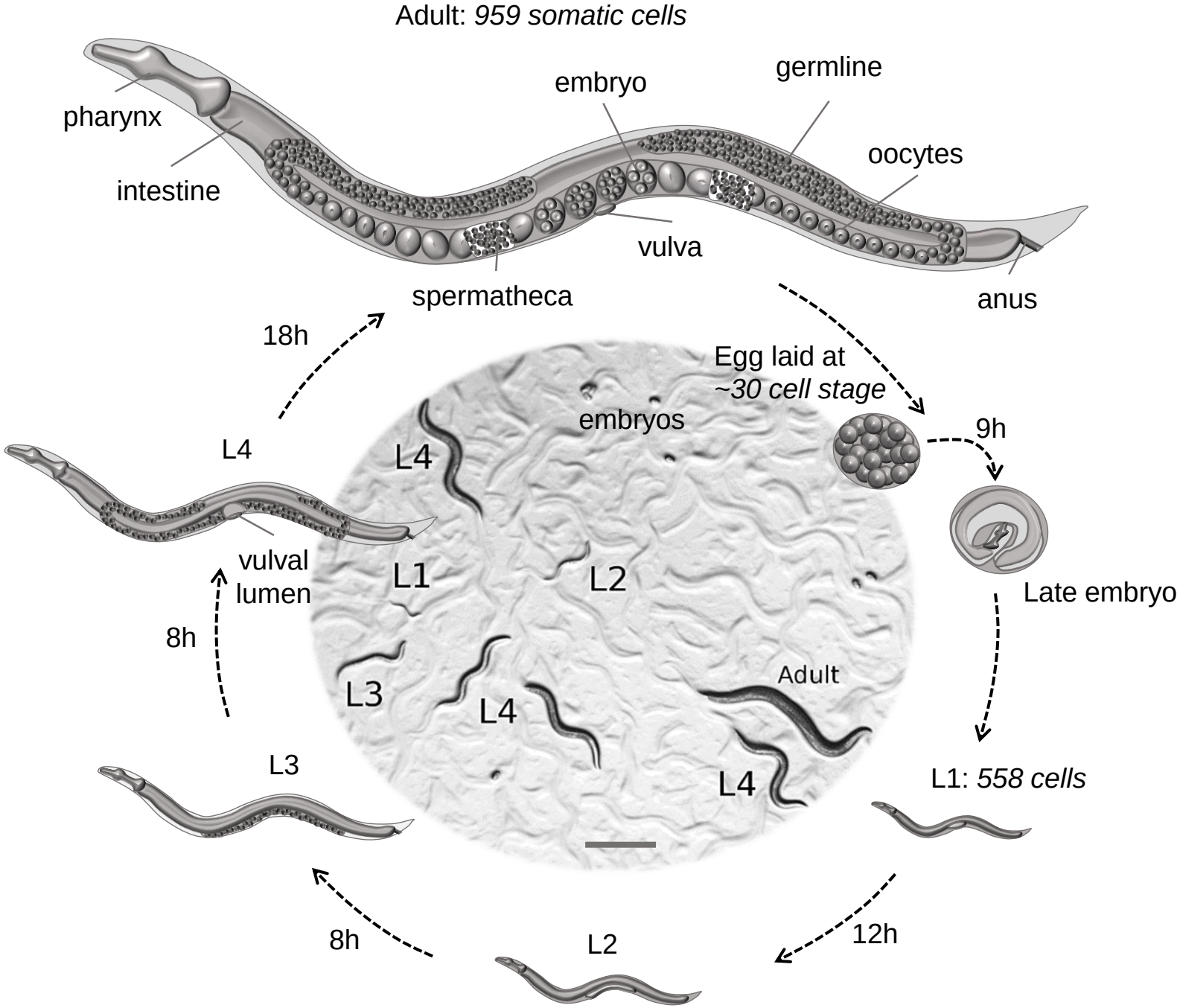
Adult: *959 somatic cells*
pharynx
intestine
embryo
germline
oocytes
vulva
spermatheca
anus
18h
Egg laid at
~30 cell stage
9h
L4
embryos
L4
L1
L2
Late embryo
vulval
lumen
8h
L3
L4
Adult
L4
L3
L1: *558 cells*
8h
L2
12h

Fig. S1

Figure S1: *C. elegans* life cycle and developmental stages. The simplified scheme shows the life cycle of *C. elegans* from fertilized embryo to egg, the four larval stages (L1-L4), and finally the fertile adult. After zygote formation (0 min), the generation of embryonic founder cells occurs within the mother until the egg is laid at the 30 cell-stage (~150 min), after which the embryo undergoes a bulk of its cell divisions and gastrulation (150-350 minutes). Over the next nine hours, the embryo passes through organogenesis/morphogenesis and elongates three-fold into an animal with fully developed organs. The newly hatched larva (L1) has 558 cells, of which 51 are blast cells that continue dividing, developing primarily into the reproductive and nervous systems through the four larval stages. A full life-cycle is completed after 2.5 days at 20°C and culminates in 959 postmitotic cells, as well as a continuously dividing germ stem population in the most distal gonad. A fully deciphered cell lineage across all developmental stages is available at www.wormatlas.org/. The inlay is a stereoscopic bright field image of mixed developmental stages on an agar plate. Scale bar is 500 µm. The picture was modified from: Rieckher, M., Lopes, A.F.C., and Schumacher, B. (2016). Genome Stability in *C. elegans*. Elsevier Book edited by I Kovalchuk and O Kovalchuk *Genome Stability - From Virus to Human Application*, 163-186.

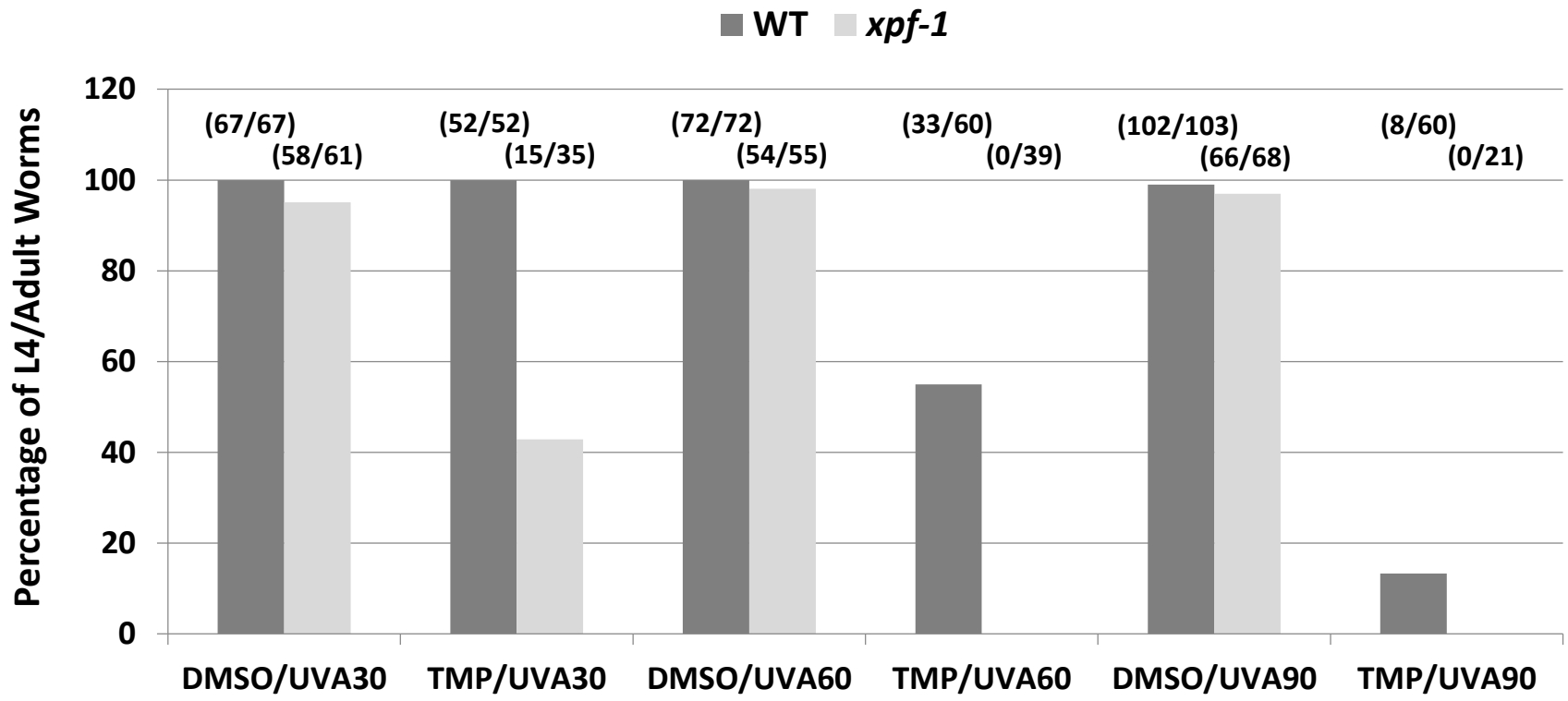
WT
xpf-1
Percentage of L4/Adult Worms
120
100
80
60
40
20
0
(67/67)
(58/61)
(52/52)
(15/35)
(72/72)
(54/55)
(33/60)
(0/39)
(102/103)
(66/68)
(8/60)
(0/21)
DMSO/UVA30
TMP/UVA30
DMSO/UVA60
TMP/UVA60
DMSO/UVA90
TMP/UVA90

Fig. S2

Figure S2: Sensitivity profile of the WT and *xpf-1* strains to the different treatment paradigms. L1 worms were treated with either DMSO or TMP (30 µg/ml) for 60 min, and then UVA irradiated for 30, 60 or 90 sec. Plotted is the percentage of L4/adult worms per total for each treatment group, 3 days after irradiation, from a representative experimental run (number of L4/adult worms per total indicated above).

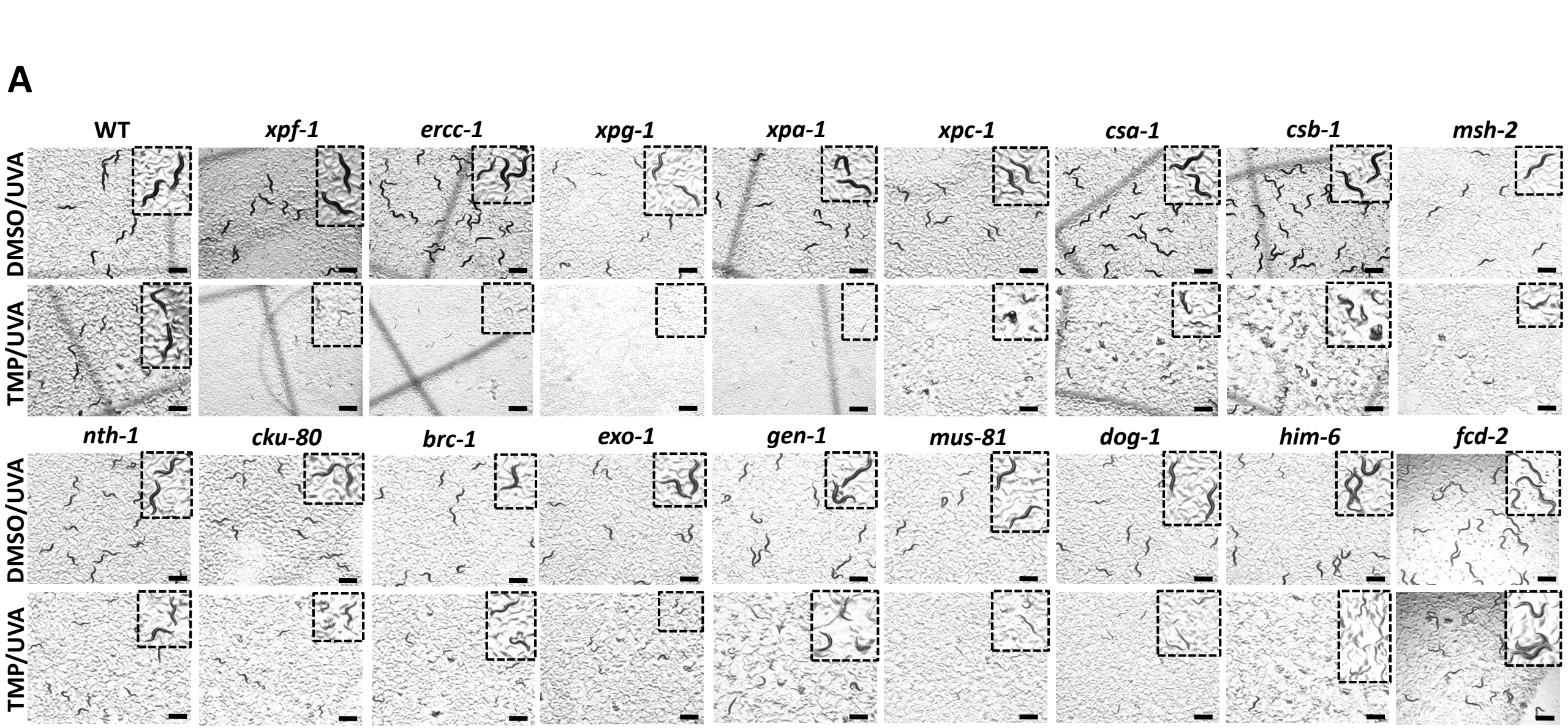
A
WT
xpf-1
ercc-1
xpg-1
xpa-1
xpc-1
csa-1
csb-1
msh-2
DMSO/UVA
TMP/UVA
nth-1
cku-80
brc-1
exo-1
gen-1
mus-81
dog-1
him-6
fcd-2
DMSO/UVA
TMP/UVA

Fig. S3A

B

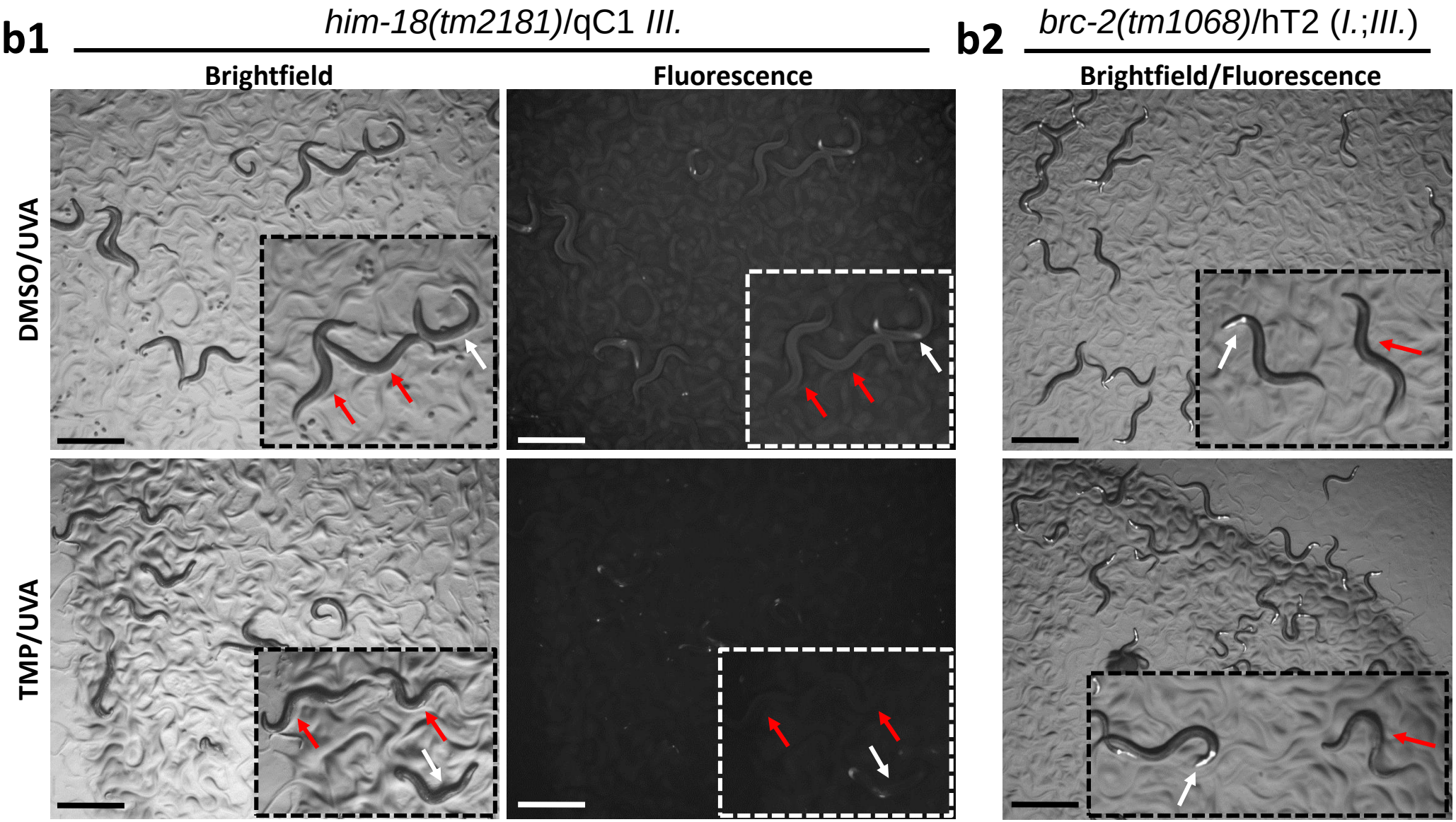
b1
him-18(tm2181)/qC1 III.
b2
brc-2(tm1068)/hT2 (I.;III.)
Brightfield
Fluorescence
Brightfield/Fluorescence
DMSO/UVA
TMP/UVA

Fig. S3B

Figure S3: TMP/UVA treatment causes developmental arrest and mis-development in various DNA repair deficient mutants. (A) L1 worms were treated with either DMSO or TMP (30 µg/ml) for 60 min, and then UVA irradiated for 60 sec. Representative images of animals three days post-treatment were recorded with a stereomicroscope. The inlays are 2x digital zoom of a representative area within the field of view. (B) The *him-18* and *brc-2* mutants that carry GFP-balancer chromosomes were recorded with a fluorescent stereomicroscope at three days post-treatment. The inlays are 1.8x digital zoom of a representative area within the field of view. (b1) Brightfield and fluorescent images were sequentially recorded to distinguish the WT and *him-18* heterozygous populations (GFP-positive and *rol-6*-positive, white arrow) from the homozygous *him-18* population (GFP-negative and no *rol-6* phenotype, red arrows). (b2) Brightfield and fluorescent images were simultaneously recorded to distinguish the WT and *brc-2* heterozygous populations (GFP-positive in the pharynx, white arrow) from the homozygous *brc-2* population (GFP-negative, red arrow). All size bars correspond to 1 mm.