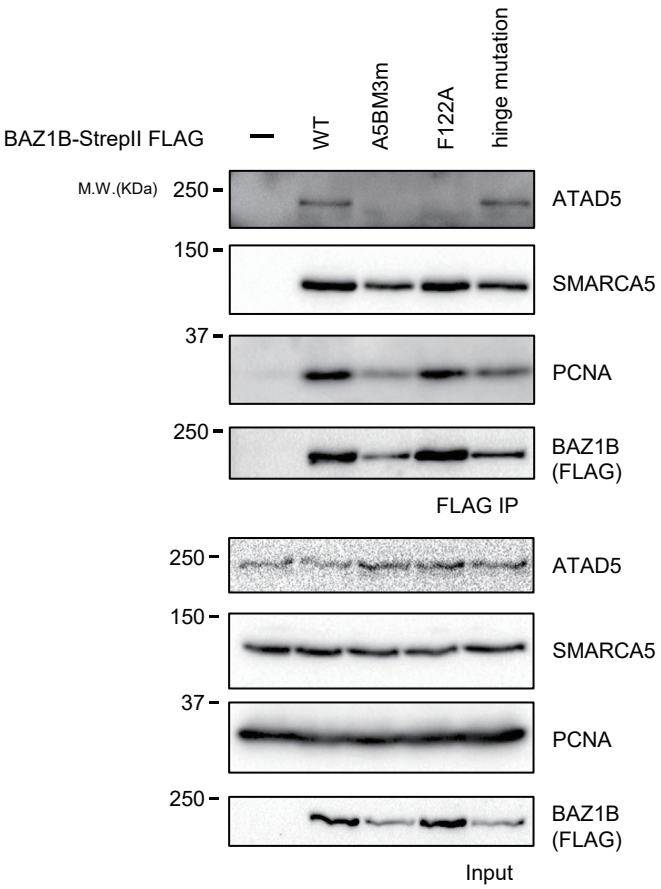
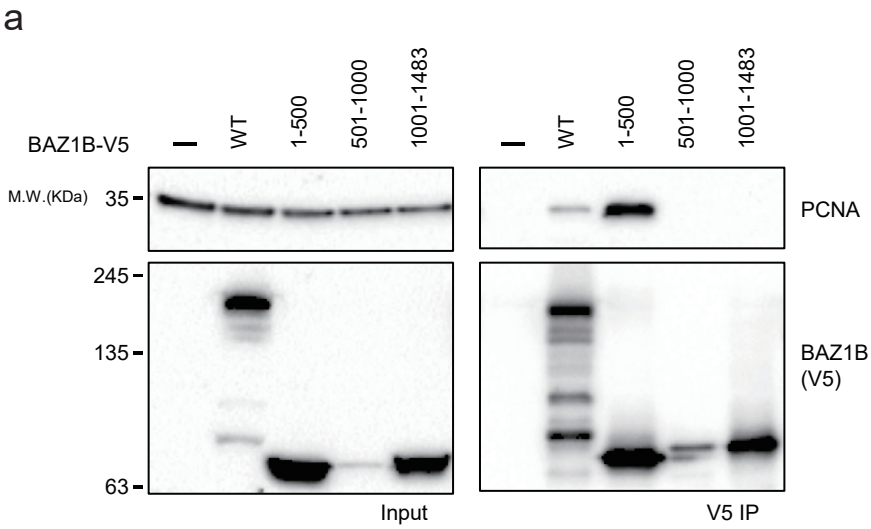


Supplementary Figure 3. BAZ1B N-terminal domain binds to ATAD5 (related to Figure 3).

(a) BAZ1B residues 42-365 are essential for the interaction with ATAD5. StrepII-FLAG-tagged wild-type or indicated BAZ1B deletion mutants were transiently expressed in cells expressing FLAG-ATAD5 (1-692), followed by Strep-Tactin pull-down. The upper panel summarizes the locations of deletions in BAZ1B mutants and their ATAD5 binding capacities. **(b)** The locations of ZNM1-8 mutations, A5BM1m, A5BM2m, A5BM3m, and the hinge mutation are indicated. **(c-f)** Other conserved motifs surrounding the A5BM1 or A5BM2 motifs are not essential for ATAD5 interaction. Indicated BAZ1B alanine substitution mutants were expressed in 293T cells, and FLAG pull-down was performed. **(g)** AlpfaFold2 prediction of ATAD5 (1-603) bound to BAZ1B (1-500). **(h)** The residues Y267 and F270 of ATAD5 form a parallel-displaced (staggered stacking) configuration of aromatic π-π interactions with the BAZ1B F122 residue. **(i)** The residues Y264 and F270 of ATAD5 are conserved among species. The location of the YFAA mutations (Y264A, F270A) were indicated. **(j)** ATAD5 YFAA fails to interact with BAZ1B. Anti-FLAG-immunoprecipitation was performed with cells expressing either wild-type or mutant FLAG-ATAD5. **(k)** The BAZ1B motifs A5BM1, A5BM2, and A5BM3 constitute the ATAD5 binding module and support the ATAD5-BAZ1B interaction. The Y222 residue in the BAZ1B A5BM2 motif forms a T-shaped π-π stacking interaction with the Y114 residue in BAZ1B, stabilizing the positioning of the A5BM1 motif. The BAZ1B E118 residue stabilizes the orientation of the β-sheet-based motif toward ATAD5. **(l)** The BAZ1B Y222A mutant failed to interact with ATAD5. Anti-FLAG-immunoprecipitation was performed with cells expressing either wild-type or mutant StrepII-FLAG-BAZ1B. **(m)** The BAZ1B F122A mutation and A5BM3 mutant (^{231}KIIS^{234} to AAAA) fail to interact with ATAD5. The BAZ1B hinge mutant (^{228}NED^{230} to AAA) does not affect the ATAD5-BAZ1B interaction. Anti-FLAG-immunoprecipitation was performed with cells expressing either wild-type or mutant StrepII-FLAG-BAZ1B. Source data are provided as a Source Data file.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BAZ1B-V5
—
WT
1-500
501-1000
1001-1483
M.W.(KDa)
35
245
135
63
PCNA
BAZ1B
(V5)
Input
V5 IP


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ZNM10 358AAAAAAA364
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BAZ1B_MOUSE 323 RDSSSLSSPLNPKLWCHVHLEKSLNGPPLKVKNSKNSKSPEEHLEGVMKIM 373
BAZ1B_CHICK 324 TDGSSLSQPLSPTLWCHVHLEKSIIGSPLKPKEELEEMMKIMSPGKL-DSN 373
BAZ1B_XENLA 330 ------KSPLSPSSWSLANLKKTAVNSSS----------SE----EEMQLM 360
ZNM12 423AAAAA427
ZNM11 413AGAA416
PIPm 436AAGAG440
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BAZ1B_MOUSE 402 PLKAKGRGKGILNGQKSTGNSKSPSKCVKTPKTKMKQMTLLDMAKGTQKMT 452
BAZ1B_CHICK 398 SLDKKQRGKRVLNGQKTSGKSKSPGKGLKTPKMKMKQMTLLDMAKGTTKVS 448
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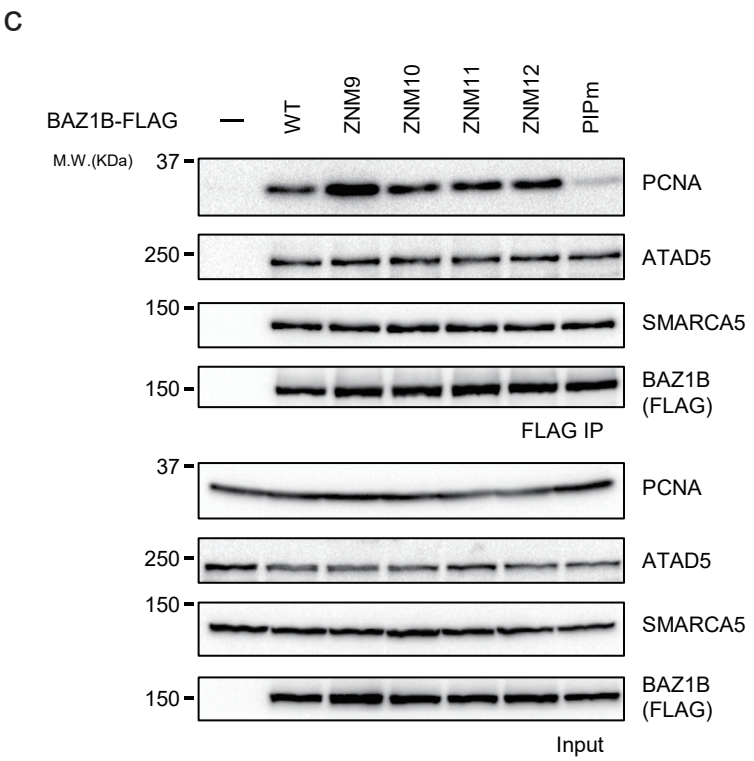
PIP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BAZ1B-FLAG
—
WT
ZNM9
ZNM10
ZNM11
ZNM12
PIPm
M.W.(KDa)
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250
150
150
PCNA
ATAD5
SMARCA5
BAZ1B
(FLAG)
FLAG IP
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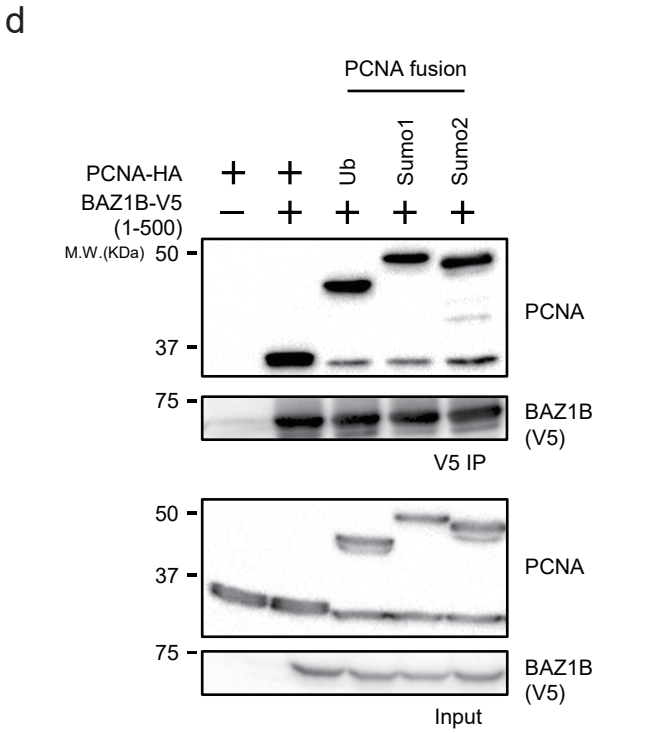
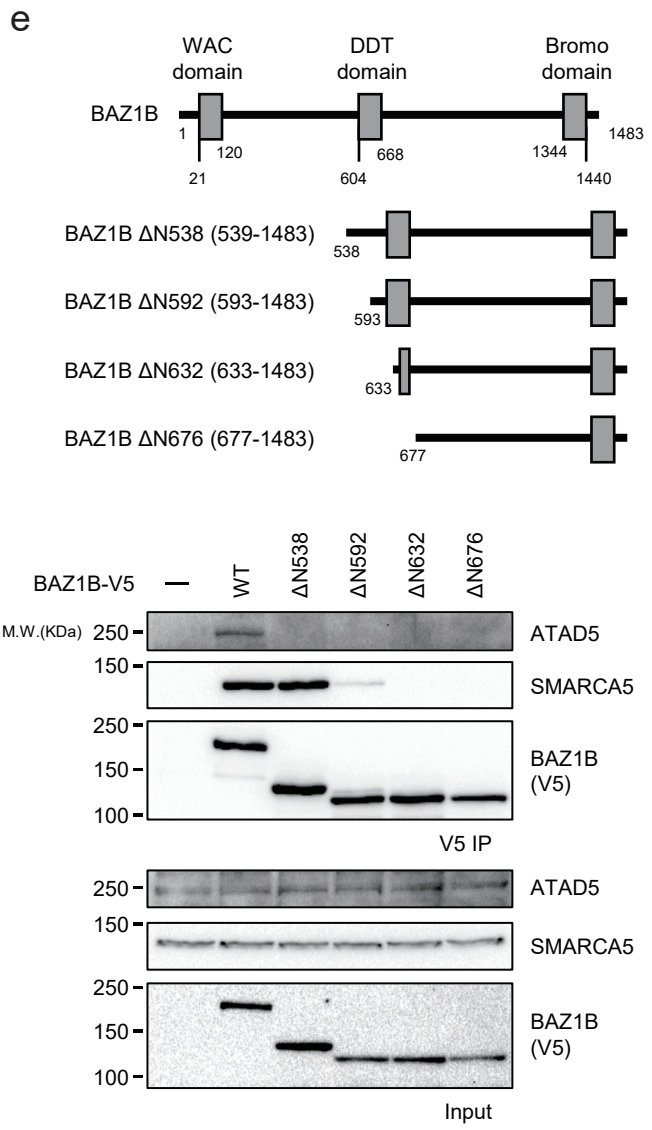
250
150
150
PCNA
ATAD5
SMARCA5
BAZ1B
(FLAG)
Input


d
PCNA fusion
PCNA-HA
BAZ1B-V5
(1-500)
Ub
Sumo1
Sumo2
M.W.(KDa)
50
37
75
PCNA
BAZ1B
(V5)
V5 IP
50
37
75
PCNA
BAZ1B
(V5)
Input


e
WAC
domain
DDT
domain
Bromo
domain
BAZ1B
1
120
21
668
604
1344
1440
1483
BAZ1B ΔN538 (539-1483)
538
BAZ1B ΔN592 (593-1483)
593
BAZ1B ΔN632 (633-1483)
633
BAZ1B ΔN676 (677-1483)
677
BAZ1B-V5
—
WT
ΔN538
ΔN592
ΔN632
ΔN676
M.W.(KDa)
250
150
250
150
100
ATAD5
SMARCA5
BAZ1B
(V5)
V5 IP
250
150
250
150
100
ATAD5
SMARCA5
BAZ1B
(V5)
Input

f

```
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BAZ1B_XENLA 512 MTEEQREEYMRKKREALKARIKEKTRERKQKE 543
                *:****:**::***: ** :::*:::**:::*

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                *:* *:.**:*:**********:******:*:**** **.**:**
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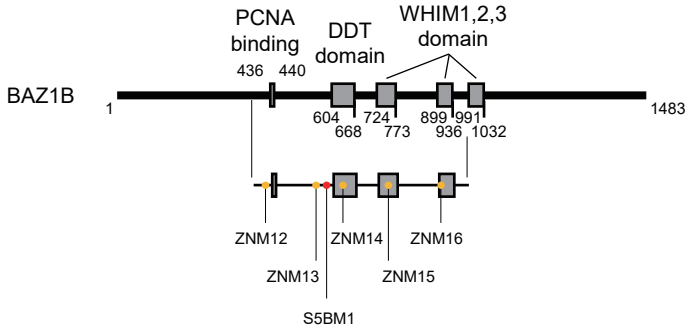

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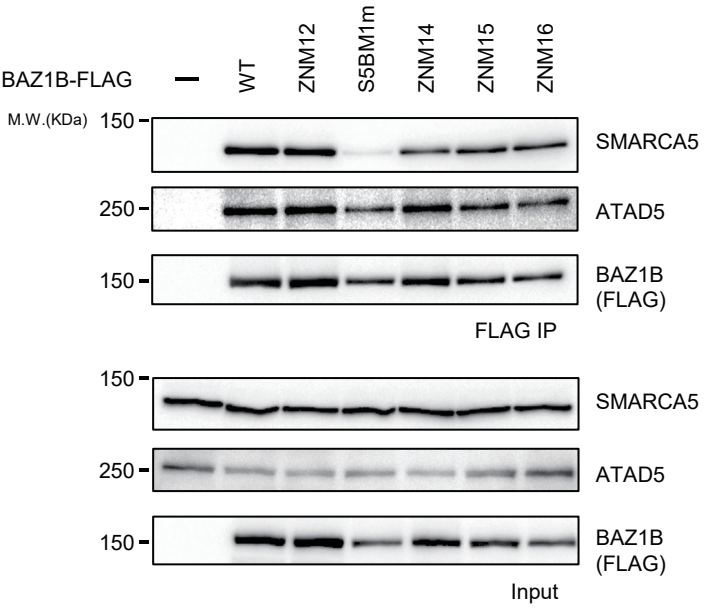

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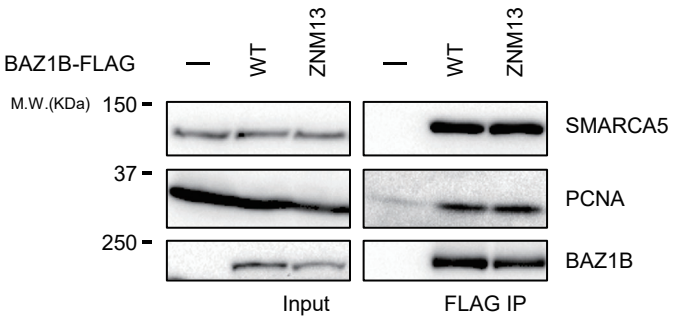

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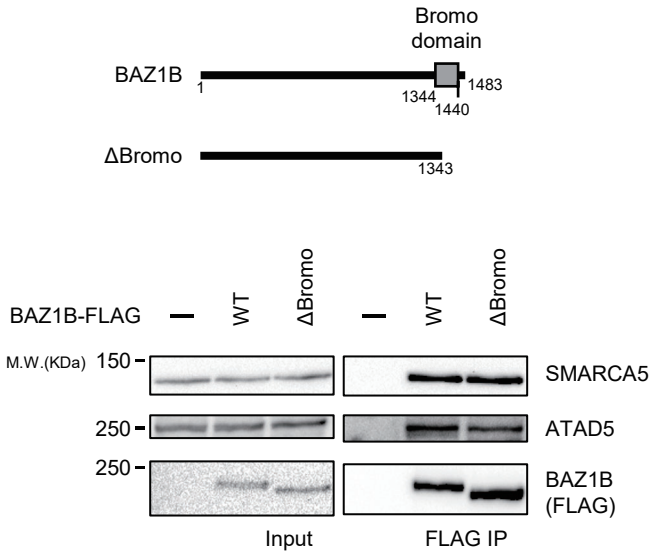

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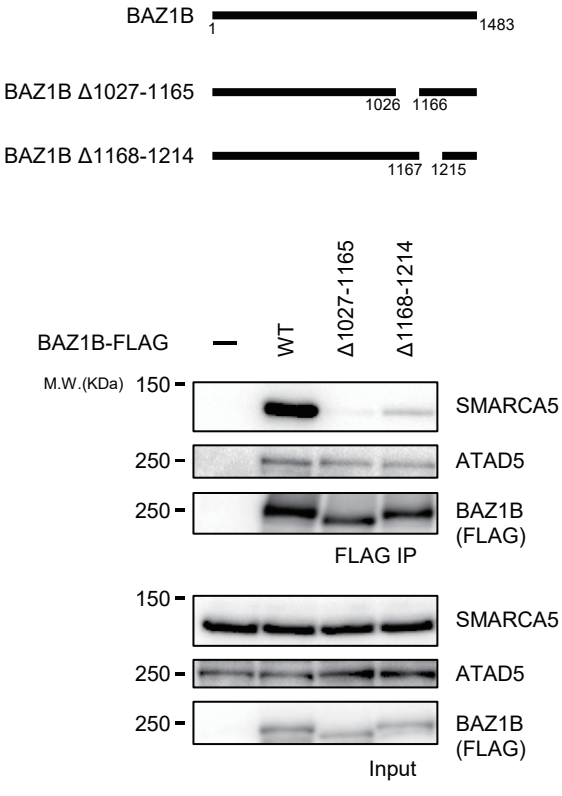

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                  ZNM17 1137AAMVAA1142    S5BM2m 1158AAAAAAA1164  ZNM19 1177AAA1179          ZNM20 1202AAAANKAFAAAA1213
BAZ1B_HUMAN 1128 AKTEEVDEEKKMVEEAKVASALEKWKTAIREAQTFSRMHVLLGMLDACIKWDMSAENARCKVCRKKGEDDKLILCDECNKAFHLFCLRPALYEVPD 1223
BAZ1B_MOUSE 1128 TKSEEVDEEKKMVEEAKVASALEKWKTAIREAQTFSRMHVLLGMLDACIKWDMSAENARCKVCRKKGEDDKLILCDECNKAFHLFCLRPALYEVPD 1223
BAZ1B_CHICK 1128 TKAEEVDEEKKMAEEAKVASAMEKWKTAIREAQTFSRMHVLLGMLDACIKWDMSAENARCKVCRKKGEDDKLILCDECNKAFHLFCLRPALYEIPD 1223
BAZ1B_XENLA 1095 AKAEEQDEEKKMAEEAKVASAVEKWKVAIRDAQTFSRMHVLLGMLDACIKWDMSSENARCKVCRKKGEDDKLILCDECNKAFHLFCLRPVLFNIPD 1190
                  :*:** ******.********:****.***:**********************:*********************************.*:::**
                                                S5BM2
```
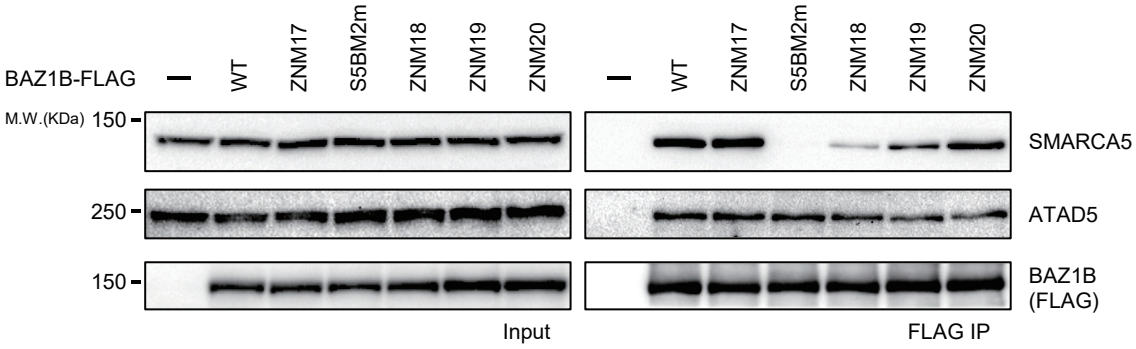

m



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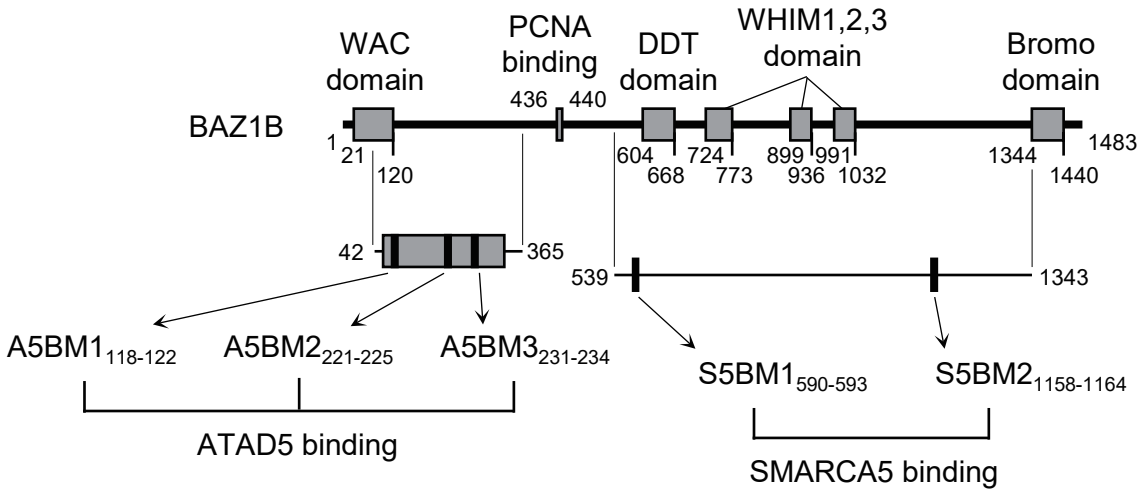
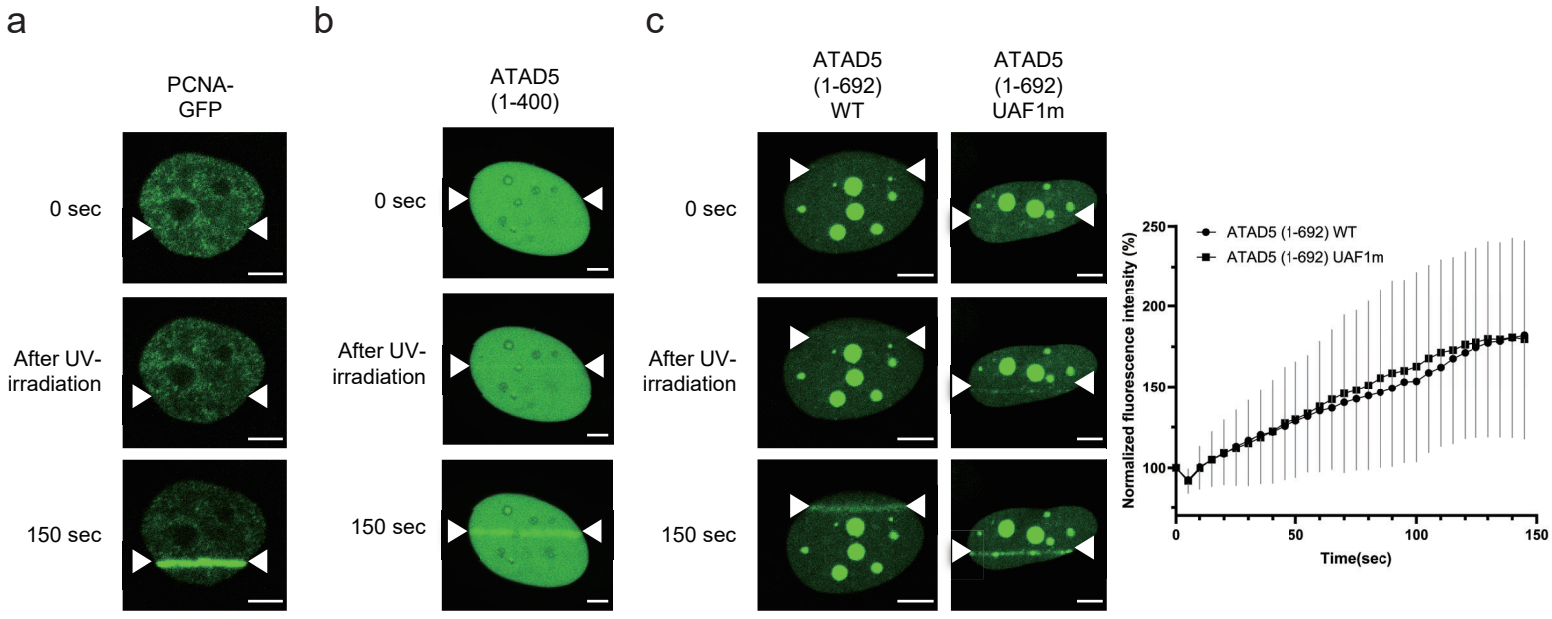


Supplementary Figure 4. BAZ1B interacts with PCNA and SMARCA5 through distinct motifs (related to Figure 3).

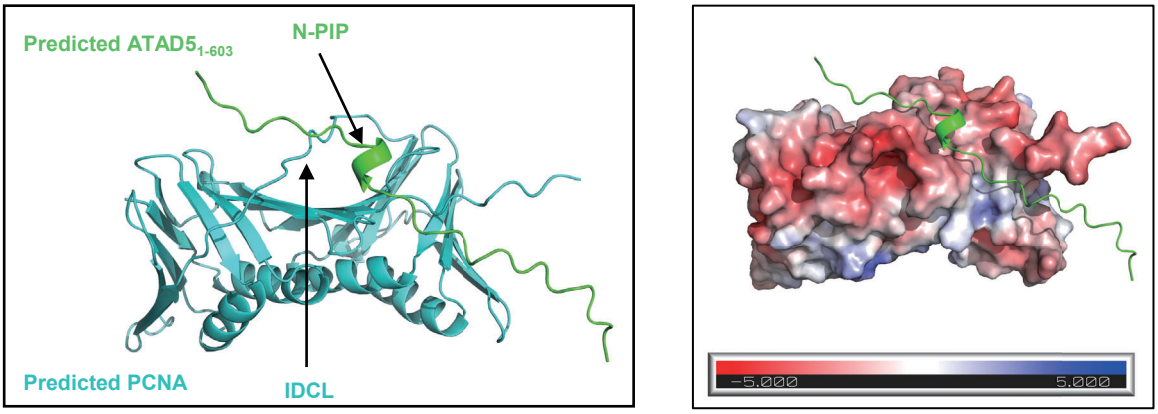
(a) The BAZ1B (1-500) is sufficient for PCNA interaction. V5-tagged wild-type and indicated BAZ1B fragments were expressed in 293T cells, followed by anti-V5 immunoprecipitation. **(b)** Locations of ZNM9-12 mutations and PIPm are indicated. **(c)** Other mutants in BAZ1B (323-466) do not significantly affect PCNA binding. Anti-FLAG-immunoprecipitation was performed with the indicated cells expressing wild-type or mutant FLAG-BAZ1B. **(d)** Ubiquitin or SUMO moieties do not affect the BAZ1B-PCNA interaction. HA-tagged SUMO- or Ubiquitin-fused PCNA and V5-tagged BAZ1B (1-500) were transiently co-expressed in 293T cells, followed by anti-V5 immunoprecipitation. **(e)** BAZ1B (539-592) is crucial for SMARCA5 binding. The indicated V5-tagged BAZ1B deletion mutants were transiently expressed in 293T cells, and anti-V5 immunoprecipitation was performed. **(f)** Positions of ZNM12-16 mutations and S5BM1m are indicated on the aligned sequences of the middle region of BAZ1B homologues. **(g)** Diagram showing the positions of ZNM12-16 mutations and S5BM1. Grey boxes indicate the domains of BAZ1B. **(h-i)** The mutations in BAZ1B S5BM1 (^{590}LPAF^{593} to AAAA) eliminate the BAZ1B-SMARCA5 interaction. The indicated Clip-BAZ1B-StrepII-FLAG variants were transiently expressed, and FLAG-immunoprecipitation was performed. **(j)** The bromodomain of BAZ1B is not necessary for SMARCA5 binding. FLAG-tagged BAZ1B wild-type or bromodomain deletion mutant was transiently expressed in 293T cells, followed by FLAG-immunoprecipitation. **(k)** The residues 1027-1214 of BAZ1B are essential for SMARCA5 binding. The indicated Clip-BAZ1B-StrepII-FLAG variants were transiently expressed, and FLAG-immunoprecipitation was performed. **(l)** Positions of ZNM17-20 mutations and S5BM2m are indicated on the aligned sequences of the C-terminal domain of BAZ1B homologues. The indicated Clip-BAZ1B-StrepII-FLAG variants were transiently expressed, and FLAG-immunoprecipitation was performed. **(m)** The mutations in S5BM2 (^{1158}EAQTFSR^{1164} to AAAAAAA) abolishes SMARCA5 binding. The indicated Clip-BAZ1B-StrepII-FLAG variants were transiently expressed, and FLAG-immunoprecipitation was performed. **(n)** Diagram illustrating the positions of ATAD5, PCNA, and SMARCA5 binding motifs in BAZ1B. Source data are provided as a Source Data file.



d

```
                                                N-PIPm  62ALDAA66
                    NTM  30AAAGAAGKAAAAALSPLGKTRDRVFAAKPSAAGAAAGAA69
ATAD5_HUMAN 21 IEPCKKRKKDDDTSTCKTITKYLSPLGKTRDRVFAPPKPSNILDYFRKTSPTNEKTQLGK 80
ATAD5_MOUSE 21 IEPCKKRRKDDDNSSCETITKYLSPIGKTGDKVFSPPKPSNILHYFRKTSLTTEKPQSTK 80
ATAD5_CHICK 21 AQPRKKRREED--ASVKTITRYFSPLAKAGGKVSSPPKANNILDYFKKTPAAEKAPAIAG 78
ATAD5_XENLA 16 CQPCKKSRKDE-EAPIKTITNYFSPVSKNTEKVLSSPRSNNIADYFKQNSPINEKKQTSK 74
               :* ** ::::  :  :***.*:**:.*   :* : *: .** .**::.    :
                                                N-PIP
```

e



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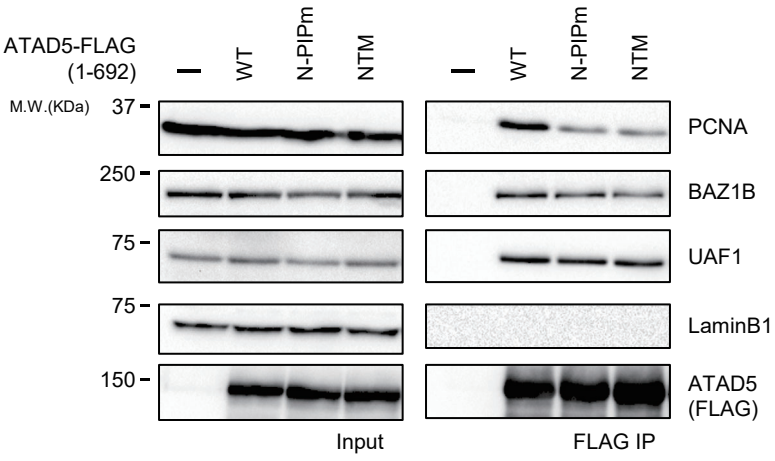
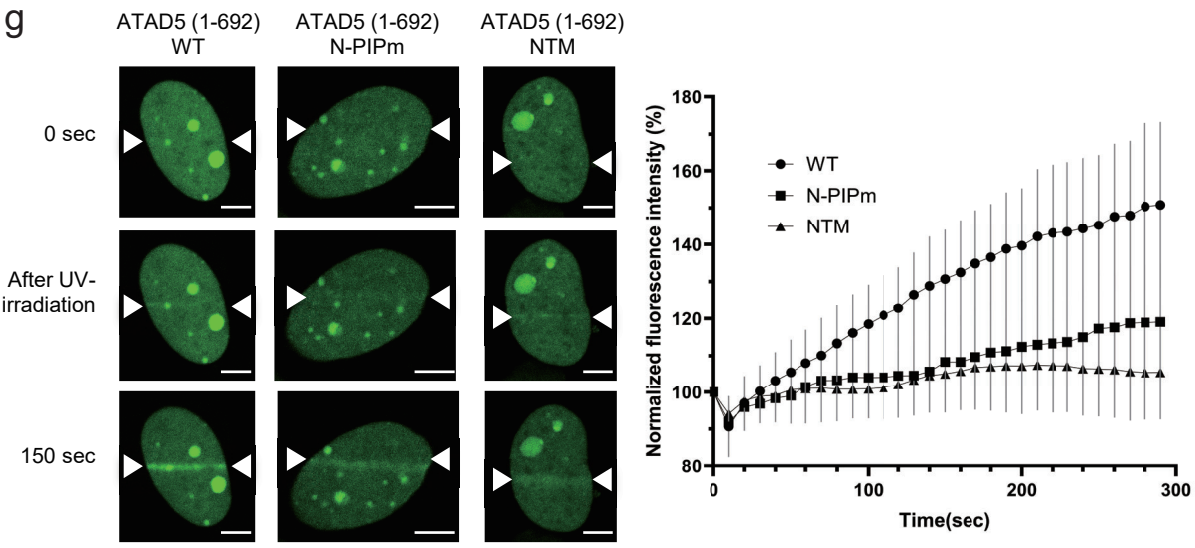
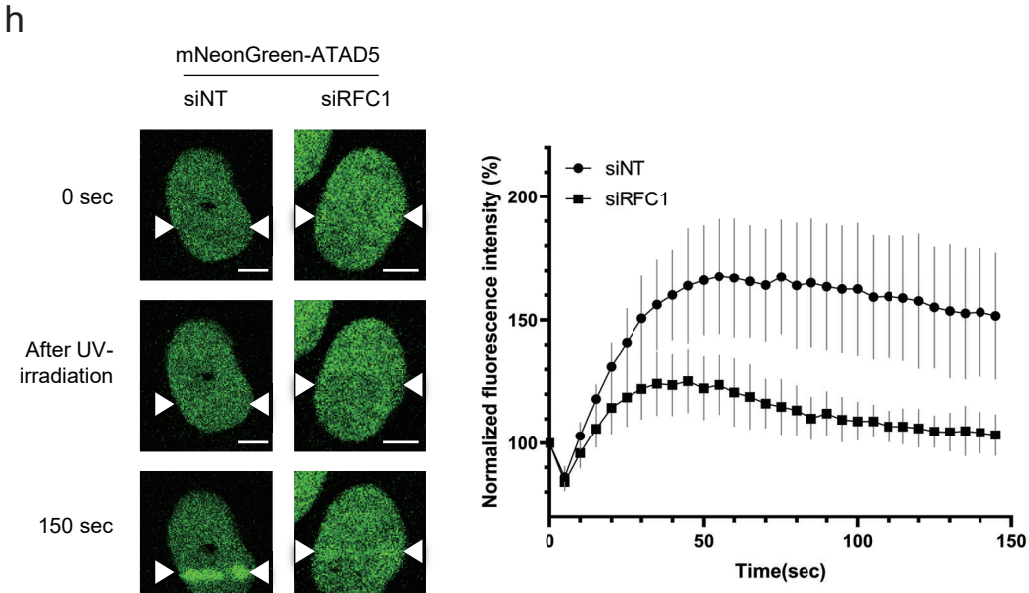



g
ATAD5 (1-692) WT
ATAD5 (1-692) N-PIPm
ATAD5 (1-692) NTM
0 sec
After UV-irradiation
150 sec
Normalized fluorescence intensity (%)
Time(sec)
WT
N-PIPm
NTM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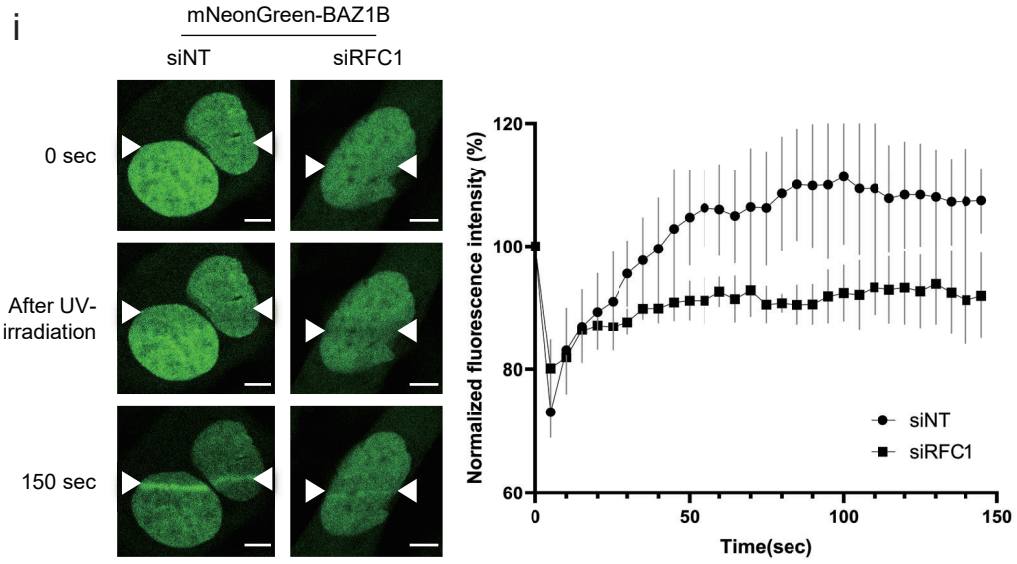
siNT
siRFC1
0 sec
After UV-irradiation
150 sec
Normalized fluorescence intensity (%)
Time(sec)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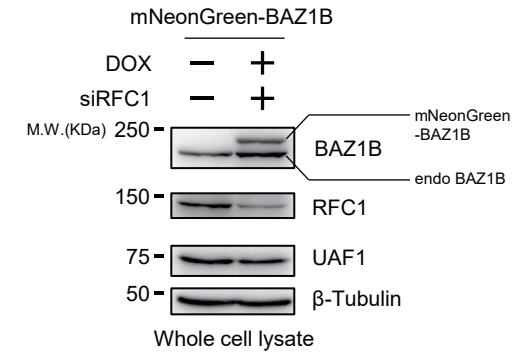
siNT
siRFC1
0 sec
After UV-irradiation
150 sec
Normalized fluorescence intensity (%)
Time(sec)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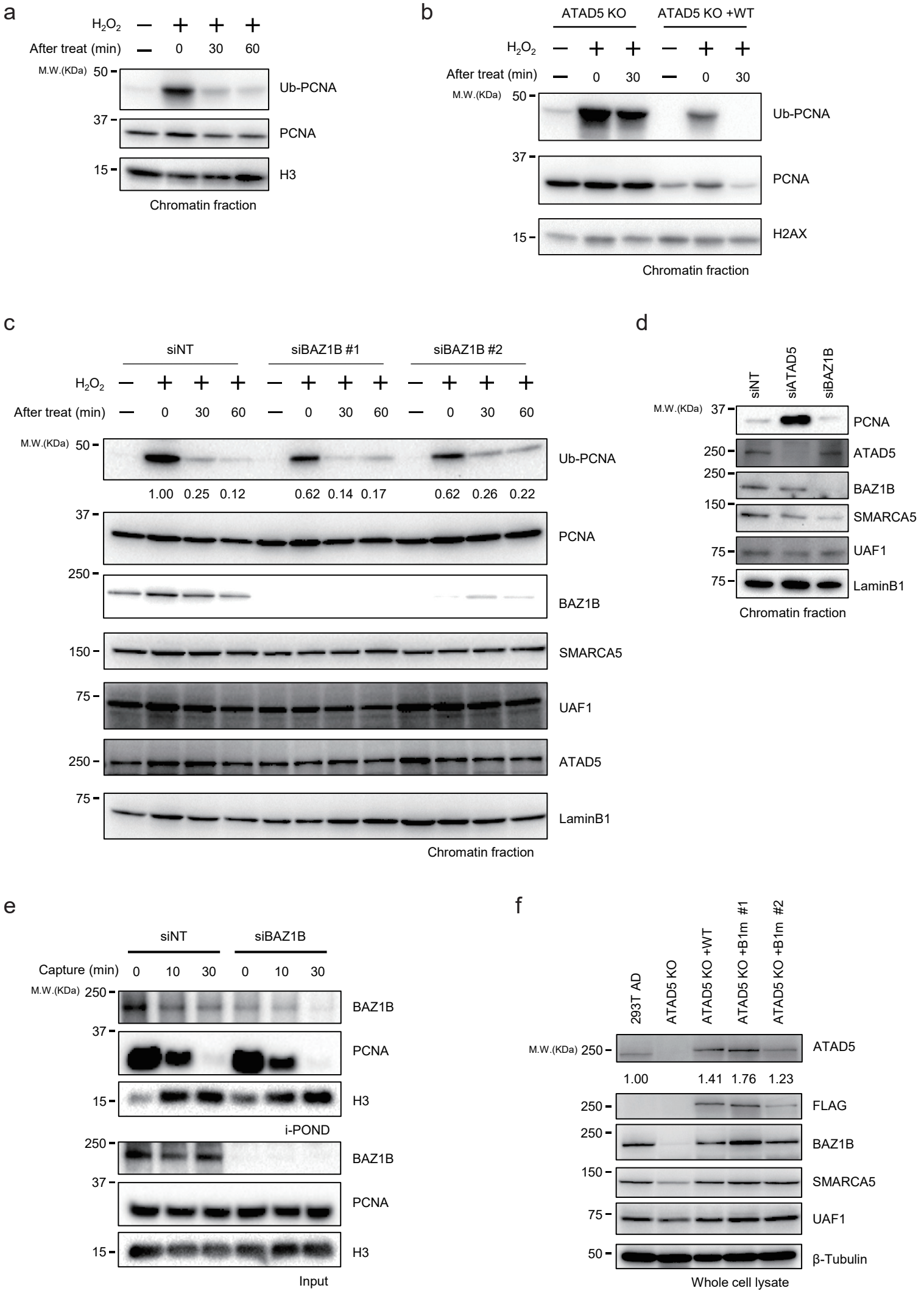
DOX
siRFC1
M.W.(KDa)
250
150
75
50
BAZ1B
mNeonGreen -BAZ1B
endo BAZ1B
RFC1
UAF1
β-Tubulin
Whole cell lysate

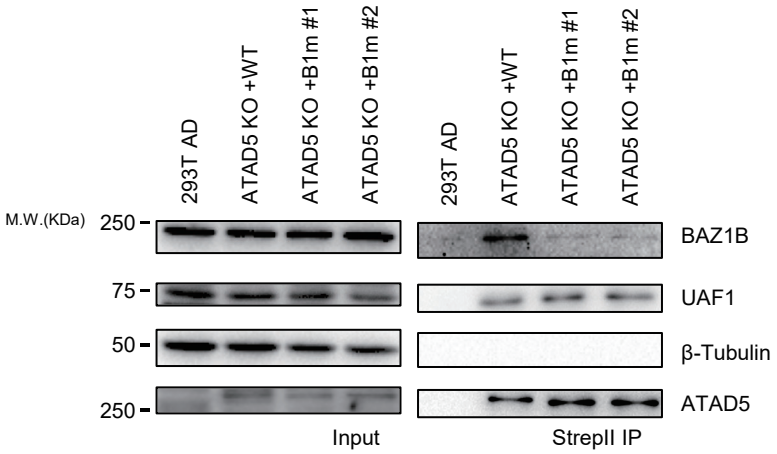
Supplementary Figure 5. ATAD5 N-terminal domain localizes to DNA damage sites (related to Figure 4).

(a) PCNA localizes to micro-irradiated sites. HeLa cells stably expressing GFP-PCNA were subjected to 355 nm UV laser micro-irradiation to the area indicated by the white arrowheads. **(b)** ATAD5 (1-400) localizes to DNA damage sites. mNeonGreen-tagged ATAD5 (1-400) was transiently expressed and then subjected to 355nm UV laser micro-irradiation to the area indicated by the white arrowheads. **(c)** UAF1-binding is not essential for the localization of ATAD5 at damage sites. Wild-type or UAF1-binding defective mNeonGreen-tagged ATAD5 (1-693) were transiently expressed in U2OS cells and subjected to 355nm UV laser micro-irradiation to the area indicated by the white arrowheads. The mean intensity of biological replicates (n=7 for wild-type and n=7 for UAF1m) are depicted as points, while the bands represent the standard deviation of all data points. **(d)** The ATAD5 ^{62}IxxYF^{66} motif is conserved among species. The locations of NTM and N-PIPm mutations are indicated. **(e)** Structural prediction of PCNA-bound ATAD5 N-terminal region using AlphaFold2 *(Left)*. The electrostatic surface of PCNA was analyzed using the APBS plugin (*Right*). The ATAD5 N-terminal domain is predicted to contact with the PCNA IDCL. The PCNA-interacting region of ATAD5 N-terminal domain is positioned within the binding pocket formed by PCNA IDCL. **(f)** ATAD5 interacts with PCNA through the N-PIP motif (residues 62-66). ATAD5-FLAG (1-692) wild-type or mutants were transiently expressed in 293T cells and immunoprecipitated using anti-FLAG beads. **(g)** PCNA interaction is crucial for the localization of ATAD5 to micro-irradiated sites. ATAD5-FLAG-mNeonGreen (1-692) wild-type or the indicated mutants were transiently expressed in U2OS cells and then subjected to 355 nm UV laser micro-irradiation to the area indicated by the white arrowheads. The mean intensity of biological replicates (n=8 for wild-type, n=10 for N-PIPm and n=10 for NTM) are depicted as points, while the bands represent the standard deviation of all data points. **(h-i)** The localization of ATAD5 and BAZ1B to damage sites depends on PCNA. HeLa cells with endogenous ATAD5 fused to mNeonGreen following RFC1 depletion, ***(h)***, or stably expressing mNeonGreen-BAZ1B following RFC1 depletion, ***(i)***, were subjected to 355 nm UV laser micro-irradiation to the area indicated by the white arrowheads. The mean intensity of biological replicates (n=8 for siNT and n=10 for siRFC1, ***(h)***, or n=6 for siNT and n=6 for siRFC1, ***(i)***) are depicted as points, while the bands represent the

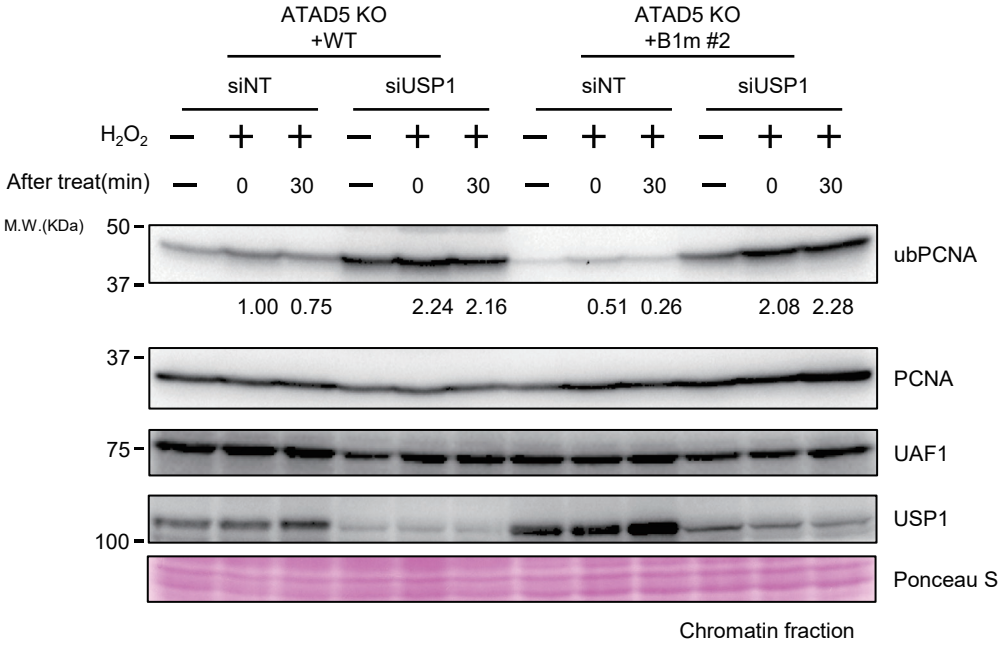
standard deviation of all data points. **(j)** The expression level of mNeonGreen-BAZ1B is comparable to that of endogenous BAZ1B. Whole cell lysates were prepared for immunoblotting. The scale bar in representative micrographs of **(a-c, g-i)** is 5 µm. Source data are provided as a Source Data file.


a
H_2O_2 – + + +
After treat (min) – 0 30 60
M.W.(KDa)
50
Ub-PCNA
37
PCNA
15
H3
Chromatin fraction
b
ATAD5 KO
ATAD5 KO +WT
H_2O_2 – + + – + +
After treat (min) – 0 30 – 0 30
M.W.(KDa)
50
Ub-PCNA
37
PCNA
15
H2AX
Chromatin fraction
c
siNT
siBAZ1B #1
siBAZ1B #2
H_2O_2 – + + + – + + + – + + +
After treat (min) – 0 30 60 – 0 30 60 – 0 30 60
M.W.(KDa)
50
Ub-PCNA
1.00 0.25 0.12 0.62 0.14 0.17 0.62 0.26 0.22
37
PCNA
250
BAZ1B
150
SMARCA5
75
UAF1
250
ATAD5
75
LaminB1
Chromatin fraction
d
siNT
siATAD5
siBAZ1B
M.W.(KDa)
37
PCNA
250
ATAD5
250
BAZ1B
150
SMARCA5
75
UAF1
75
LaminB1
Chromatin fraction
e
siNT
siBAZ1B
Capture (min) 0 10 30 0 10 30
M.W.(KDa)
250
BAZ1B
37
PCNA
15
H3
i-POND
250
BAZ1B
37
PCNA
15
H3
Input
f
293T AD
ATAD5 KO
ATAD5 KO +WT
ATAD5 KO +B1m #1
ATAD5 KO +B1m #2
M.W.(KDa)
250
ATAD5
1.00 1.41 1.76 1.23
250
FLAG
250
BAZ1B
150
SMARCA5
75
UAF1
50
β-Tubulin
Whole cell lysate

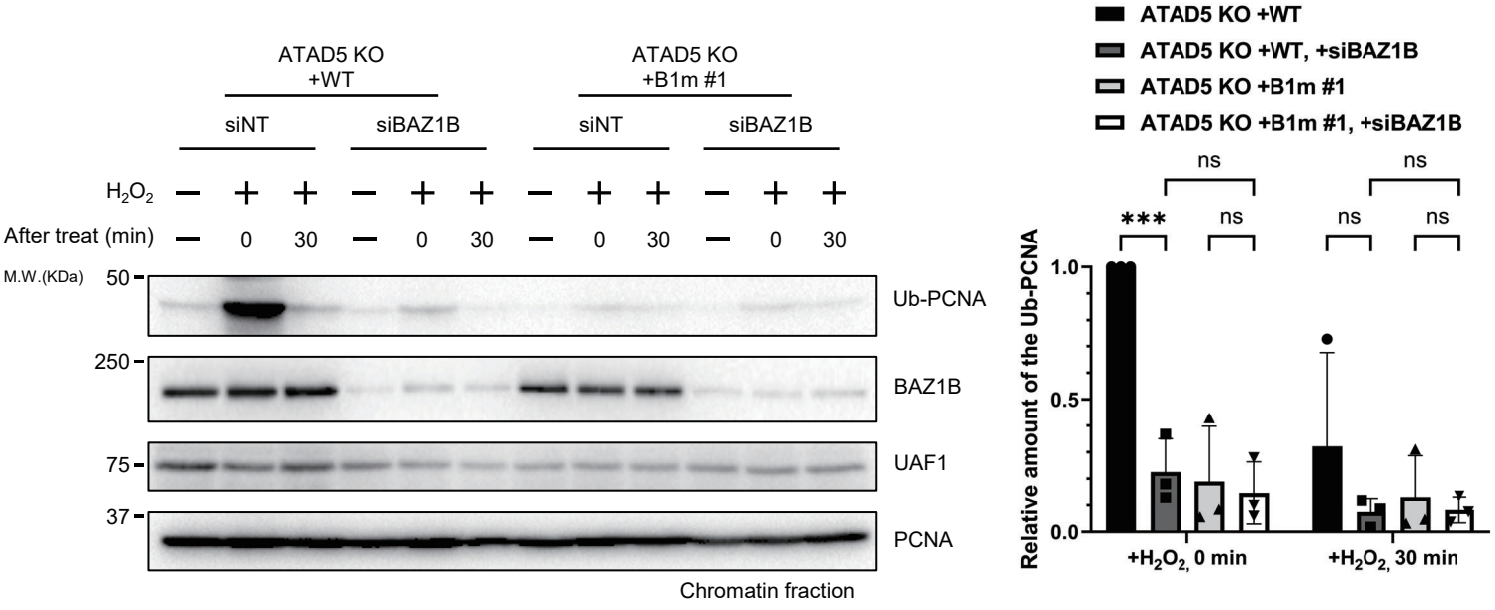
g


293T AD
ATAD5 KO +WT
ATAD5 KO +B1m #1
ATAD5 KO +B1m #2
293T AD
ATAD5 KO +WT
ATAD5 KO +B1m #1
ATAD5 KO +B1m #2
M.W.(KDa)
250
75
50
250
BAZ1B
UAF1
β-Tubulin
ATAD5
Input
StrepII IP

h


ATAD5 KO +WT
ATAD5 KO +B1m #2
siNT
siUSP1
siNT
siUSP1
H2O2
After treat(min)
0
30
M.W.(KDa)
50
37
ubPCNA
1.00 0.75 2.24 2.16 0.51 0.26 2.08 2.28
37
PCNA
75
UAF1
100
USP1
Ponceau S
Chromatin fraction

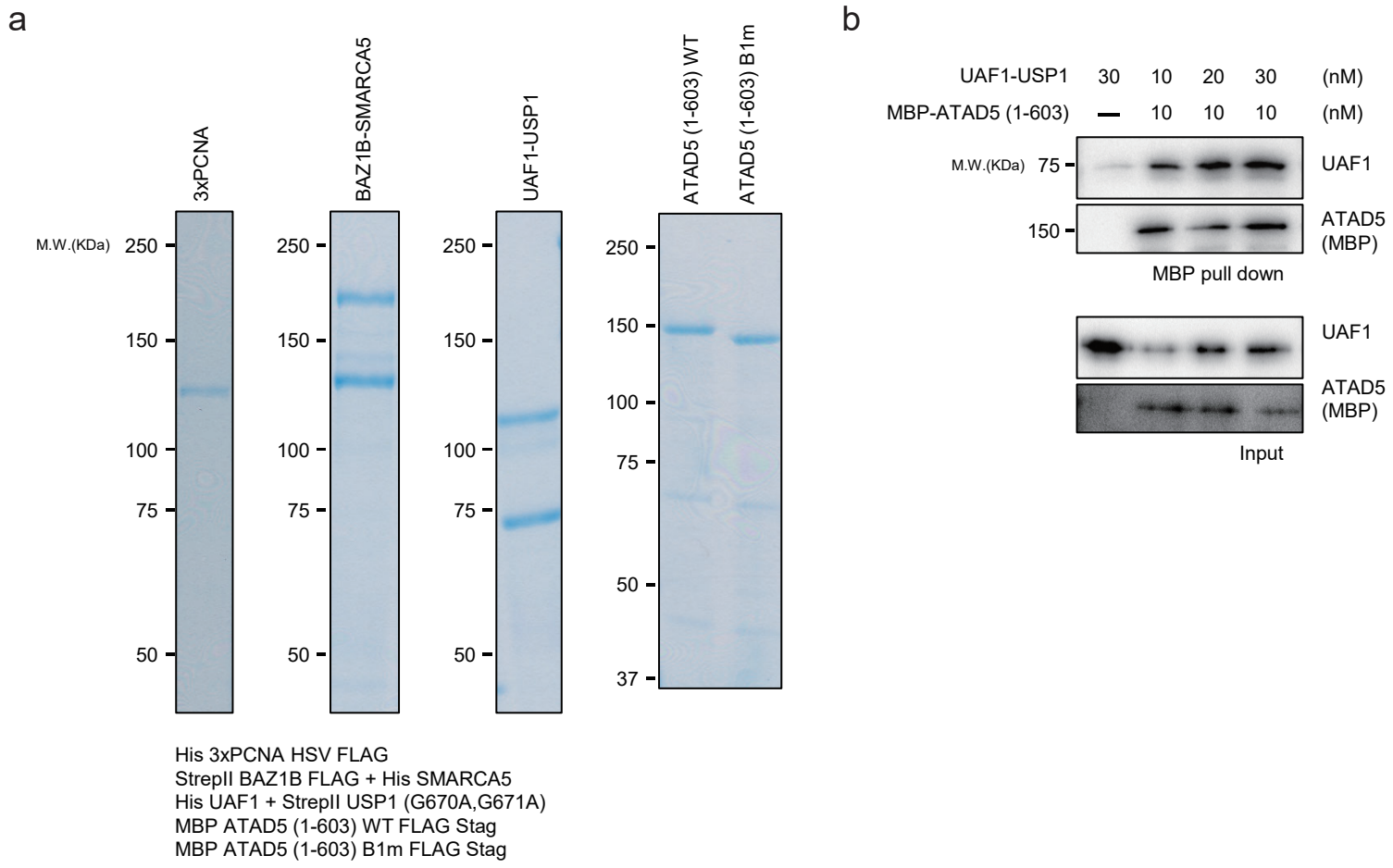
i

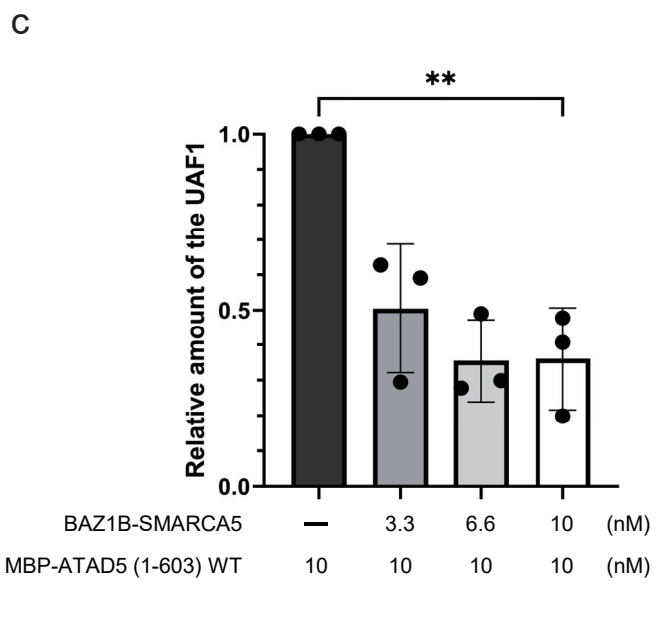

ATAD5 KO +WT
ATAD5 KO +B1m #1
siNT
siBAZ1B
siNT
siBAZ1B
H2O2
After treat (min)
0
30
M.W.(KDa)
50
Ub-PCNA
250
BAZ1B
75
UAF1
37
PCNA
Chromatin fraction
ATAD5 KO +WT
ATAD5 KO +WT, +siBAZ1B
ATAD5 KO +B1m #1
ATAD5 KO +B1m #1, +siBAZ1B
ns
ns
***
ns
ns
ns
1.0
0.5
0.0
Relative amount of the Ub-PCNA
+H2O2, 0 min
+H2O2, 30 min

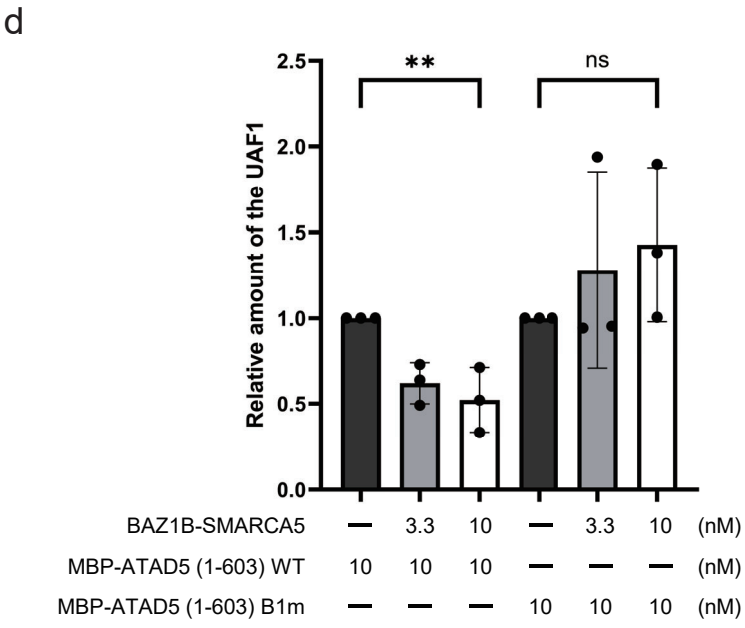
Supplementary Figure 6. The ATAD5-BAZ1B interaction regulates Ub-PCNA de-ubiquitination (related to Figure 5).

(a) Ub-PCNA are rapidly generated and de-ubiquitinated during recovery from oxidative stress. 293T cells were treated with 1 mM of H_2O_2 for 20 minutes and then incubated in fresh media for the indicated times. Cells were harvested at specified time points, and chromatin fractions were prepared. **(b)** ATAD5 deficiency increases Ub-PCNA levels on the chromatin following hydrogen peroxide treatment. ATAD5 knock-out (ATAD5 KO) cells and ATAD5 KO cells stably expressing wild-type ATAD5 (ATAD5 KO+WT) were treated with 1 mM H_2O_2. **(c)** BAZ1B depletion reduces the Ub-PCNA levels after H_2O_2 treatment. Two independent BAZ1B-targeting siRNAs were used for BAZ1B depletion. Cells were treated with 1 mM H_2O_2 for 20 minutes and then incubated in fresh media for the indicated time. Chromatin fractions were prepared and analyzed by immunoblotting. Numbers below the Ub-PCNA blot indicate the relative amount of Ub-PCNA. **(d)** BAZ1B depletion does not affect PCNA unloading. 293T cells were transfected with the indicated siRNAs for 48 hours, and chromatin fractions were isolated. **(e)** Depletion of BAZ1B does not affect PCNA levels on nascent DNA. Nascent DNA-associated proteins were examined by i-POND analysis in BAZ1B-depleted cells. Cells were harvested at the indicated time points after an EdU pulse-chase procedure, and nascent DNA-bound proteins were isolated following the Click reaction. **(f)** The expression levels of ATAD5 in ATAD5 add-back clones are comparable to endogenous ATAD5. Numbers below the ATAD5 blot indicate the relative amount of ATAD5. **(g)** The ATAD5 B1m mutation disrupts BAZ1B binding. Wild-type ATAD5 or ATAD5 B1m was re-expressed in ATAD5 knock-out cells using lentiviral expression system. After isolating single clones, Strep-Tactin pull-down was performed, and ATAD5-bound proteins were analyzed by immunoblotting. **(h)** BAZ1B regulates Ub-PCNA de-ubiquitination by USP1. The experiments shown in Figure 5C were conducted using the ATAD5 B1m #2 clone. Numbers below the Ub-PCNA blot indicate the relative amount of Ub-PCNA. **(i)** Ub-PCNA levels after oxidative stress are regulated by the ATAD5-BAZ1B interaction. BAZ1B was depleted in ATAD5 KO+WT and ATAD5 KO+B1m #1 cells, which were treated with 0.3 mM H_2O_2 for 20 minutes. Following treatment, cells were incubated in fresh media for the indicated times and chromatin fractionation was performed. The right panel shows the quantified relative amount of Ub-PCNA. Error bars represent the standard deviation of the mean from

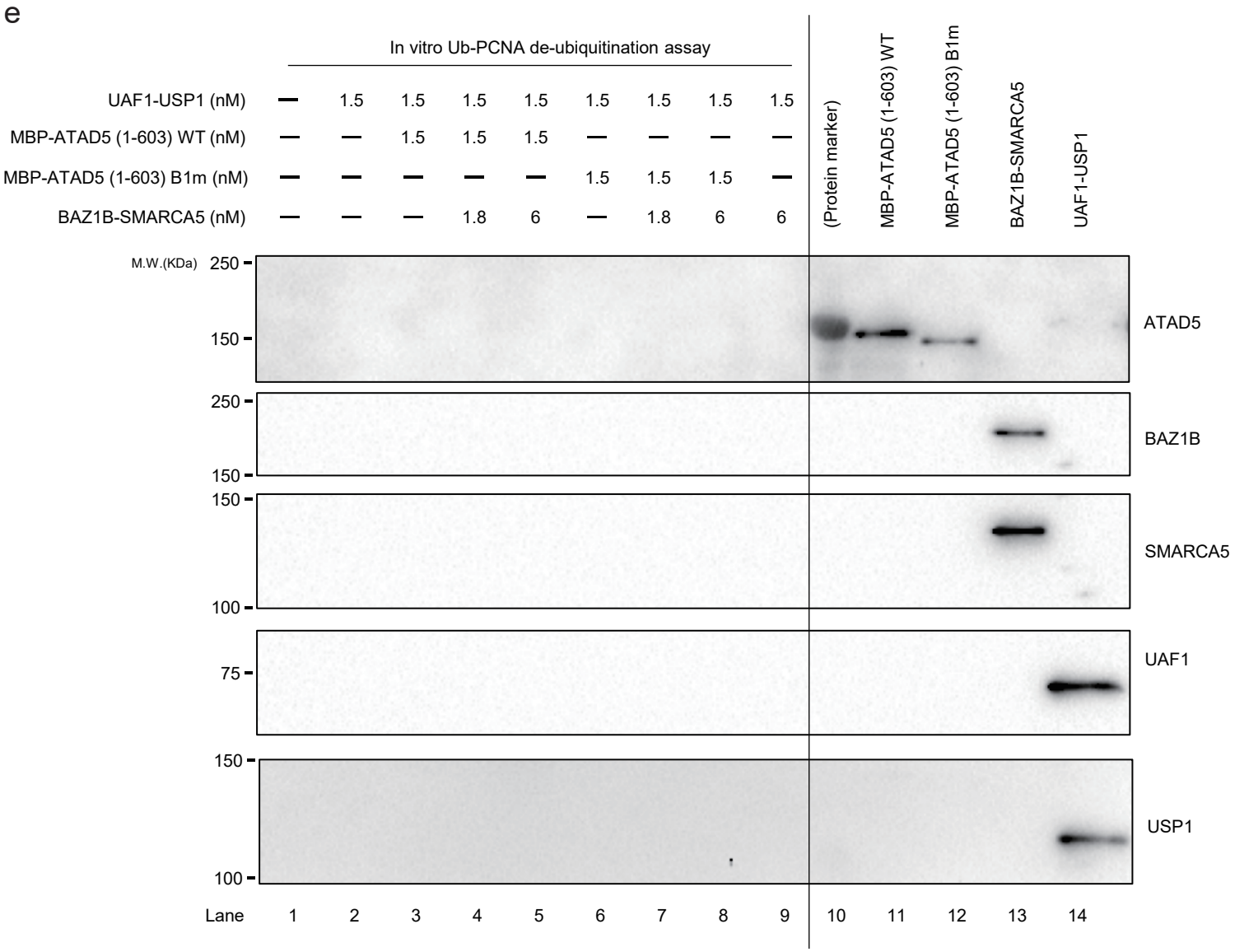
three independent replicates. Statistical analysis: Two-way ANOVA; ***P≤0.001 and ns: not significant. Source data are provided as a Source Data file.


a
3xPCNA
BAZ1B-SMARCA5
UAF1-USP1
ATAD5 (1-603) WT
ATAD5 (1-603) B1m
M.W.(KDa)
250
150
100
75
50
37
His 3xPCNA HSV FLAG
StrepII BAZ1B FLAG + His SMARCA5
His UAF1 + StrepII USP1 (G670A,G671A)
MBP ATAD5 (1-603) WT FLAG Stag
MBP ATAD5 (1-603) B1m FLAG Stag
b
UAF1-USP1 30 10 20 30 (nM)
MBP-ATAD5 (1-603) — 10 10 10 (nM)
M.W.(KDa)
75
150
UAF1
ATAD5 (MBP)
MBP pull down
Input


c
**
Relative amount of the UAF1
1.0
0.5
0.0
BAZ1B-SMARCA5 — 3.3 6.6 10 (nM)
MBP-ATAD5 (1-603) WT 10 10 10 10 (nM)


d
**
ns
Relative amount of the UAF1
2.5
2.0
1.5
1.0
0.5
0.0
BAZ1B-SMARCA5 — 3.3 10 — 3.3 10 (nM)
MBP-ATAD5 (1-603) WT 10 10 10 — — — (nM)
MBP-ATAD5 (1-603) B1m — — — 10 10 10 (nM)

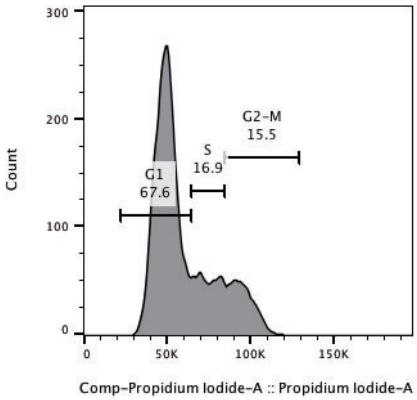
e


In vitro Ub-PCNA de-ubiquitination assay
UAF1-USP1 (nM)
MBP-ATAD5 (1-603) WT (nM)
MBP-ATAD5 (1-603) B1m (nM)
BAZ1B-SMARCA5 (nM)
— 1.5 1.5 1.5 1.5 1.5 1.5 1.5 1.5
— — 1.5 1.5 1.5 — — — —
— — — — — 1.5 1.5 1.5 —
— — — 1.8 6 — 1.8 6 6
(Protein marker)
MBP-ATAD5 (1-603) WT
MBP-ATAD5 (1-603) B1m
BAZ1B-SMARCA5
UAF1-USP1
M.W.(KDa)
250
150
ATAD5
250
150
BAZ1B
150
100
SMARCA5
75
UAF1
150
100
USP1
Lane 1 2 3 4 5 6 7 8 9 10 11 12 13 14

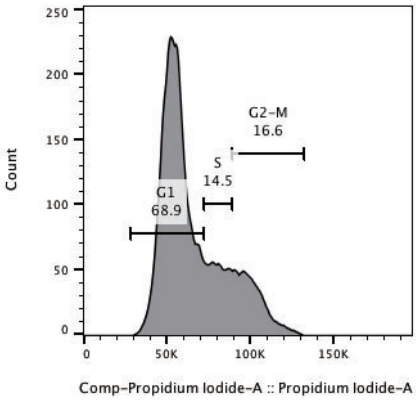
Supplementary Figure 7. ATAD5 and BAZ1B interfere with Ub-PCNA de-ubiquitination activity in vitro (related to Figure 6).

(a) Coomassie-stained SDS-PAGE of purified 3xPCNA, BAZ1B-SMARCA5, UAF1-USP1, and wild-type and B1m ATAD5 (1-603). **(b)** ATAD5 (1-603) interacts with UAF1-USP1 in vitro. MBP-tagged ATAD5 (1-603) was mixed with UAF1-USP1 and incubated with amylose resin for pull-down. Isolated proteins were analyzed by immunoblotting. **(c-d)** Quantified relative amounts of the UAF1 in Fig. 6b or Fig. 6c, respectively. Error bars represent the standard deviation of the mean from three independent replicates. Statistical analysis: Ordinary one-way ANOVA; **P≤0.001 and ns: not significant. **(e)** The UAF1-USP1 complex, ATAD5 (1-603), and the BAZ1B-SMARCA5 complex were not detected in DNA-associated fraction after the *in vitro* Ub-PCNA deubiquitination assay. Lanes 1-9 correspond to the same samples shown in Fig. 6d. Lane 10 contains the protein marker, and Lanes 11-14 serve as positive controls. Source data are provided as a Source Data file.

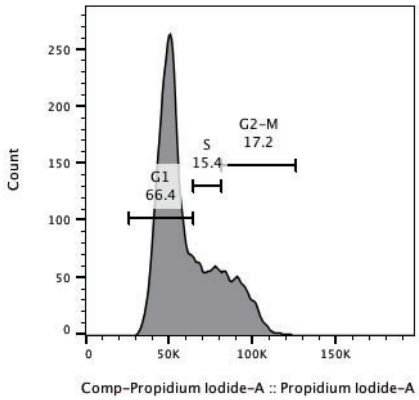
a



ATAD5 KO +WT

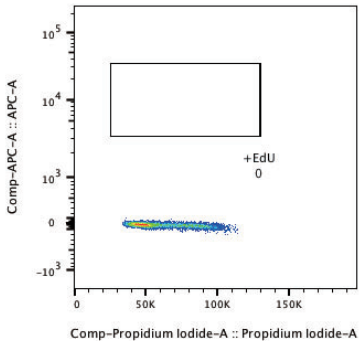


ATAD5 KO +B1m #1

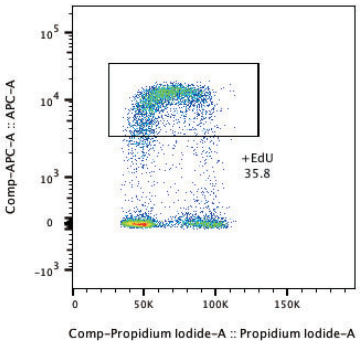


ATAD5 KO +B1m #2

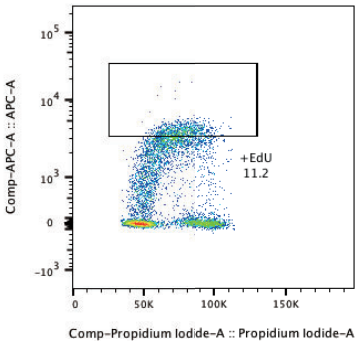
b



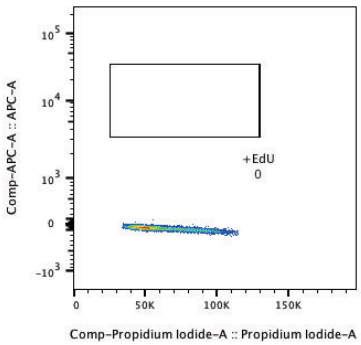
-EdU
ATAD5 KO +WT



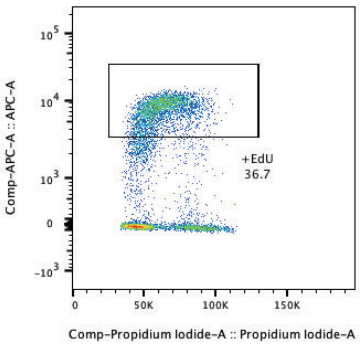
+EdU
ATAD5 KO +WT



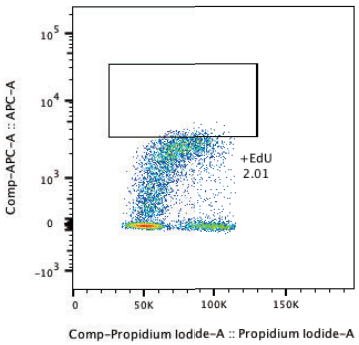
+EdU
+H_2O_2
ATAD5 KO +WT



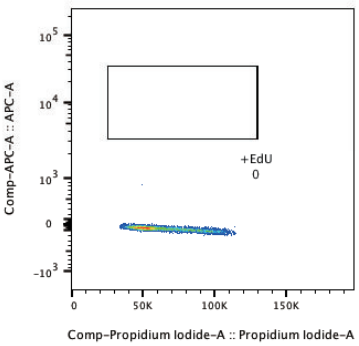
-EdU
ATAD5 KO +B1m #1



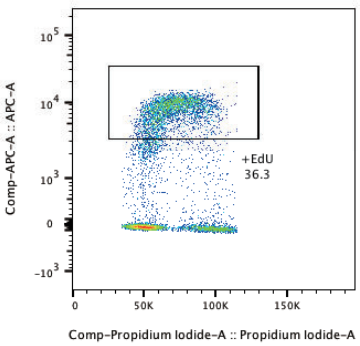
+EdU
ATAD5 KO +B1m #1



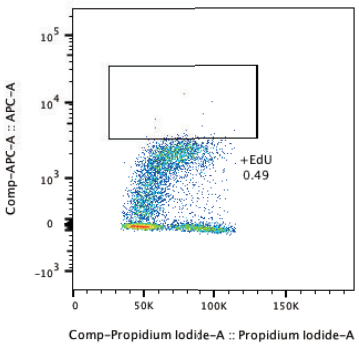
+EdU
+H_2O_2
ATAD5 KO +B1m #1



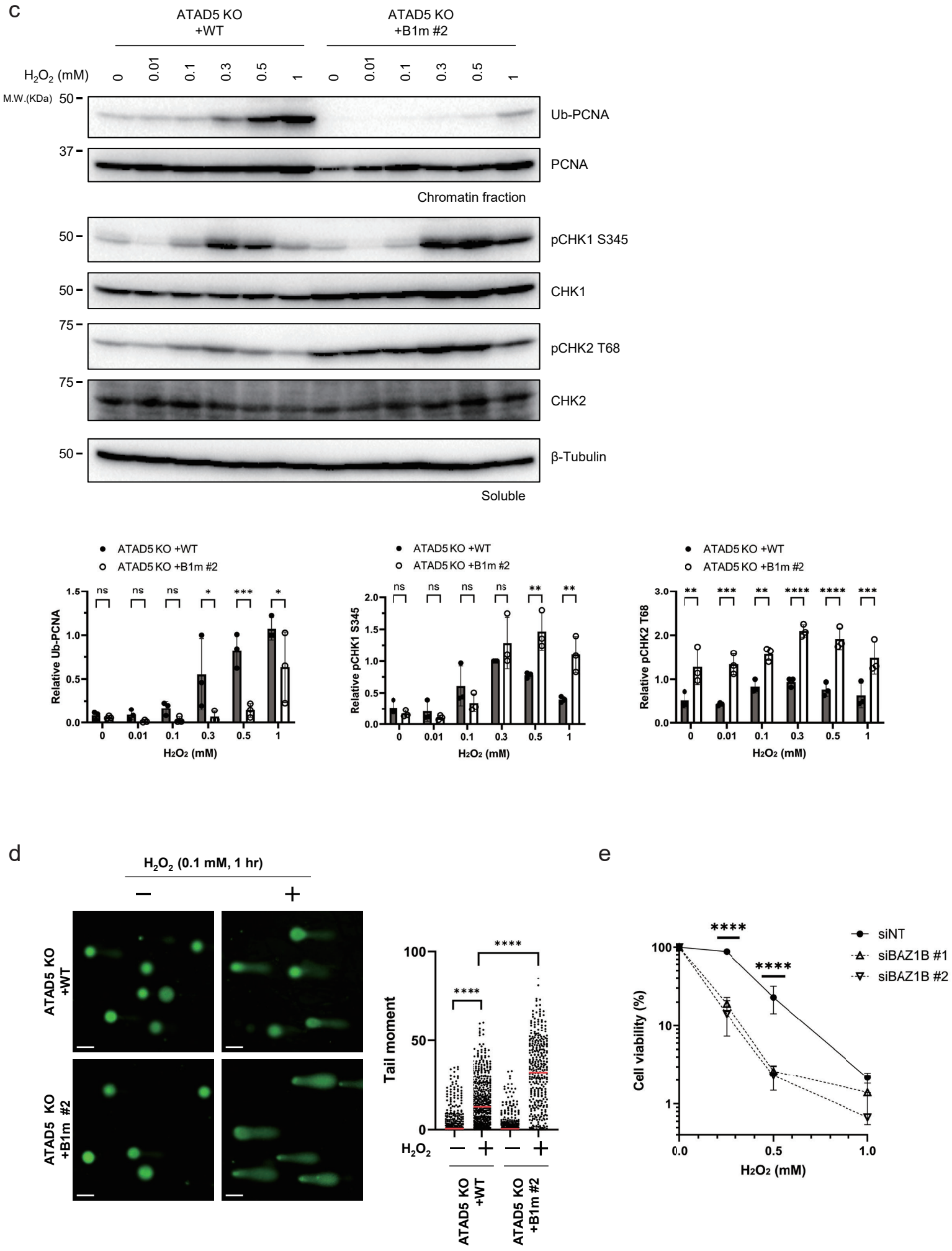
-EdU
ATAD5 KO +B1m #2



+EdU
ATAD5 KO +B1m #2



+EdU
+H_2O_2
ATAD5 KO +B1m #2


c
ATAD5 KO +WT
ATAD5 KO +B1m #2
H2O2 (mM)
0
0.01
0.1
0.3
0.5
1
M.W.(KDa)
50
37
Ub-PCNA
PCNA
Chromatin fraction
pCHK1 S345
CHK1
pCHK2 T68
CHK2
β-Tubulin
Soluble
ATAD5 KO +WT
ATAD5 KO +B1m #2
Relative Ub-PCNA
Relative pCHK1 S345
Relative pCHK2 T68
H2O2 (mM)
ns
*
**
***
****
d
H2O2 (0.1 mM, 1 hr)
ATAD5 KO +WT
ATAD5 KO +B1m #2
Tail moment
e
siNT
siBAZ1B #1
siBAZ1B #2
Cell viability (%)
H2O2 (mM)

Supplementary Figure 8. ATAD5-BAZ1B interaction is important for maintaining genomic integrity (related to Figure 7).

(a) Cell cycle progression is not significantly altered by the abrogation of ATAD5-BAZ1B interaction. The cell cycle profile was analyzed based on the intensity of propidium iodide (PI). The proportion of each cell cycle phase is indicated in the graphs. **(b)** The representative EdU incorporation profile as quantified in Fig. 7a. The proportion of EdU-positive cells within the highlighted rectangle in each plot is indicated. **(c)** ATAD5 KO+WT and ATAD5 KO+B1m #2 cells were fractionated after treatment with the indicated concentrations of hydrogen peroxide for 20 minutes. The bottom panel shows the quantified relative amounts of Ub-PCNA, phosphorylated CHK1 (S345), and phosphorylated CHK2 (T68). Error bars represent the standard deviation of the mean from three independent replicates. Statistical analysis: Two-way ANOVA; ****P≤0.0001, ***P≤0.001, **P≤0.01, *P≤0.05, and ns: not significant. **(d)** Abrogation of the ATAD5-BAZ1B interaction causes increased DNA breaks after oxidative stress. Another ATAD5 KO+B1m clone (B1m #2) also shows more DNA breaks after oxidative stress, consistent with the B1m #1 clone. The alkaline comet assay was performed with ATAD5 KO+WT and ATAD5 KO+B1m #2 cells after treatment with H_2O_2 (0.1 mM for 1 hour). The scale bar in representative micrographs is 100 μm. The right panel shows the quantification of the tail moment. At least 340 nuclei were quantified for each condition and red lines indicate the corresponding mean values. Statistical analysis: Two-way ANOVA; ****P≤0.0001. **(e)** BAZ1B depletion increases sensitivity to H_2O_2. A clonogenic survival assay was conducted with BAZ1B-depleted cells following treatment with H_2O_2 (0-1 mM for 16 hours). Error bars represent the standard deviation of the mean from three independent replicates. Statistical analysis: Ordinary one-way ANOVA; ****P≤0.0001. Source data are provided as a Source Data file.

Table S1. Plasmids used in this study.

Plasmid	Reference
pcDNA5/FRT/TO FLAG-(ATAD5)	[1]
pcDNA5/FRT/TO FLAG-(ATAD5 BET M2)	[2]
pcDNA5/FRT/TO FLAG-(ATAD5 B1m)	This Study
pcDNA5/FRT/TO FLAG-(ATAD5 YFAA)	This Study
pcDNA5/FRT/TO FLAG-(ATAD5 1-692)	[1]
pcDNA5/FRT/TO FLAG-(ATAD5 1-29+241-692)	This Study
pcDNA5/FRT/TO FLAG-(ATAD5 1-240+321-692)	This Study
pcDNA5/FRT/TO FLAG-(ATAD5 1-320+401-692)	This Study
pcDNA5/FRT/TO FLAG-(ATAD5 1-400)	This Study
pcDNA5/FRT/TO FLAG-(ATAD5 1-692 B1m)	This Study
pcDNA5/FRT/TO FLAG-(ATAD5 1-500)	This Study
pcDNA5/FRT/TO FLAG (ATAD5 1-500 B1m)	This Study
pcDNA5/FRT/TO FLAG-(ATAD5 1-500 S261A)	This Study
pcDNA5/FRT/TO FLAG-(ATAD5 1-500 S266A)	This Study
pcDNA5/FRT/TO FLAG-(ATAD5 1-500 S266D)	This Study
pcDNA5/FRT/TO FLAG-(ATAD5 1-500 K272A)	This Study
pcDNA5/FRT/TO FLAG-(ATAD5)	This Study
pcDNA5/FRT/TO FLAG-(ATAD5 1-692)	This Study
pcDNA5/FRT/TO FLAG-(ATAD5 693-1844)	This Study
pcDNA5/FRT/TO (ATAD5)-FLAG-mNeonGreen	This Study
pcDNA5/FRT/TO (ATAD5 B1m)-FLAG-mNeonGreen	This Study
pcDNA5/FRT/TO (ATAD5 NTM)-FLAG-mNeonGreen	This Study
pcDNA5/FRT/TO (ATAD5 1-692)-FLAG-mNeonGreen	This Study
pcDNA5/FRT/TO (ATAD5 1-692 N-PIPm)-FLAG-mNeonGreen	This Study
pcDNA5/FRT/TO (ATAD5 1-692 NTM)-FLAG-mNeonGreen	This Study
pcDNA5/FRT/TO (ATAD5 1-692 UAF1m)-FLAG-mNeonGreen	This Study
pcDNA5/FRT/TO (ATAD5 1-400)-FLAG-mNeonGreen	This Study
pcDNA5/FRT/TO (ATAD5 693-1844)-FLAG-mNeonGreen	This Study
pcDNA5/FRT/TO (BAZ1B)-V5	This Study
pcDNA5/FRT/TO (BAZ1B 1-1000)-V5	This Study
pcDNA5/FRT/TO (BAZ1B 501-1000)-V5	This Study
pcDNA5/FRT/TO (BAZ1B 1-500)-V5	This Study
pcDNA5/FRT/TO (BAZ1B 1-466)-V5	This Study
pcDNA5/FRT/TO (BAZ1B 1-322)-V5	This Study
pcDNA5/FRT/TO (BAZ1B 1001-1483)-V5	This Study
pcDNA5/FRT/TO (BAZ1B 539-1483)-V5	This Study
pcDNA5/FRT/TO (BAZ1B 593-1483)-V5	This Study
pcDNA5/FRT/TO (BAZ1B 633-1483)-V5	This Study
pcDNA5/FRT/TO (BAZ1B 677-1483)-V5	This Study
pcDNA5/FRT/TO (BAZ1B)-FLAG	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B A5BM1)-StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B A5BM2)-StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B PIPm)-StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B ΔBromo)-StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B 1-1026+1166-1483)- StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B 1-1167+1215-1483)-StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B)-StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B E118A)-StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B D121A)-StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B F122A)-StrepII-FLAG-NLS	This Study

pcDNA5/FRT/TO CLIP-(BAZ1B K221A)-StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B Y222A)-StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B D223A)-StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B V224A)-StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B K225A)-StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B ZNM1)-StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B ZNM2)-StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B ZNM3)-StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B ZNM4)-StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B ZNM5)-StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B ZNM6)- StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B ZNM7)- StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B ZNM8)- StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B ZNM9)- StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B ZNM10)- StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B ZNM11)- StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B ZNM12)- StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B ZNM13)- StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B ZNM14)- StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B ZNM15)- StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B ZNM16)- StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B ZNM17)- StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B ZNM18)- StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B ZNM19)- StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B ZNM20)- StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B A5BM1m)- StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B A5BM2m)- StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B S5BM1m)- StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B S5BM2m)- StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B)-StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B A5BM3m)-StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B hinge mutation)-StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B PIPm)-StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B 42-1483)-StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B 129-1483)-StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B 190-1483)-StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B 1-189+366-1483)-StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO CLIP-(BAZ1B 1-365+531-1483)-StrepII-FLAG-NLS	This Study
pcDNA5/FRT/TO (BAZ1B)-FLAG-mNeonGreen	This Study
pcDNA5/FRT/TO (BAZ1B PIPm)-FLAG-mNeonGreen	This Study
pcDNA5/FRT/TO (BAZ1B F122A)-FLAG-mNeonGreen	This Study
pcDNA5/FRT/TO (BAZ1B ΔBromo)-FLAG-mNeonGreen	This Study
pcDNA5/FRT/TO (SMARCA5)-V5	This Study
pcDNA5/FRT/TO (SMARCA5 ΔSLIDE)-V5	This Study
pcDNA5/FRT/TO HA-(PCNA)	3
pcDNA5/FRT/TO HA-Ubiquitin- (PCNA)	This Study
pcDNA5/FRT/TO HA-SUMO1-(PCNA)	This Study
pcDNA5/FRT/TO HA-SUMO2-(PCNA)	This Study
pLVX CLIP-(ATAD5)-StrepII-FLAG-IRES-mNeonGreen	This Study
pLVX CLIP-(ATAD5 B1m)-StrepII-FLAG-IRES-mNeonGreen	This Study
pFL StrepII-(BAZ1B)-FLAG, His-(SMARCA5)	This Study
pFL His-(UAF1), StrepII-(USP1 G670A,G671A)	This Study
pFastBac His-(3xPCNA)-FLAG-HSV	This Study

Supplementary Reference

1. Kang, M.S. et al. Regulation of PCNA cycling on replicating DNA by RFC and RFC-like complexes. *Nat Commun* **10**, 2420 (2019).
2. Kang, M.S. et al. PCNA Unloading Is Negatively Regulated by BET Proteins. *Cell Rep* **29**, 4632-4645 e5 (2019).
3. Kim, S. et al. PCNA Ser46-Leu47 residues are crucial in preserving genomic integrity. *PLoS One* **18**, e0285337 (2023).