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Supplemental Information

Atoh1 Directs Regeneration and Functional Recovery of the Mature Mouse Vestibular System

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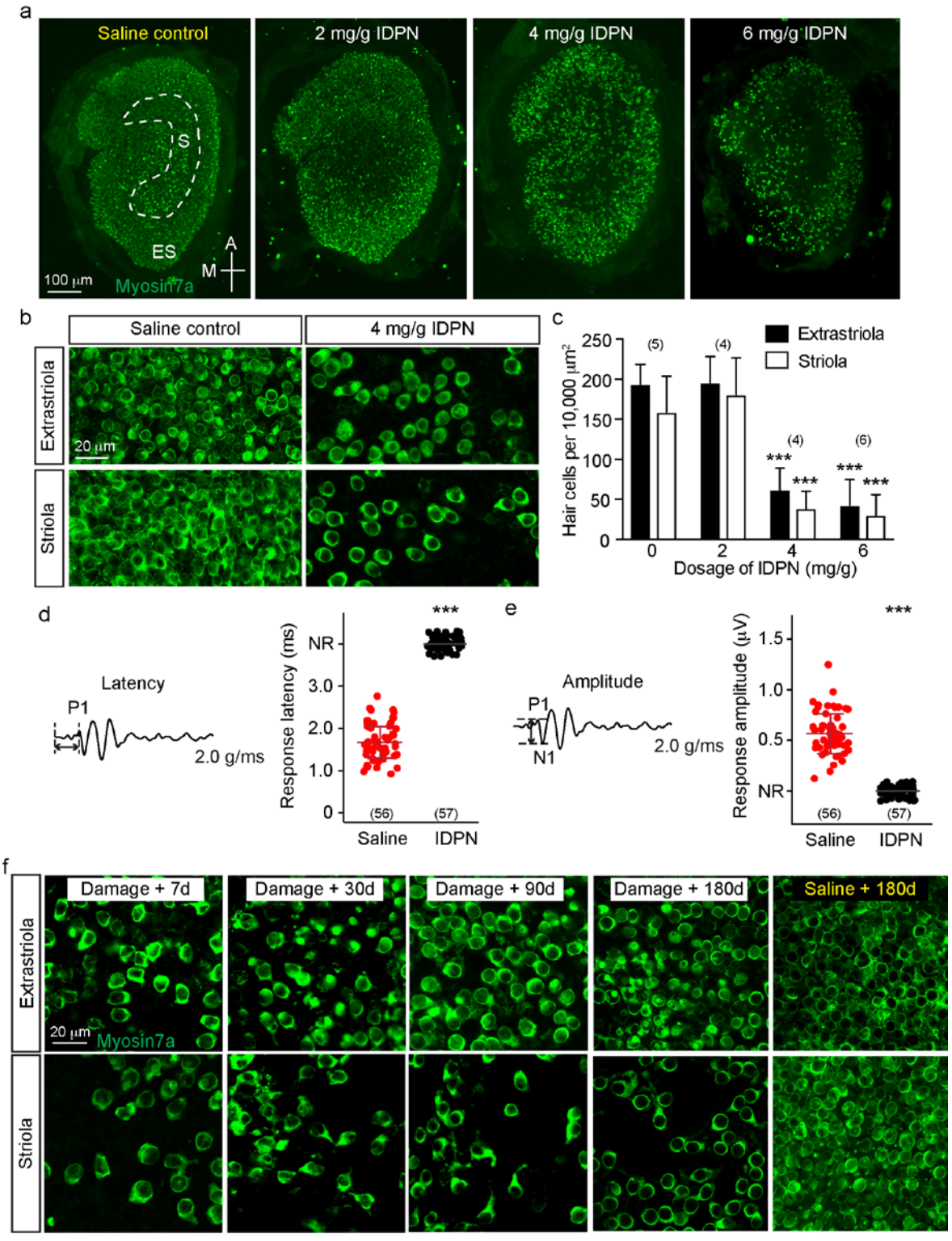
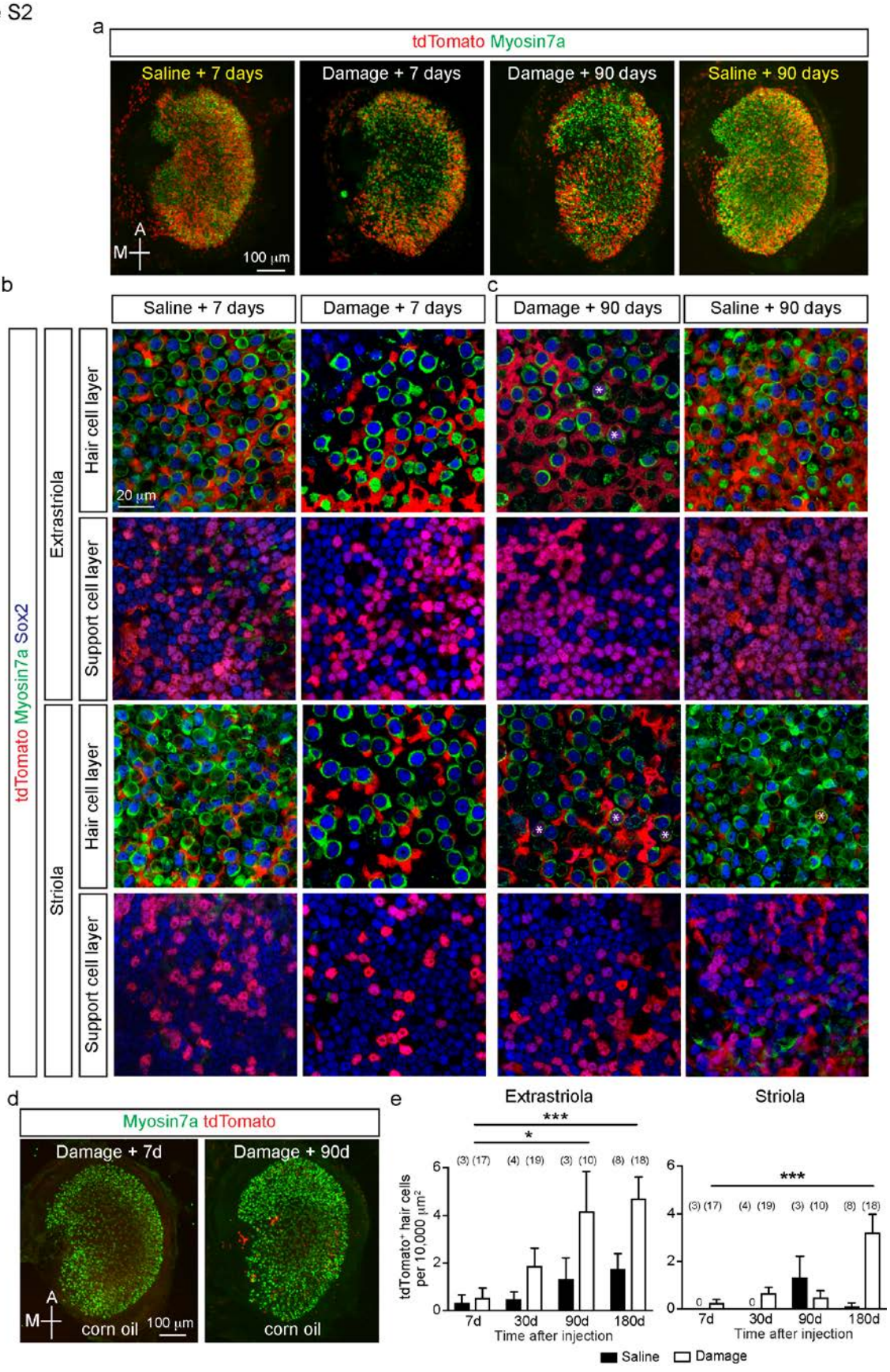
Supplementary Materials

Figure S1


a
Saline control
2 mg/g IDPN
4 mg/g IDPN
6 mg/g IDPN
S
ES
A
M
100 μm
Myosin7a
b
Saline control
4 mg/g IDPN
Extrastriola
Striola
20 μm
c
Extrastriola
Striola
Hair cells per 10,000 μm²
Dosage of IDPN (mg/g)
(5)
(4)
(4)
(6)
***
d
Latency
P1
2.0 g/ms
Response latency (ms)
NR
(56)
(57)
Saline
IDPN
e
Amplitude
P1
N1
2.0 g/ms
Response amplitude (μV)
f
Damage + 7d
Damage + 30d
Damage + 90d
Damage + 180d
Saline + 180d
Extrastriola
Striola
20 μm
Myosin7a

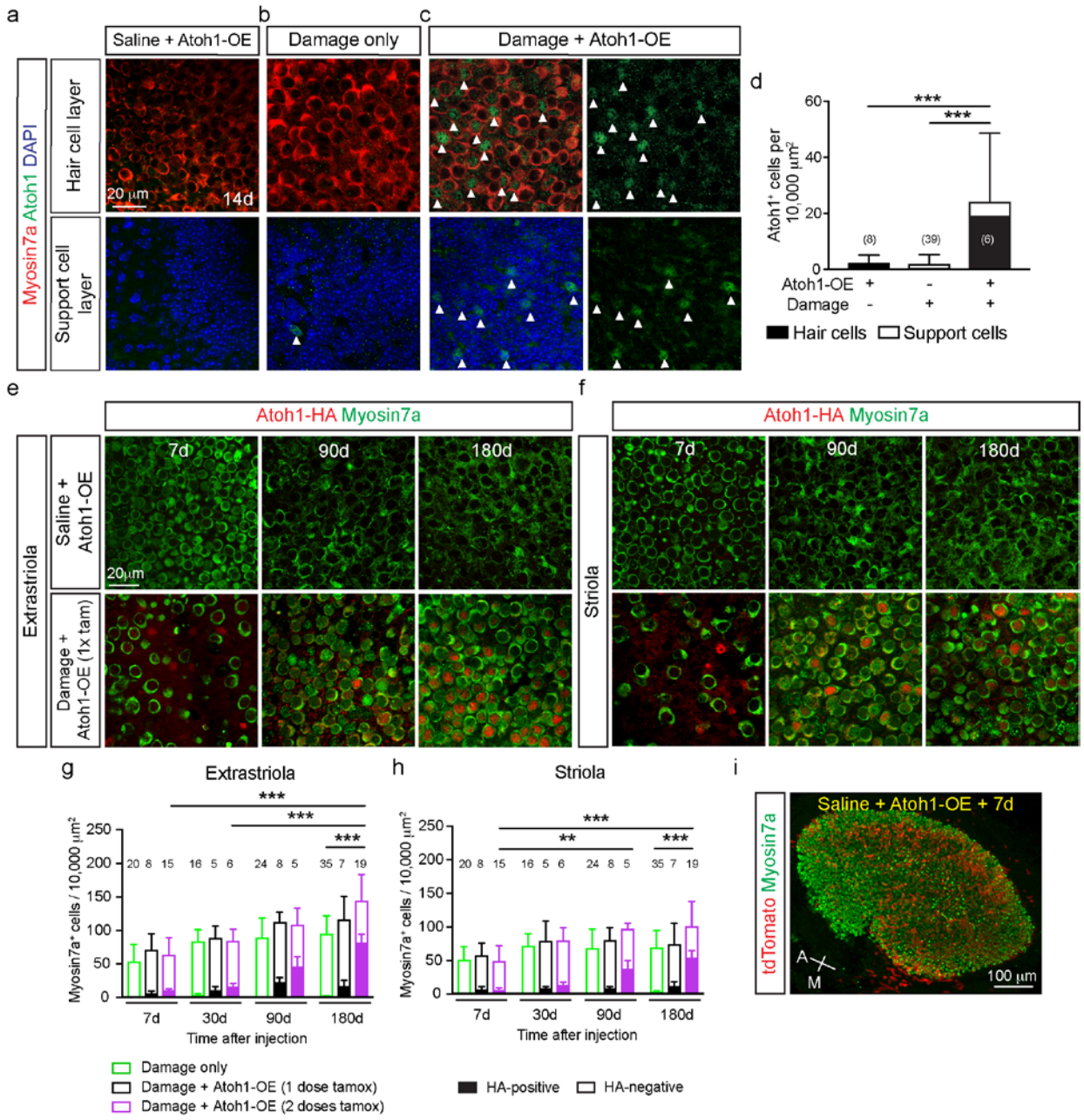
Supplemental Figure 1. Model of hair cell degeneration and regeneration (related to Figure 1). (a) IDPN (P30) caused a dose-dependent loss of HCs seven days later in wildtype mice *in vivo*. Dashed lines outline the striolar region. (b) Representative high magnification, confocal images of the extrastriolar and striolar regions showing HC loss in the IDPN damaged utricles as compared to undamaged controls. (c) Myosin7a^{+} cell counts showing significant HC loss in the extrastriolar and striolar regions at doses 4 mg/g or higher. A moderate dose (4 mg/g) was selected for use hereon, as the higher dose (6 mg/g) increased morbidity and mortality of mice. (d-e) Representative VsEP traces denoting P1 latency and P1-N1 amplitude in highest input stimulus (2.0 g/ms). IDPN-treated animals displayed significantly longer latencies and smaller amplitudes than undamaged controls. (f) Representative high magnification images showing HC loss, with regeneration primarily in the extrastriolar region. Two-way ANOVA with Dunnett's multiple comparisons test (c), two-way Student's t-tests for (d-e), n for utricles (c) or animals (d-e). * in (e) refers to comparisons to the 0 mg/kg dose.

Figure S2


a
tdTomato Myosin7a
Saline + 7 days
Damage + 7 days
Damage + 90 days
Saline + 90 days
A
M
100 μm
b
c
tdTomato Myosin7a Sox2
Extrastriola
Striola
Hair cell layer
Support cell layer
20 μm
d
Myosin7a tdTomato
Damage + 7d
Damage + 90d
corn oil
e
Extrastriola
Striola
tdTomato+ hair cells per 10,000 μm2
Time after injection
7d
30d
90d
180d
Saline
Damage

Supplemental Figure 2. Lineage tracing of support cells in the mature mouse utricle (related to Figure 1). (a) P32 $Plp1^{CreERT/+}$; $R26^{tdTomato/+}$ mice were administered tamoxifen. Cre recombination occurs primarily in the extrastriolar regions of the damaged and undamaged adult mouse utricles. (b) Representative high magnification images showing loss of HCs and tdTomato-labeling of support cells in both the extrastriolar and striolar regions seven days post treatment with saline or IDPN. The extrastriolar region displayed more robust tdTomato-labeling than the striolar region. (c) Ninety days post IDPN or saline treatment, many $tdTomato^+$ support cells and few fate-mapped, $tdTomato^+$, $Myosin7a^+$ HCs were identified (asterisks). (d) Mice treated with IDPN at P30 and vehicle control (corn oil at P32) had minimal Cre recombination (<1%) at both seven and 90 days post treatment, indicating low Cre recombinase leakiness. (e) Quantification shows significant increases in traced, $tdTomato^+$, $Myosin7a^+$ HCs in both the extrastriolar and striolar regions after IDPN-induced damage. Two-way ANOVA with Tukey's multiple comparisons test, n for utricles.

Figure S3

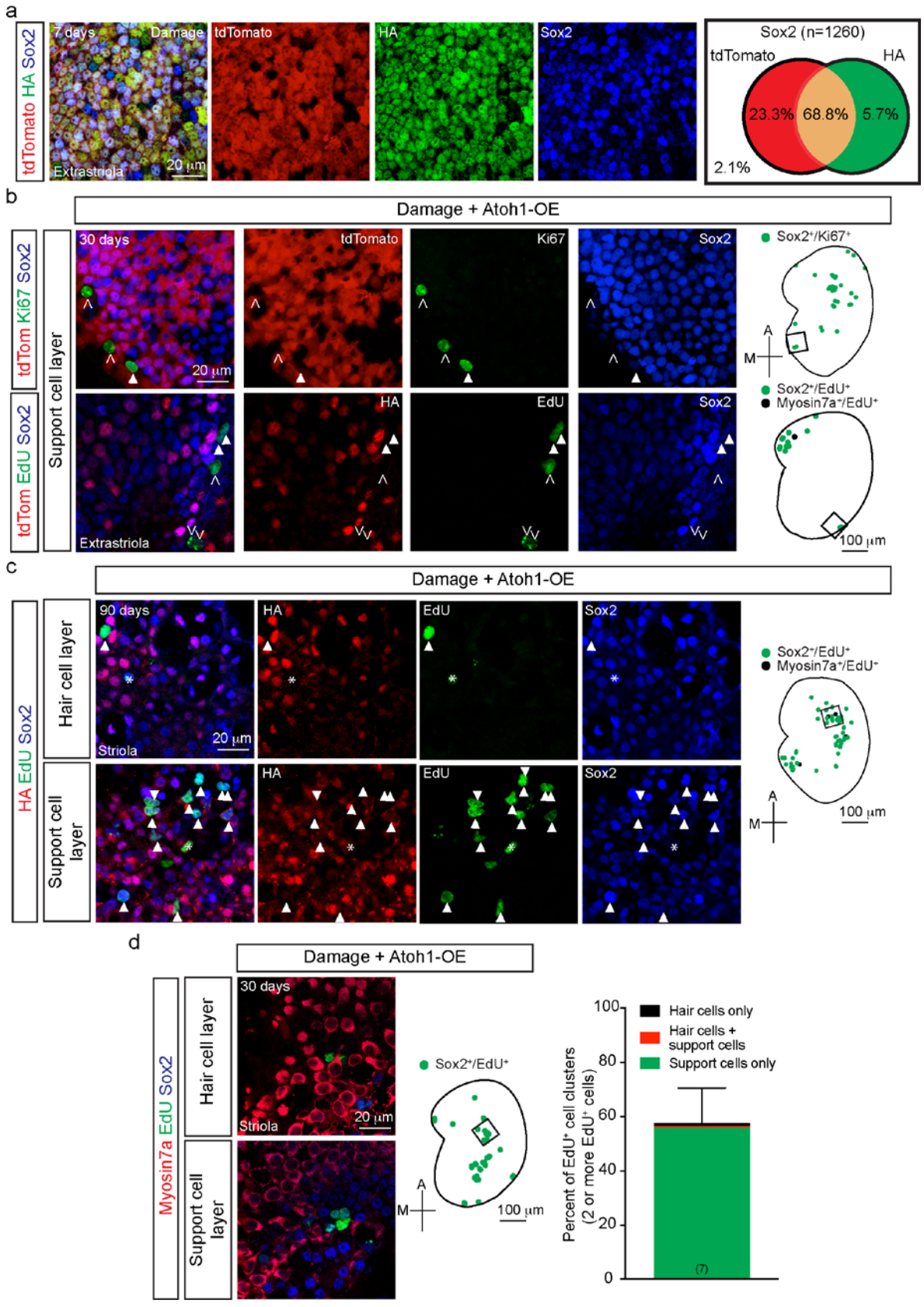


Supplemental Figure 3. Tamoxifen dosage affects the degree of Atoh1 OE in the damaged utricle (related to Figure 2). (a) With saline administration and Atoh1 OE, immunolabeling with Atoh1 antibody showed that Atoh1$^+$ cells (green) were almost always absent in the sensory epithelium at 14 days. (b) Rare Atoh1$^+$ cells were labeled 14 days after damage only (arrowheads). (c) Many Atoh1$^+$ HCs and support cells were

detected via antibody staining 14 days after damage and Atoh1 OE (arrowheads). (d) Quantification of Atoh1$^+$ cells shows significantly more Atoh1$^+$ cells in Atoh1 OE, damaged utricles than either damage-only or Atoh1 OE, undamaged organs. (e) Representative images of the extrastriolar region seven to 180 days after saline and Atoh1 OE or IDPN and one dose of tamoxifen to overexpress Atoh1. No HA$^+$ cells (red) were identified in the undamaged tissues. Myosin7a$^+$/HA$^+$ HCs were only detected in utricles after damage and Atoh1 OE, where more Myosin7a$^+$ HCs were found at 180 days post treatment. (f) Representative images of the striolar region from Atoh1 OE, undamaged and Atoh1 OE, damaged utricles at defined time points after treatment. No HA$^+$ cells were identified in the undamaged tissues. Myosin7a$^+$/HA$^+$ HCs gradually increased and more Myosin7a$^+$ HCs were noted at 90 and 180 days after damage and Atoh1 OE. (g) Quantification of extrastriolar HC density in damage-only, Atoh1 OE, damaged (single dose of tamoxifen), and Atoh1 OE, damaged (two doses of tamoxifen) tissues. HC density gradually increased after IDPN-induced damage. In the extrastriolar region, HC density is significantly higher in Atoh1 OE, damaged tissues (two doses of tamoxifen) than damage-only ones at 180 days after damage. HA$^+$ HCs gradually increased after damage and Atoh1 OE. (h) Quantification of HC density in the striolar region from damage-only, Atoh1 OE, damaged (single dose of tamoxifen), and Atoh1 OE, damaged (two doses of tamoxifen) tissues. HC density gradually increased only after IDPN-induced damage with two doses of tamoxifen to induce Atoh1 OE. Striolar HC density is significantly higher in Atoh1 OE, damaged tissues than damage-only ones at 180 days after damage. Traced HA$^+$ HCs in the striolar region also increased over time after damage and Atoh1 OE. (i) Robust tdTomato expression in undamaged

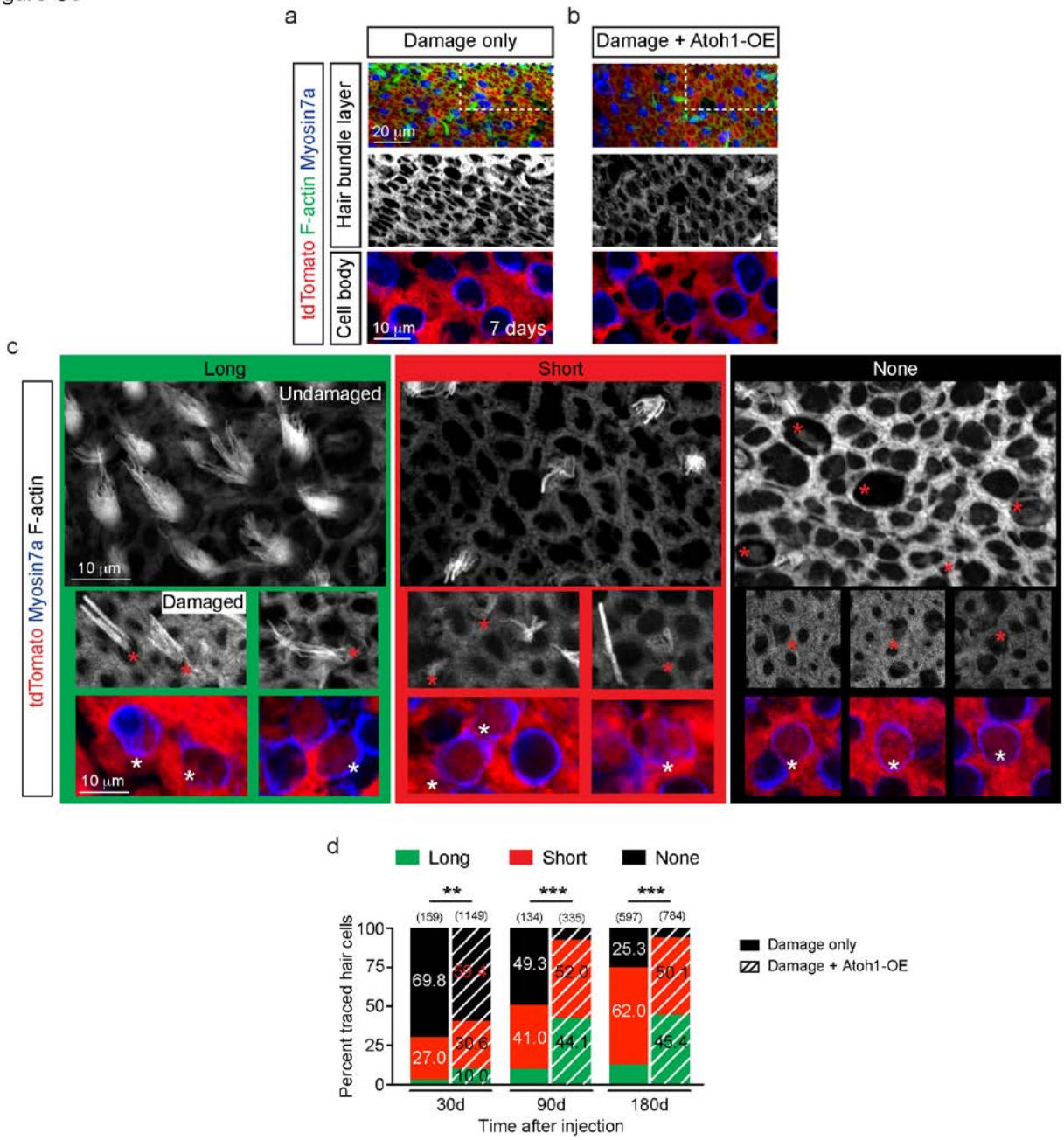
utricles from *Plp1*$^{CreERT/+}$*; R26*$^{tdTomato/+}$*; CAG*$^{Atoh1\text{-}HA/+}$ mice treated with saline and tamoxifen. One-way ANOVA with Tukey's multiple comparisons test (d), two-way ANOVA with Tukey's multiple comparisons test (f, h), n for utricles.

Figure S4


a
tdTomato HA Sox2
7 days
Damage
Extrastriola
20 μm
tdTomato
HA
Sox2
Sox2 (n=1260)
tdTomato
HA
23.3%
68.8%
5.7%
2.1%
b
Damage + Atoh1-OE
tdTom Ki67 Sox2
tdTom EdU Sox2
Support cell layer
30 days
tdTomato
Ki67
Sox2
HA
EdU
Extrastriola
20 μm
Sox2+/Ki67+
Sox2+/EdU+
Myosin7a+/EdU+
A
M
100 μm
c
Damage + Atoh1-OE
HA EdU Sox2
Hair cell layer
Support cell layer
90 days
HA
EdU
Sox2
Striola
20 μm
Sox2+/EdU+
Myosin7a+/EdU+
A
M
100 μm
d
Damage + Atoh1-OE
Myosin7a EdU Sox2
Hair cell layer
Support cell layer
30 days
Striola
20 μm
Sox2+/EdU+
A
M
100 μm
Hair cells only
Hair cells + support cells
Support cells only
Percent of EdU+ cell clusters (2 or more EdU+ cells)
100
80
60
40
20
0
(7)

Supplemental Figure 4. Atoh1 OE increased proliferation in the damaged mature mouse utricle (related to Figure 2). (a) Representative images of support cells from *Plp1*$^{CreERT/+}$*; R26*$^{tdTomato/+}$*; CAG*$^{Atoh1\text{-}HA/+}$ mouse utricle after IDPN and tamoxifen administration. Nearly 70% of Sox2$^+$ support cells in the extrastriolar region were doubled labeled with tdTomato and HA. (b) Ki67$^+$ and EdU$^+$ support cells were detected in the extrastriolar region 30 days after damage and Atoh1 OE. Arrowheads and chevrons indicate Sox2-positive and -negative proliferative (Ki67$^+$ or EdU$^+$) cells. Drawings of utricles show location of proliferative Sox2-positive (green) and Myosin7a-positive cells (black), with black boxes representing areas in the extrastriolar regions where high magnification images were captured. (c) Ninety days after damage and Atoh1 OE, many EdU$^+$ cells were detected in the striolar region, consisting of many EdU$^+$/Sox2$^+$/Myosin7a$^-$ support cells (arrowheads) and a few EdU$^+$/Sox2$^+$/Myosin7a$^+$ HCs (asterisks). Cartoon shows EdU$^+$/Sox2$^+$ (green) and EdU$^+$/Myosin7a$^+$ (black) cells. The black box outlines area in the striolar region where high magnification images were captured. This is the same utricle as shown in Figure 4f. Note that none of the EdU$^+$ cells are HA$^+$. (d) A cluster of EdU$^+$ cells was identified spanning the HC layer and support cell layer of the utricular sensory epithelium 30 days after damage and Atoh1 OE. Cartoon shows EdU$^+$/Sox2$^+$ (green) cells. Among the utricles treated with IDPN and Atoh1 OE, most (58.1%) proliferating cells appeared in foci (two or more cells directly adjacent to one another). Nearly all of these clusters consist of groups of support cells. We rarely found collections of EdU$^+$ support cells adjacent to EdU$^+$ HCs or clusters of EdU$^+$ HCs. N for utricles.

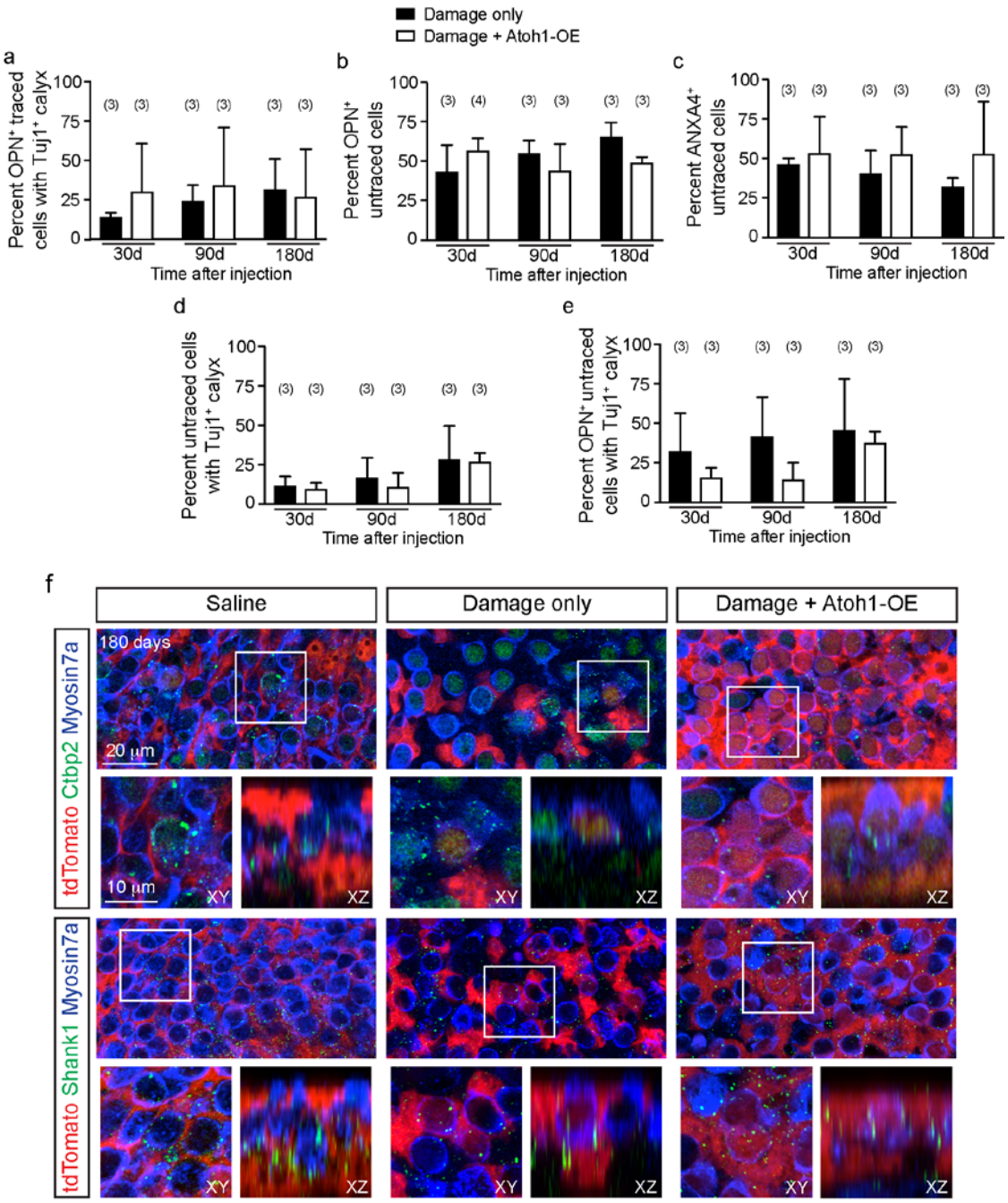
Figure S5



Supplemental Figure 5. Atoh1 OE promotes stereociliary bundle formation in the damaged mature utricle (related to Figure 3). (a-b) Seven days after damage only (a) and damage and Atoh1 OE (b), the apical surface of utricles was largely devoid of stereociliary bundles, and no tdTomato^{+}/Myosin7a^{+} HCs were identified. Dashed boxes represent area from which high magnification images at the hair bundle and cell body

levels are shown. (c) Representative areas depicting long (green), short (red), and no (black) hair bundles. Select traced HCs (asterisks) with associated bundles among the three bundle groups. Lower magnification image of long hair bundles were captured from undamaged utricle, images of short and no bundles were taken from damaged utricles. (d) Quantitative analyses showing the proportion of fate-mapped, tdTomato^{+} HCs with long, short, and no associated stereociliary bundles in damage-only utricles and in Atoh1 OE, damaged utricles. 30 days after damage, most traced HCs had no associated bundles. Bundles detected on traced HCs were primarily short in damage-only and Atoh1 OE, damaged utricles. The proportion of traced cells with long and short bundles 30 days after damage and Atoh1 OE was comparable to that with damage alone. By 90-180 days, nearly 100% of traced HCs after damage and Atoh1 OE had associated bundles, significantly more of which appeared long and thus more mature than those from damage-only utricles. χ^2 test, n for HCs.

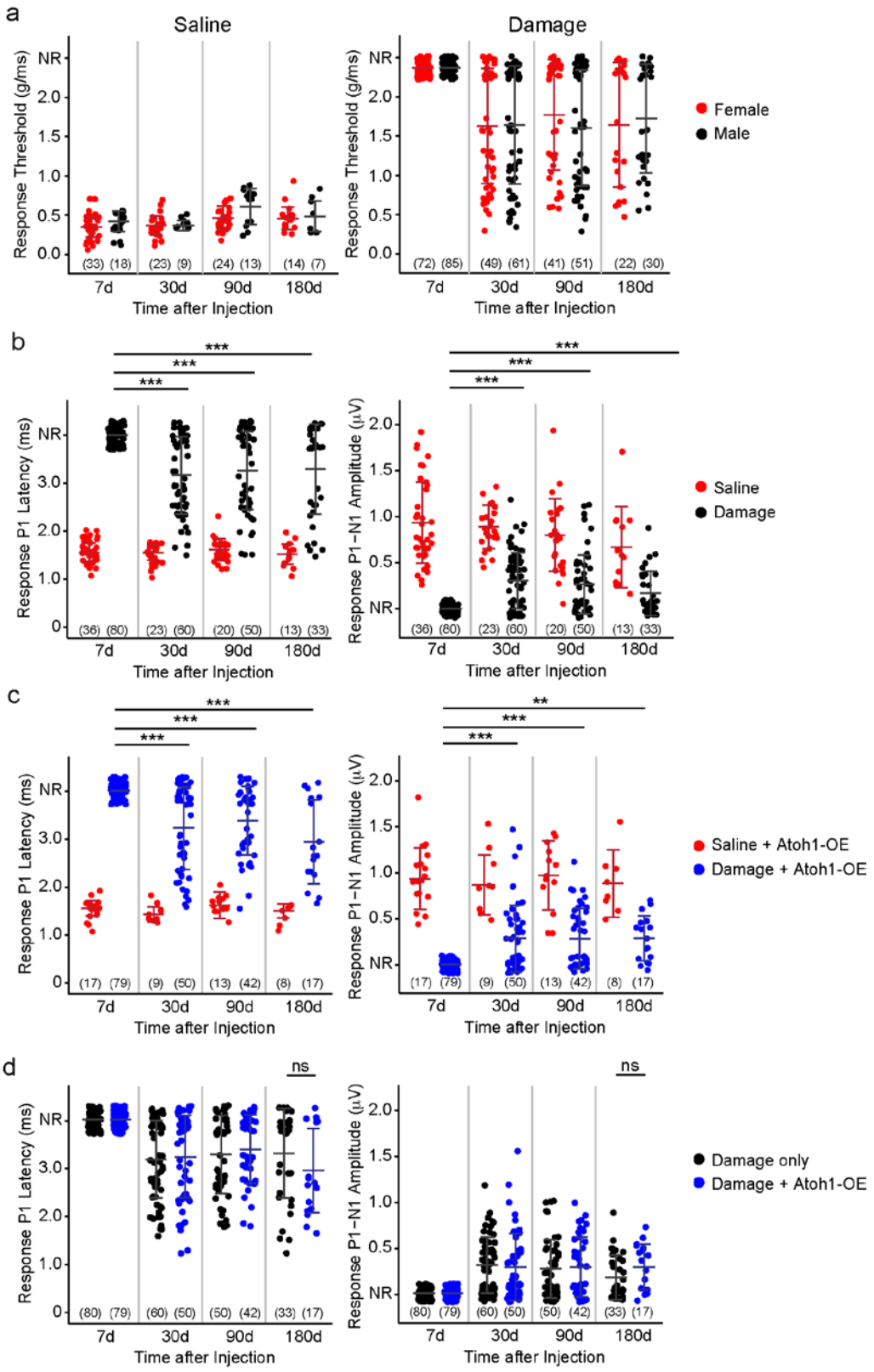
Figure S6



Supplemental Figure 6. Atoh1 increases regeneration of neurally integrated type II hair cells (related to Figure 4). (a) Quantification of traced OPN$^+$ HCs with Tuj1$^+$ calyces

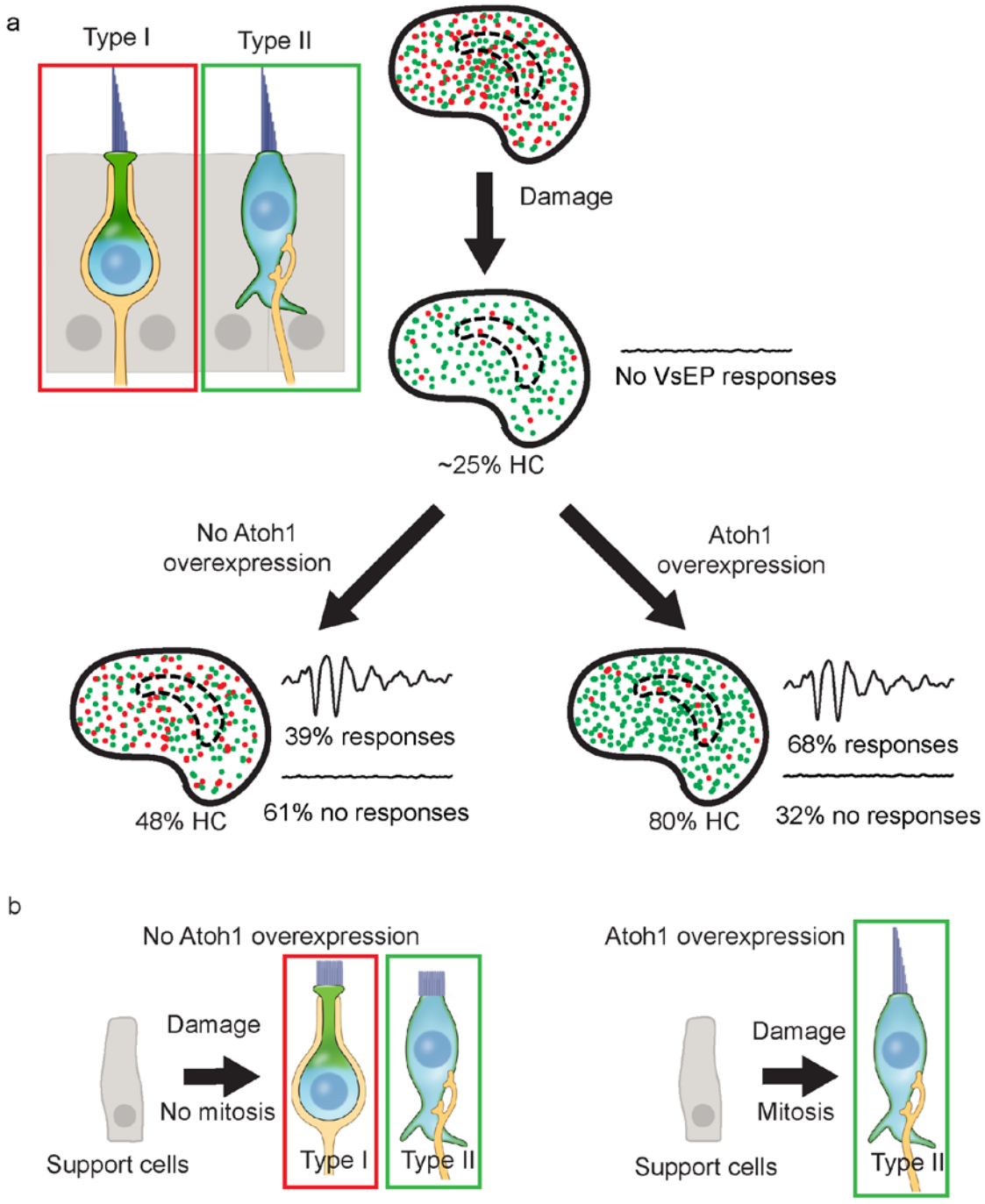
showing no significant change in damage-only and Atoh1 OE, damaged utricles over time. (b-c) Quantification of untraced tdTomato$^-$, Myosin7a$^+$ HCs shows that the percentage of tdTomato$^-$, Myosin7a$^+$ HCs expressing OPN and ANXA4 remain relatively stable over time in both damage-only and Atoh1 OE, damaged utricles, with no significant differences across time points or genotypes. (d) Quantification of untraced tdTomato$^-$, Myosin7a$^+$ HCs with Tuj1$^+$ calyces shows an increase (albeit not statistically significant) in tdTomato$^-$, Myosin7a$^+$ HCs over time in both damage-only and Atoh1 OE, damaged utricles. (e) Quantification of untraced OPN$^+$ HCs with a Tuj1$^+$ calyx showing no significant change in damage-only and Atoh1 OE, damaged utricles over time. (f) Representative images of utricles 180 days after saline, damage only, and damage and Atoh1 OE. The white box outlines area where higher magnification images were captured showing representative traced and untraced HCs expressing presynaptic (Ctbp2) and postsynaptic (Shank1) markers. Two-way ANOVA with Tukey's multiple comparisons test, n for utricles.

Figure S7


a
Saline
Damage
Response Threshold (g/ms)
NR
2.0
1.5
1.0
0.5
0.0
(33) (18)
(23) (9)
(24) (13)
(14) (7)
(72) (85)
(49) (61)
(41) (51)
(22) (30)
7d
30d
90d
180d
Time after Injection
Female
Male
b
***
***
***
Response P1 Latency (ms)
3.0
2.0
1.0
0
(36) (80)
(23) (60)
(20) (50)
(13) (33)
Response P1−N1 Amplitude (μV)
Saline
Damage
c
**
Saline + Atoh1-OE
Damage + Atoh1-OE
(17) (79)
(9) (50)
(13) (42)
(8) (17)
d
ns
Damage only
Damage + Atoh1-OE
(80) (79)
(60) (50)
(50) (42)
(33) (17)

Supplemental Figure 7. Functional recovery of the damaged, mature mouse utricle (related to Figure 5). (a) No sex differences of VsEP thresholds were found in saline-treated or IDPN-treated mice across all time points examined. (b) As a group, VsEP latencies and amplitudes of damaged-only animals significantly improved over time relative to seven days post damage (black). Latencies and amplitudes of undamaged animals were stable over multiple time points (red). (c) As a group, VsEP latencies and amplitudes of Atoh1 OE, damaged animals significantly improved over time relative to seven days post damage (blue). Latencies and amplitudes of Atoh1 OE, undamaged animals were stable over multiple time points (red). (d) In comparison to damage-only animals (black), VsEP latencies and amplitudes of Atoh1 OE, damaged animals (blue) were not significantly different across all time points examined. Two-way ANOVA with Tukey's multiple comparisons test, n for animals.

Figure S8



Supplemental Figure 8. Proposed mechanisms of Atoh1 OE in augmenting hair cell regeneration and recovery of vestibular physiology (related to Figures 1-5). (a) Treatment with IDPN results in significant loss of utricular HCs to ~25% of normal seven

days later, with mostly type II HCs remaining. Functionally, this results in a complete loss of VsEP waveforms. 180 days later, the adult mouse utricle regenerates type I and type II HCs—primarily in the extrastriolar region—to 48% of control levels, resulting in a sustained recovery of VsEP waveforms in 39% of mice. 180 days after IDPN-induced damage and Atoh1 OE, total HC number increased to 80% of age-matched control levels, with mostly type II HCs regenerated in both the striolar and extrastriolar regions. Functionally, 68% of IDPN-treated, Atoh1-overexpressed mice have a sustained recovery of VsEP function. (b) After damage only, support cells of the spontaneously regenerating adult mouse utricle differentiate into type I and type II HCs through non-mitotic mechanisms. After damage and Atoh1 OE, support cells proliferate and also regenerate type II HCs with elongated stereocilia and innervation.

Supplemental Table 1. Quantification of Myosin7a$^+$ hair cells (related to Figure 2).

Supplemental Table 1. Quantification of Myosin7a$^+$ hair cells

Extrastriola					
	Time after injection	No damage	Atoh1 OE only	Damage	Damage + Atoh1 OE
Total cells	7 days	182.4±41.9$^{\#\#\#}$ (9)	161.4±39.3$^{\#\#\#}$ (6)	53.5±24.7 (20)	63.2±24.9 (15)
	30 days	188.7±31.2$^{\#\#\#}$ (10)	208.7±41.4$^{\#\#\#}$ (3)	83.1±17.0 (16)	84.2±16.8 (6)
	90 days	159.6±37.9 (7)	199.3±47.0 (3)	89.2±28.5* (24)	108.4±23.8 (5)
	180 days	167.6±37.4 (11)	159.0±15.2 (4)	95.2±25.8$^{***,\#\#\#}$ (35)	144.8±39.2*** (19)
HA$^+$ cells	7 days	0.2±0.4 (5)	1.3±1.2 (3)	0.3±0.8 (7)	10.8±4.3 (8)
	30 days	0.0±0.0 (3)	1.7±1.2 (3)	3.0±4.2 (5)	16.2±11.9 (5)
	90 days	0.0±0.0$^{\#\#\#}$ (5)	2.3±1.5$^{\#\#}$ (3)	0.0±0.0$^{\#\#\#}$ (5)	46.3±28.5** (4)
	180 days	1.3±1.9$^{\#\#\#}$ (7)	5.3±3.1$^{\#\#\#}$ (3)	1.0±1.1$^{\#\#\#}$ (8)	81.4±36.3*** (7)
Striola					
	Time after injection	No damage	Atoh1 OE only	Damage	Damage + Atoh1 OE
Total cells	7 days	162.8±42.3$^{\#\#\#}$ (9)	135.3±43.2$^{\#\#\#}$ (6)	51.1±18.9 (20)	48.6±24.1 (15)
	30 days	159.3±45.8$^{\#\#\#}$ (10)	192.8±28.7$^{\#\#\#}$ (3)	71.2±18.8 (16)	79.5±18.3 (6)
	90 days	150.1±47.6 (7)	155.3±14.6 (3)	68.0±27.7 (24)	97.2±7.1 (5)
	180 days	149.9±36.4$^{\#\#}$ (11)	145.5±34.0 (4)	69.1±25.7$^{\#}$ (35)	100.8±36.2*** (19)
HA$^+$ cells	7 days	0.2±0.4 (5)	1.7±1.5 (3)	0.0±0.0 (7)	6.1±5.6 (8)
	30 days	0.0±0.0 (3)	1.0±1.0 (3)	0.0±0.0 (5)	13.2±8.0 (5)
	90 days	0.2±0.4$^{\#\#\#}$ (5)	1.3±0.6$^{\#\#}$ (3)	0.0±0.0$^{\#\#}$ (5)	37.0±23.9*** (4)
	180 days	0.6±1.0$^{\#\#\#}$ (7)	2.7±1.2$^{\#\#\#}$ (3)	2.0±4.9$^{\#\#\#}$ (8)	53.6±25.8*** (7)

Shown are Myosin7a$^+$ hair cell counts as well as Myosin7a$^+$ HA$^+$ hair cells per 10,000 µm^2 from 7, 30, 90, and 180 days after treatment. Mean ± SD. Number of animals listed in parentheses.

* Represents significant difference with respect to the 7-day time point within a damage group.

Represents significant difference within the same time point between damaged + Atoh1-OE and the other groups.

* p<0.05, ** p<0.01, *** p<0.001 (two-way ANOVA followed by post-hoc analysis via Tukey's multiple comparisons test).

$^{\#}$ p<0.05, $^{\#\#}$ p<0.01, $^{\#\#\#}$ p<0.001 (two-way ANOVA followed by post-hoc analysis via Tukey's multiple comparisons test).

Supplemental Table 2. Quantification of HA$^+$ hair cells

Extrastriola				
	Days post damage	Damage alone	Damage + Atoh1 OE (1 dose)	Damage + Atoh1 OE (2 doses)
Total hair cells	7 days	53.5±24.7 (20)	70.6±23.9 (8)	63.2±24.9 (15)
	30 days	83.1±17.0 (16)	88.8±16.5 (5)	84.2±16.8 (6)
	90 days	89.2±28.5** (24)	112.1±14.1 (8)	108.4±23.8 (5)
	180 days	95.2±25.8** (35)	115.9±34.1*** (7)	144.8±39.2***,### (19)
HA$^+$ hair cells	7 days	0.3±0.8 (7)	6.3±7.9 (11)	10.8±4.3 (8)
	30 days	3.0±4.2 (5)	10.6±11.3 (5)	16.2±11.9 (5)
	90 days	0.0±0.0 (5)	22.8±20.3 (11)	46.3±28.5## (4)
	180 days	1.0±1.1 (8)	16.6±26.0 (10)	81.4±36.3***,### (7)
Striola				
	Days post damage	Damage alone	Damage + Atoh1 OE (1 dose)	Damage + Atoh1 OE (2 doses)
Total hair cells	7 days	51.1±18.9 (20)	57.3±17.6 (10)	48.6±24.1 (15)
	30 days	71.2±18.8 (16)	78.6±29.0 (5)	79.5±18.3 (6)
	90 days	68.0±27.7 (24)	80.4±17.6 (11)	97.2±7.1 (5)*
	180 days	69.1±25.7 (35)	74.2±30.2 (10)	100.8±36.2***,## (19)
HA$^+$ hair cells	7 days	0.0±0.0 (7)	6.3±11.7 (11)	6.1±5.6 (8)
	30 days	0.0±0.0 (5)	7.8±4.3 (4)	13.2±8.0 (5)
	90 days	0.0±0.0 (5)	8.0±6.2 (11)	37.0±23.9 (4)**,##
	180 days	2.0±4.9 (8)	11.4±18.2 (10)	53.6±25.8 (7)***,###

Shown are Myosin7a$^+$ hair cell counts as well as Myosin7a$^+$ HA$^+$ hair cells per 10,000 µm^2 from 7, 30, 90, and 180 days after IDPN-induced damage. Mean ± SD. Number of animals listed in parentheses.

* Represents significant difference across time points within a damage group between 7 days and later time points.

Represents significant difference within 1 time point between damage alone and other groups.

* p<0.05, ** p<0.01, *** p<0.001 (two-way ANOVA followed by post-hoc analysis via Tukey's multiple comparisons test).

p<0.01, ### p<0.001 (two-way ANOVA followed by post-hoc analysis via Tukey's multiple comparisons test).

Supplemental Table 2. Quantification of HA$^+$ hair cells (related to Figure 2).

Supplemental Table 3. Quantification of proliferating cells in the mature mouse utricle (related to Figure 2).

Supplemental Table 3. Quantification of proliferating cells in the mature mouse utricle

		Ki67		
	Time after injection	No damage	Atoh1 OE only	Damage only	Damage + Atoh1 OE
Hair cells	7 days	0.0±0.0 (5)	0.0±0.0 (3)	0.7±2.0 (9)	1.3±2.1 (10)
	30 days	0.0±0.0 (5)	0.0±0.0 (5)	0.0±0.0 (6)	0.1±0.3 (10)
	90 days	n/a	n/a	0.0±0.0 (3)	0.0±0.0 (4)
Support cells	7 days	0.0±0.0 (5)	0.0±0.0 (3)	0.0±0.0 (9)	0.1±0.3 (10)
	30 days	0.0±0.0 (5)	0.0±0.0 (5)	0.0±0.0 (6)	4.4±9.6 (10)
	90 days	n/a	n/a	0.0±0.0 (3)	0.0±0.0 (4)
		EdU			
	Time after injection	No damage	Atoh1 OE only	Damage only	Damage + Atoh1 OE
Hair cells	7 days	0.0±0.0 (3)	0.0±0.0 (3)	0.2±0.4 (5)	0.0±0.0 (6)
	30 days	0.0±0.0 (4)	0.2±0.4 (5)	0.0±0.0 (3)	1.5±4.1 (19)
	90 days	n/a	n/a	0.0±0.0 (4)	1.5±3.0 (4)
Support cells	7 days	0.0±0.0 (3)	0.0±0.0 (3)	0.0±0.0 (5)	0.0±0.0 (6)
	30 days	0.0±0.0 (4)	0.0±0.0 (5)	0.0±0.0 (3)	20.9±37.3 (19)
	90 days	n/a	n/a	0.8±1.5 (4)	15.8±28.9 (4)

Shown are $Ki67^+$ and EdU^+ hair cell and support cell counts per utricle from 7, 30, and 90 days after treatment. Mean ± SD. Number of animals listed in parentheses.

Supplemental Table 4. Physiology of the damaged and regenerating mouse utricle (related to Figure 5).

Supplemental Table 4. Physiology of the damaged and regenerating mouse utricle

Spontaneous				
Time after injection	Control (11)	Sustained recovery (11)	Unsustained recovery (13)	No recovery (5)
7 days	0.35±0.11	NR	NR	NR
30 days	0.39±0.14	1.32±0.76	1.56±0.76	NR
90 days	0.44±0.12	1.60±0.79	1.73±0.54	NR
180 days	0.52±0.18	1.08±0.29	NR	NR
Atoh1 OE				
Time after injection	Control (5)	Sustained recovery (13)	Unsustained recovery (2)	No recovery (4)
7 days	0.42±0.17	NR	NR	NR
30 days	0.37±0.07	1.84±0.75	2.03±0.49	NR
90 days	0.69±0.13	1.72±0.78	1.19±0.00	NR
180 days	0.44±0.15	0.89±0.37	NR	NR

Shown are average VsEP response thresholds (g/ms) from various recovery groups from 7, 30, 90, and 180 days after saline-treated and IDPN-induced damage. Mean ± SD. Number of animals listed in parentheses.