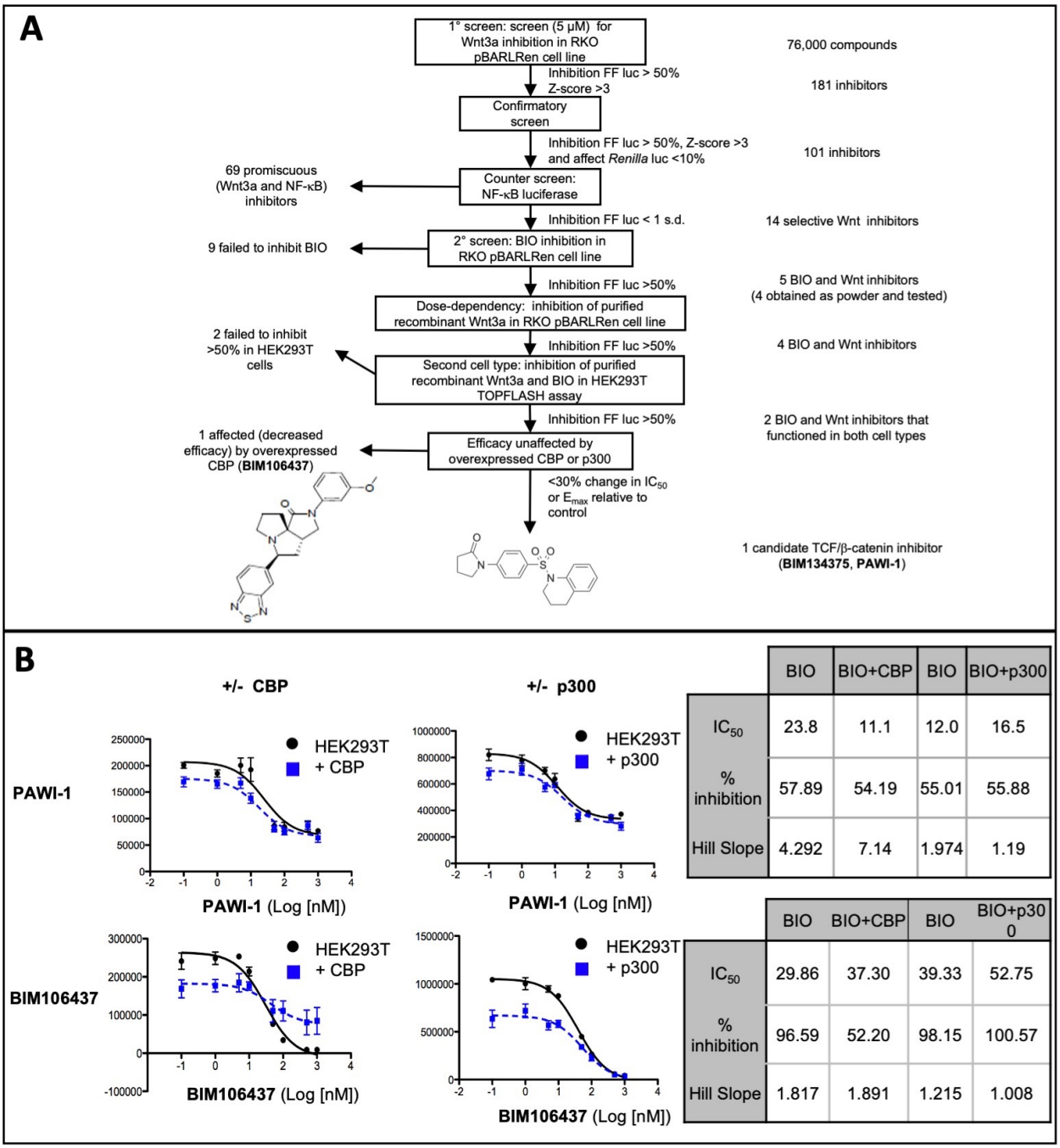
## Supplementary Figures

## Supplementary Fig. 1. Identification of candidate TCF/β–catenin inhibitors (relevant to Fig. 1)




| | BIO | BIO+CBP | BIO | BIO+p300 |
|---|---|---|---|---|
| $IC_{50}$ | 23.8 | 11.1 | 12.0 | 16.5 |
| % inhibition | 57.89 | 54.19 | 55.01 | 55.88 |
| Hill Slope | 4.292 | 7.14 | 1.974 | 1.19 |

| | BIO | BIO+CBP | BIO | BIO+p300 |
|---|---|---|---|---|
| $IC_{50}$ | 29.86 | 37.30 | 39.33 | 52.75 |
| % inhibition | 96.59 | 52.20 | 98.15 | 100.57 |
| Hill Slope | 1.817 | 1.891 | 1.215 | 1.008 |

### A) Screen strategy

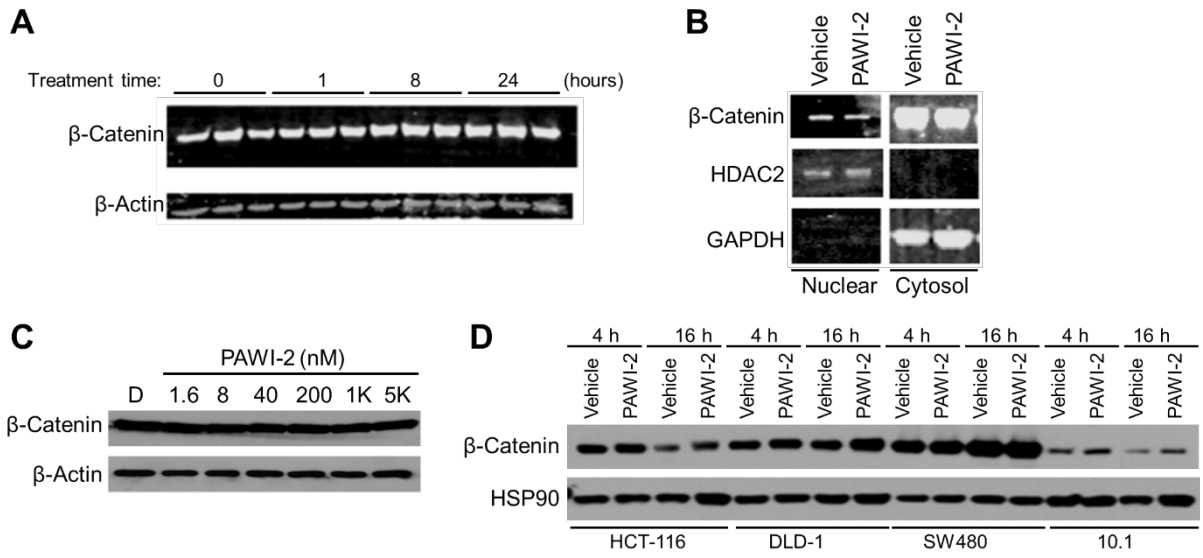
~76,000 small molecules from the Prebys Center for Drug Discovery chemical library were screened using a human colon carcinoma cell line (RKO) that had previously been engineered to harbor a firefly (FF) luciferase reporter cassette (pBARLRen (Biechele et al., 2009)) driven by twelve multimerized TCF response elements, with each separated by unique five nucleotide linkers specifically designed to minimize recombination that can lead to loss of the

integrated transgene, and a second PGK-*Renilla* luciferase cassette used to control for toxicity. The primary assay was optimized for 1536-well plate format high throughput screening (HTS) (Z' >0.5, S/B > 20). ~76,000 drug-like compounds from the Prebys Center for Drug Discovery at the Sanford-Burnham-Prebys Medical Discovery Institute (SBP) selected for chemical diversity (https://www.sbpdiscovery.org/medical-discovery/drug-discovery/prebys-center-for-drug-discovery/overview) were screened at a single concentration (5 μM) for the ability to block firefly (FF) luciferase activity induced by addition of recombinant human Wnt3a to the culture media (see **Methods**). 181 primary hits were obtained based on a cut-off of ≥50% inhibition and Z-score ≥3 (hit rate = 0.24%). Hits were confirmed by retesting and lack of inhibition of *Renilla* luciferase in the primary assay cell line, yielding 101 confirmed hits (0.13% of the initial screen). The 101 hits were evaluated at single concentration (5 μM) in counter screens to rule out promiscuous inhibitors using HEK293T cells stably transfected with an NF-κB responsive luciferase gene cassette (293-NF-kB-Luc) and a NOD1 expression transgene to induce NF-κB-dependent luciferase activity (cell line and assay described in Pubchem AID 1290). FF luciferase activities were measured 24 hours after compound addition. A secondary assay then evaluated the ability to inhibit the Wnt3a/β-catenin induced by BIO (1 μM), which stabilizes β-catenin through its inhibition of GSK3β, yielding 5 active compounds. Four of the 5 potent BIO and Wnt3a inhibiting compounds could be obtained from commercial sources as powders and were tested for dose-dependent inhibition (7 concentrations, 300-fold dose range, 10 nM to 3 μM) of recombinant purified human Wnt3a and BIO in both RKO pBARLRen cells (as in the 1° assay) and HEK393T cells transiently transfected with Super TOPflash plasmid (Addgene), yielding two compounds that had similar potencies and efficacy in both cell lines. These 2 compounds (BIM106437 and BIM134375) were re-tested in a final assay that evaluated inhibition following transfection to overexpress CBP or p300, two transcriptional co-activators that interact with β-catenin. The rationale for this final assay is to remove compounds that might be inhibitors of these proteins, rather than selective for the β-catenin/TCF response. A single compound (BIM134375, named **PAWI-1**) was minimally affected by CBP and p300 overexpression (**Supplementary Fig. 1B**).

**B) Discrimination of candidate inhibitors based on CBP and p300 interactions**

CBP (CREB binding protein) and p300 are closely related transcriptional co-activators that interact with numerous transcription factors, including β-catenin, to activate downstream genes. Both proteins have histone acetyl transferase (HAT) activity, and the interaction with β-catenin can be considered the last step in the canonical Wnt/β-catenin pathway. We reasoned that some compounds might affect the function of either of these proteins, and hence would not be specific modulators of TCF/β-catenin regulated gene expression outside the canonical pathway. Therefore, the final step of the HTS campaign was to discriminate compounds on the basis of whether their inhibition of Wnt/β-catenin depended upon either CBP or p300. HEK293T cells were transfected with plasmids directing the expression of either p300 or CBP and Super TOPflash (Addgene) in the presence of recombinant Wnt3a (10% conditioned media as for the primary screen). Compounds were added 24 hours later at the indicated concentrations, and luciferase was measured 24 hours later (n=4). Compounds are as in **Supplementary Fig. 1A**.

**Supplementary Fig. 2. PAWI-2 does not affect the protein level and cellular localization of β-catenin (relevant to Fig. 2).**

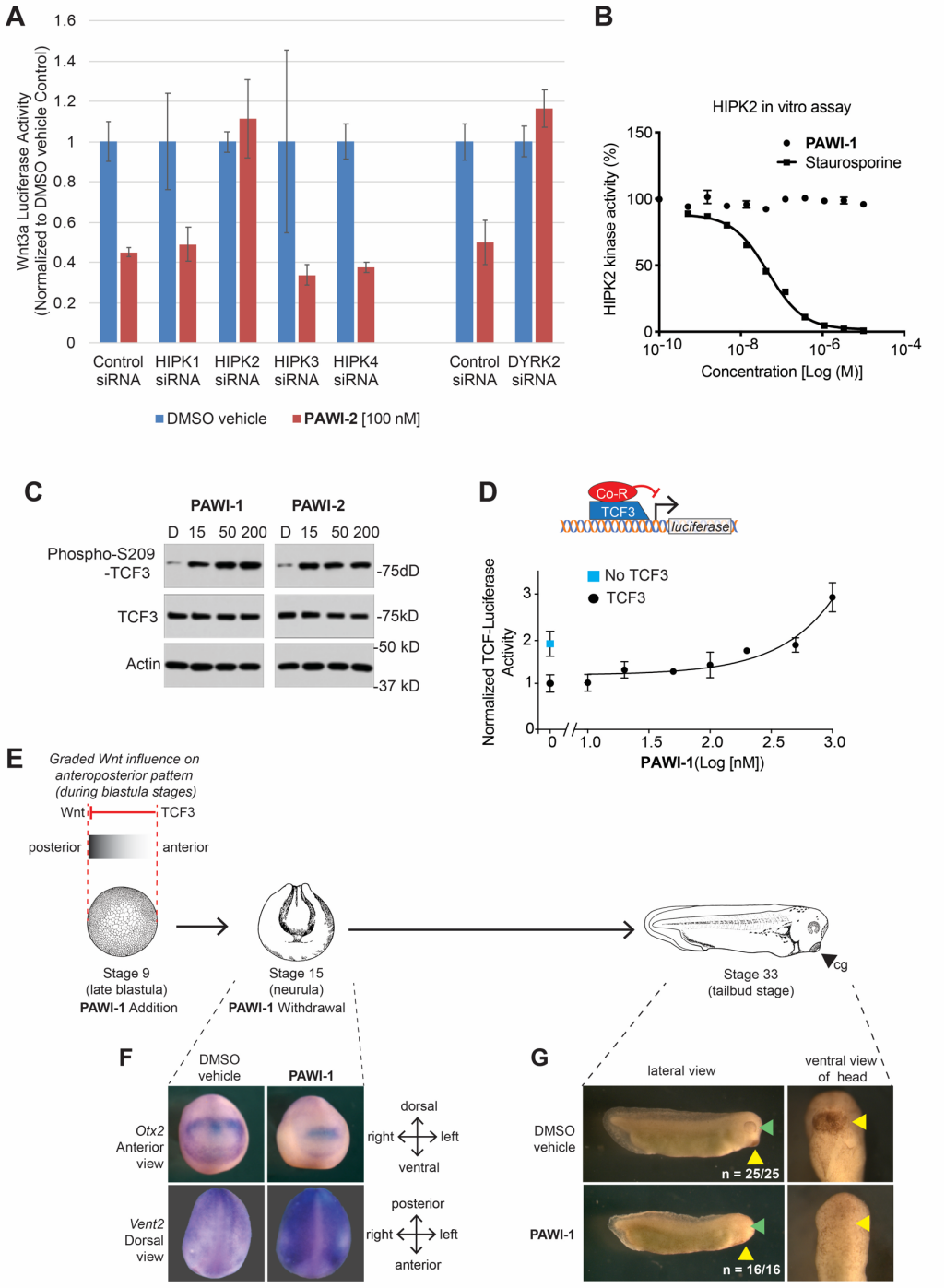



**A,B**) Effect of **PAWI-2** (500 nM) on time-dependent changes (**A**) and cellular localization (**B**) of β-catenin in SW480 cells. HDAC2 and GAPDH were used as markers of nuclear and cytosolic fractions.

**C**) Dose-dependent effect of **PAWI-2** (1.6 to 5000 nM) on the abundance of β-catenin in HCT-116 cells.

**D**) Effect of **PAWI-2** (100 nM) on the abundance of β-catenin in HCT-116, DLD-1, SW480 and 10.1 cells following 4 hour or 16 hour treatment.

## Supplementary Fig. S3. Effect on HIPK2 kinases and TCF3 (relevant to Fig. 3)




**A)** Effect of siRNAs against HIPK and DYRK kinases on PAWI-2 function in the Wnt3a luciferase assay. pBARLRen cells were seeded in 384-well plates and transfected 24 hours later with 10 nanomoles of either individual siRNAs (ON-TARGETplus, Dharmacon/GE Healthcare) or inert sequence control siRNA in triplicate, as in Methods. Two days later (to permit knockdown of target protein), recombinant Wnt3a (10% final concentration of Wnt3a conditioned medium)

together with either PAWI-2 (100 nM) or DMSO vehicle was added to each well. Firefly luciferase activities was measured 6 hours later using SteadyLite HTS (PerkinElmer). Luciferase activity for each siRNA was normalized to its respective DMSO control. Note that siRNAs against HIPK2 and the structurally related DYRK2 blocked the ability of PAWI-2 to inhibit Wnt3a luciferase activity. Data points are mean ± s.e.m., n=3

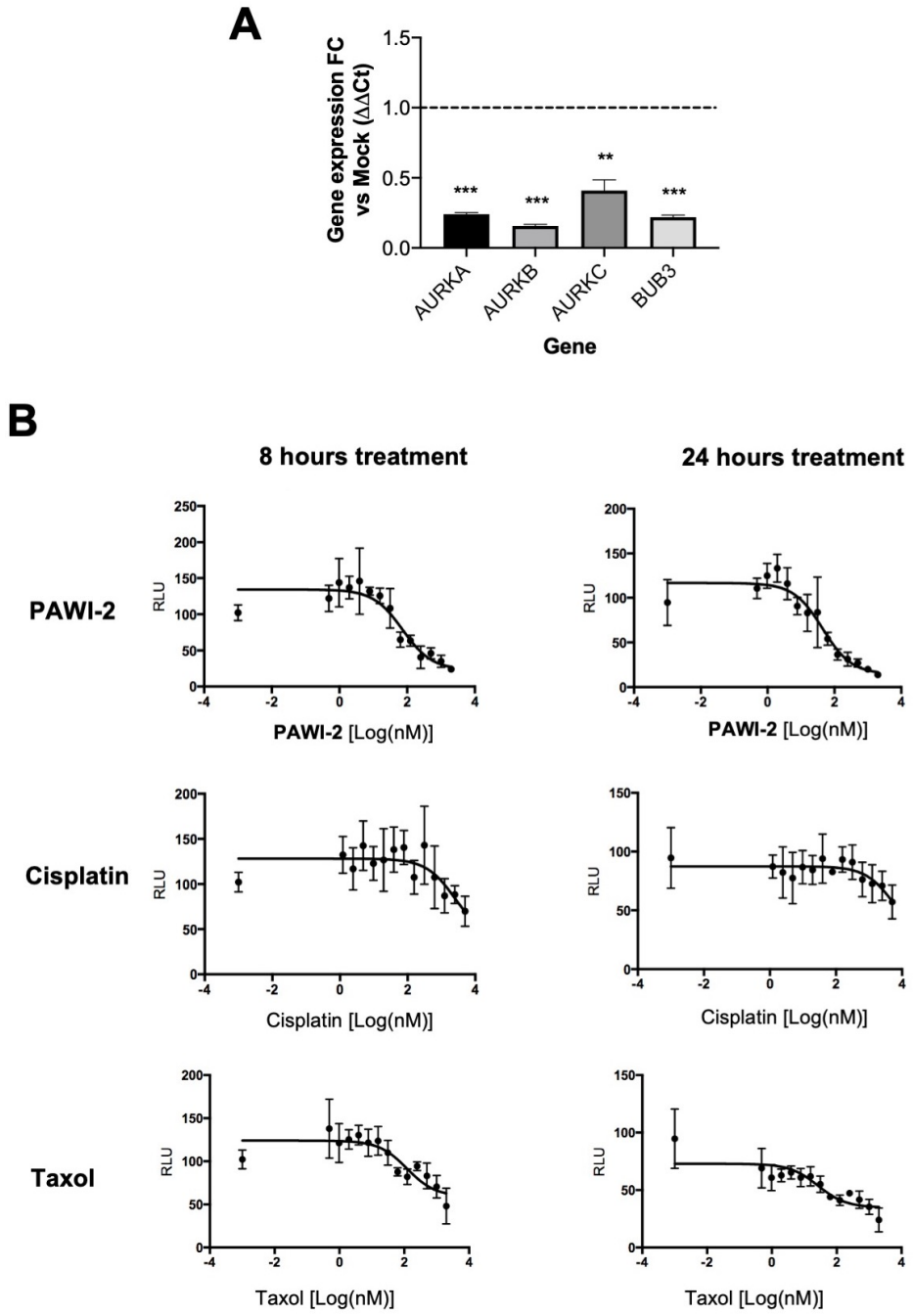
**B)** Kinase activity of purified HIPK2 assayed *in vitro* in response to **PAWI-1** and staurosporine**.** Data points are mean ± s.d, n=3 (in some cases error bars are smaller than the symbols).

**C)** Western blots showing the effect of **PAWI-1** and **PAWI-2** (500 nM, 24 hours) on abundance of TCF3 and phospho-S209-TCF3 [P4 antibody of ref. (Hikasa and Sokol, 2011)] proteins in HEK293T cells treated with compounds as indicated for 4 hours. Note no decrease in TCF3 abundance but increased phospho-TCF3 in response to **PAWI** treatment. **PAWI-2** data reproduced from Fig. 3B.

**D)** Effect of **PAWI-1** (200 nM) on TCF3 activity. TCF3 often acts as a repressor of Wnt signaling (Cadigan and Waterman, 2012; Hikasa et al., 2010), and the schematic illustrates the recruitment of co-repressors by TCF3 on target gene promoters. HEK293T cells were transfected with Super TOPFlash (Addgene) plasmid (containing a multimerized TCF response element and luciferase reporter) together and a HIPK2 expression plasmid together with a TCF3 expression plasmid as indicated. 24 hours after transfection, cells were treated with recombinant Wnt3A (10% conditioned media from Wnt3A overexpressing HEK293T cells) and **PAWI-1** at the indicated concentrations and luciferase was measured 24 hours later. ** indicates P-value <0.01 (T-test). Error bars = s.e.m. (n=3).

**E-G)** *In vivo* effect of **PAWI-1** on *Xenopus* development. Diagram **E** (upper) depicts the normal function of TCF3 to inhibit canonical Wnt signaling in the late blastula stage *Xenopus* embryo (Hikasa and Sokol, 2011). Experimental design (**C**, lower) shows *Xenopus* development from late blastula (stage 9) to neurula (stage 15) and tailbud (stage 33) stage embryos. **PAWI-1** (200 nM) was added to the water at stage 9 and the embryos continued development until stage 15, when the embryos were processed for *in situ* hybridization for mRNA expression (blue stain) at neurula (stage 15) with probes for the anterior marker *Otx2* and the ventral marker *Vent2* (**D**), or continued to culture until stage 33, when larvae were examined for formation of the cement gland, a pigmented structure at the anteriormost tip of the embryo (yellow arrowheads (**E**). Note that **PAWI-1** suppressed *Otx2* mRNA and increased expression and expanded the domain of *Vent2* mRNA, consistent with an inhibition of TCF3 function as in (Hikasa and Sokol, 2011). Lateral and ventral views of tailbud (stage 33) embryos (**E** shows the effect of **PAWI-1** on formation of the pigmented cement gland. Since cement glands are very anterior structures, their loss indicates that **PAWI-1** suppressed anterior cell fates. This activity was consistent with the inhibition of TCF3 function by **PAWI-1** (**D**).

## Supplementary Fig. S4. Effect on stress signaling (relevant to Fig. 4).




**A)** Confirmation of activity of siRNAs against sensor proteins evaluated in **Fig. 4D.** Data points are mean ± s.e.m.; n=3. **, $p<0.01$; ***, $p<0.001$ (T-test). FC, fold change.

**B)** Wnt response to treatment with Cisplatin and Taxol. pBARLRen cells (the same as used in the primary screen) were treated with **PAWI-2** (**Cmpd 2)**, Cisplatin and Taxol. Luciferase activity was determined at the indicated times. Data points are mean ± s.e.m., n=3.

# Supplementary Table S1. Small molecule screening data (relevant to Fig. 1)

| *Category* | *Parameter* | *Description* |
|---|---|---|
| ***Assay*** | Type of assay | Cell based reporter |
| | Target | Wnt/β-catenin dependent transcription |
| | Primary measurement | Firefly luciferase |
| | Key reagents | pBARLRen cell line (see Methods) |
| | Assay protocol | See Methods |
| | Additional comments | |
| ***Library*** | Library size | 76,000 |
| | Library composition | Drug-like small molecules selected for chemical diversity |
| | Source | Prebys Center for Drug Discovery at the Sanford-Burnham-Prebys Medical Discovery Institute (SBP) (https://www.sbpdiscovery.org/medical-discovery/drug-discovery/prebys-center-for-drug-discovery/overview) |
| | Additional comments | |
| ***Screen*** | Format | 1536 well format |
| | Concentration(s) tested | 5 μM in primary assay; |
| | Plate controls | Wnt3a alone (negative); IWR (positive) |
| | Reagent/ compound dispensing system | Labcyte acoustic dispenser |
| | Detection instrument and software | Envision plate reader (PerkinElmer) |
| | Assay validation/QC | Z' > 0.5; Signal/background >20 |
| | Correction factors | None |
| | Normalization | None in primary screen; normalization to *Renilla* luciferase in secondary screens |
| | Additional comments | See Methods |
| ***Post-HTS analysis*** | Hit criteria | ≥50% inhibition and Z-score ≥3 |
| | Hit rate | 0.24% (after primary)<br>0.13% (after confirmatory screen) |
| | Additional assay(s) | See scheme of secondary assays in Suppl Fig. 1.<br>**1)** Confirmatory screen: same as primary screen firefly luciferase was normalized to *Renilla* luciferase<br>**2)** Counter screen to eliminate inhibitors of NF-κB-dependent luciferase activity<br>**3)** Secondary screen to identify inhibitors of GSK3β inhibitor IX (BIO)-induced β-catenin responsive luciferase activity<br>**4)** Dose dependency against purified recombinant Wnt3a-dependent luciferase activity<br>**5)** Screen to confirm activity in a second cell type (HEK293T)<br>**6)** Screen to eliminate compounds that affect histone acetyltransferase (HAT)-dependent activity |
| | Confirmation of hit purity and structure | Resynthesis and analytical data are described in references (Cashman et al., 2016; Okolotowicz et al., 2018) |
| | Additional comments | See Methods |

## Supplementary Table S2. siRNA suppressor screen of 98 proteins involved in sensing and responding to DNA damage and mitotic checkpoint control (relevant to Figs. 4B and C).

RKO (pBARLRen) cells were plated at ~2400 per well of 384-well plates pre-spotted with 10 nanomoles of either individual siRNAs or inert sequence control siRNA (Dharmacon). Cells were cultured for 2 days to allow siRNA-mediated knockdown of cognate protein expression. Recombinant Wnt3a together with either **PAWI-2** (100 nM) or DMSO vehicle was added to each well and luciferase activity was determined 6 hours later. **PAWI-2** and DMSO vehicle conditions for each siRNA were tested in quadruplicate wells. **PAWI-2** activity was normalized relative to the corresponding DMSO control. The effect of each siRNA on **PAWI-2** activity was calculated as % inhibition relative to that observed in the control siRNA samples on each plate (0% inhibition). These activities were then expressed as a Z-score.

| *Gene Symbol* | *% inhibition of **PAWI-2*** | *% inhibition s.e.m* | *Z-score* | *z-score s.e.m.* |
|---|---|---|---|---|
| *Control siRNA.1* | 0.0 | 27.33 | 0.00 | 0.49 |
| *Control siRNA.2* | 0.0 | 8.96 | 0.00 | 0.38 |
| *ABL1* | -40.8 | 9.01 | -0.73 | 0.16 |
| *AP3B2* | 80.9 | 5.83 | 1.45 | 0.10 |
| *ARK5* | 69.1 | 21.05 | 1.24 | 0.38 |
| *AURKB* | 8.6 | 9.52 | 0.36 | 0.40 |
| *AURKC* | 35.8 | 8.63 | 1.52 | 0.37 |
| *BARD1* | 119.5 | 21.90 | 2.14 | 0.39 |
| *BIRC5/survivin* | 46.6 | 19.42 | 1.98 | 0.83 |
| *BLM* | -40.5 | 6.87 | -0.72 | 0.12 |
| *BRAT1* | 22.6 | 15.04 | 0.96 | 0.64 |
| *BRCA1* | 78.5 | 11.04 | 1.81 | 0.30 |
| *BUB1* | 17.8 | 22.44 | 0.75 | 0.95 |
| *BUB3* | 89.0 | 27.54 | 3.78 | 1.17 |
| *CDC20* | 37.9 | 25.96 | 1.61 | 1.10 |
| *CDC25A* | 132.4 | 20.88 | 2.37 | 0.37 |
| *CDC25C* | 46.3 | 10.65 | 0.83 | 0.19 |
| *CDCA1* | 2.5 | 8.92 | 0.11 | 0.38 |
| *CDCA8/borealin* | -12.0 | 7.42 | -0.51 | 0.32 |
| *CHK1* | 56.3 | 20.83 | 2.39 | 0.89 |
| *CHK2* | 3.9 | 7.42 | 0.17 | 0.32 |
| *CKN1* | 66.9 | 7.78 | 1.20 | 0.14 |
| *CUL4A* | -29.2 | 8.02 | -0.52 | 0.14 |
| *DCLRE1C* | 104.7 | 44.46 | 1.87 | 0.79 |
| *DDB1* | -5.6 | 14.32 | -0.10 | 0.26 |
| *DDB2* | 64.5 | 27.54 | 1.15 | 0.49 |

| *Gene Symbol* | *% inhibition of **PAWI-2*** | *% inhibition s.e.m* | *Z-score* | *z-score s.e.m.* |
|---|---|---|---|---|
| *DDX1* | -17.4 | 12.98 | -0.31 | 0.23 |
| *DYRK2* | 179.2 | 40.97 | 3.20 | 0.73 |
| *EEF1E1* | 64.0 | 16.21 | 1.09 | 0.40 |
| *ERCC1* | 56.3 | 3.78 | 1.01 | 0.07 |
| *ERCC2* | 51.4 | 9.00 | 0.92 | 0.16 |
| *ERCC3* | 27.0 | 22.50 | 0.48 | 0.40 |
| *ERCC4* | 46.6 | 26.14 | 0.83 | 0.47 |
| *ERCC5* | 78.3 | 24.14 | 1.40 | 0.43 |
| *ERCC6* | 159.0 | 12.73 | 2.84 | 0.23 |
| *EVI5* | -3.4 | 10.97 | -0.14 | 0.47 |
| *FANCD2* | 53.9 | 15.01 | 0.96 | 0.27 |
| *FRAP1* | 2.5 | 15.40 | 0.05 | 0.28 |
| *H2AFX* | 66.7 | 38.72 | 1.19 | 0.69 |
| *HIPK2* | 50.9 | 10.65 | 1.18 | 0.31 |
| *HMGN1* | 47.7 | 14.42 | 0.85 | 0.26 |
| *HTATIP* | 90.0 | 30.08 | 1.61 | 0.54 |
| *HUS1* | 136.5 | 5.11 | 2.44 | 0.09 |
| *INCENP* | 13.0 | 12.30 | 0.55 | 0.52 |
| *JTB* | 3.3 | 8.38 | 0.14 | 0.36 |
| *KIAA0683* | 67.7 | 12.28 | 1.21 | 0.22 |
| *KIAA1052* | 4.9 | 8.58 | 0.09 | 0.15 |
| *LIG4* | 50.1 | 27.52 | 0.90 | 0.49 |
| *Mad1* | 18.0 | 18.52 | 0.77 | 0.79 |
| *Mad2* | -36.5 | 1.96 | -1.55 | 0.08 |
| *MCAK* | 31.0 | 17.15 | 1.32 | 0.73 |
| *MDC1* | 34.4 | 22.22 | 0.35 | 0.54 |
| *MDM2* | 29.4 | 15.91 | 0.53 | 0.28 |
| *MGC2731* | 43.9 | 33.15 | 0.78 | 0.59 |
| *MPS1* | 60.4 | 21.81 | 2.57 | 0.93 |
| *MRE11* | 105.3 | 52.67 | 4.47 | 2.24 |
| *MRE11A* | 16.5 | 15.47 | 0.29 | 0.28 |
| *MYST1* | 89.6 | 15.96 | 1.60 | 0.29 |
| *NBS1* | -6.1 | 10.55 | -0.26 | 0.45 |
| *NBS1* | 31.3 | 3.07 | 0.56 | 0.05 |
| *NDC80* | 13.9 | 12.84 | 0.59 | 0.55 |
| *NUMA* | -14.2 | 9.87 | -0.60 | 0.42 |
| *p53/TP53* | 23.8 | 12.99 | 1.01 | 0.55 |
| *PARP1* | 44.1 | 15.42 | 0.79 | 0.28 |

| *Gene Symbol* | *% inhibition of **PAWI-2*** | *% inhibition s.e.m* | *Z-score* | *z-score s.e.m.* |
|---|---|---|---|---|
| *PCNA* | 33.9 | 10.53 | 0.61 | 0.19 |
| *PPM1D* | 178.3 | 23.62 | 3.19 | 0.42 |
| *PPP2R4* | 61.6 | 14.12 | 1.10 | 0.25 |
| *PPP5C* | 244.2 | 31.32 | 4.36 | 0.56 |
| *PRKDC* | 212.9 | 29.33 | 3.81 | 0.52 |
| *PSMA3* | -2.6 | 4.14 | -0.11 | 0.18 |
| *RACGAP1* | -14.8 | 8.79 | -0.63 | 0.37 |
| *RAD1* | 67.6 | 6.34 | 1.21 | 0.11 |
| *RAD17* | 160.3 | 24.16 | 2.87 | 0.43 |
| *RAD23B* | 7.6 | 2.88 | 0.14 | 0.05 |
| *RAD50* | 172.0 | 56.64 | 3.08 | 1.01 |
| *RAD51* | 124.4 | 18.21 | 2.22 | 0.33 |
| *RAD9A* | 147.5 | 32.10 | 2.64 | 0.57 |
| *RBBP8* | 77.8 | 44.50 | 1.39 | 0.80 |
| *RFC3* | 70.9 | 19.35 | 1.27 | 0.35 |
| *RHEB* | -32.9 | 14.63 | -0.59 | 0.26 |
| *RIT1* | 39.5 | 14.34 | 0.71 | 0.26 |
| *RNF168* | 72.2 | 2.90 | 1.29 | 0.05 |
| *RNF8* | 236.2 | 29.50 | 4.22 | 0.53 |
| *RPA1* | 22.9 | 9.51 | 0.41 | 0.17 |
| *RRM2B* | -5.1 | 22.27 | -0.09 | 0.40 |
| *SEPT1* | -23.5 | 4.18 | -1.00 | 0.18 |
| *SIAH1* | 109.5 | 9.19 | 4.66 | 0.39 |
| *SIAH2* | 10.3 | 15.45 | 0.44 | 0.66 |
| *SMC1* | 27.9 | 6.79 | 1.19 | 0.29 |
| *SMC1L1* | 201.5 | 13.17 | 3.60 | 0.24 |
| *SPDYC* | 28.1 | 8.36 | 1.19 | 0.36 |
| *TACC1* | 2.9 | 7.63 | 0.12 | 0.32 |
| *TERF1* | 20.7 | 3.06 | 0.37 | 0.05 |
| *TNKS* | 11.6 | 3.59 | 0.49 | 0.15 |
| *TOPBP1* | 150.7 | 22.11 | 2.69 | 0.40 |
| *TP53BP1* | 163.1 | 34.25 | 2.92 | 0.61 |
| *XAB2* | 83.2 | 13.30 | 1.49 | 0.24 |
| *XPA* | 94.8 | 39.16 | 1.69 | 0.70 |
| *XPC* | 58.1 | 6.52 | 1.04 | 0.12 |
| *XRCC4* | 73.8 | 17.85 | 1.32 | 0.32 |

## Supplementary Table S3. Oligonucleotide Primers (relevant to Figs. 1 and 2).

| Table S3. Oligonucleotide Primers | | | |
|---|---|---|---|
| **Gene name** | **NCBI accession** | **Purpose** | **Sequence** |
| **qRT-PCR** | | | |
| GAPDH | NM_002046 | Reference | F: GGAGCGAGATCCCTCCAAAAT<br>R: GGCTGTTGTCATACTTCTCATGG |
| ACTA2 | NM_001613 | Muscle marker | F: CAGGGCTGTTTTCCCATCCAT<br>R: GCCATGTTCTATCGGGTACTTC |
| AXIN2 | NM_004655 | Wnt core pathway component | F: TACACTCCTTATTGGGCGATCA<br>R: TTGGCTACTCGTAAAGTTTTGGT |
| CCND1 | NM_053056 | Wnt core pathway component | F: GCTGCGAAGTGGAAACCATC<br>R: CCTCCTTCTGCACACATTTGAA |
| cMYC | NM_002467 | Wnt/β-catenin target gene | F: GTCAAGAGGCGAACACACAAC<br>R: TTGGACGGACAGGATGTATGC |
| **ChIP qPCR** | | | |
| Ctrl Primers (Untr12, | Active Motif | corresponds to a gene desert on chromosome 12 | Cat# 71001 |
| AXIN2 +66 | Gene ID: 8313 | Wnt core pathway component | GGCTGCGCTTTGATAAGGT<br>TTTTTATTTCCCGGCTCTCG |
| NKD1 -246 | GeneID: 85407 | Wnt core pathway component | CCTGCCCGTTTAGAGAGAAC<br>GAGGCGTATTGGGACAGAAC |
| MYC +132 | GeneID: 4609 | Wnt core pathway component | GCGTTGCTGGGTTATTTTAATC<br>CAGAGCGTGGGATGTTAGTG |
| MSX1 -586 | GeneID: 4609 | Wnt core pathway component | AAGGCCCACTTTTACCTCGA<br>CGAAACAAAACCAAACCTTCA |
| MSX2 -199 | GeneID: 4488 | Wnt core pathway component | TTAGGTCTCGGGCTTTTCAG<br>AGGAGCAGTCAGCAGAGTTG |
| DKK1 +105 | GeneID: 22943 | Wnt core pathway component | CACCCAAGTTCCCAGAGTTC<br>CAGCGAGCGTTATAGCAGAC |
| CCND1 +105 | GeneID: 595 | Wnt core pathway component | AGGGGAGTTTTGTTGAAGTTG<br>ACTCTGCTGCTCGCTGCTAC |
| AXIN2 +280 | Gene ID: 8313 | Wnt core pathway component | TCGCAAGAACTGCAAGCAAG<br>AGTCCTCCAAGCCCAAATTC |
| CLDN1 +472 | GeneID: 595 | Wnt core pathway component | TCGTTCCATCACTTGTCTCTG<br>TCTGGGTCTGTTTCCCAATC |
| TCF7 -153 | GeneID: 6932 | Wnt core pathway component | GTAAGCGGGGTCAGGAGTTC<br>GGACAAGCCTGGGAATGATC |

## Supplementary Table S4. Taqman Primers (relevant to Figs. 2 and 4).

| **Table 2: Taqman primers for qRT-PCR** | | |
|---|---|---|
| **Gene name** | **Purpose** | **Assay ID** |
| GAPDH | reference | Hs02786624 |
| 18S | reference | Hs99999901 |
| ADAMTS18 | Wnt/β-catenin target gene | Hs00373501 |
| AXIN2 | Wnt/β-catenin target gene | Hs00610344 |
| CCND1 | Wnt/β-catenin target gene | Hs00765553 |
| CLDN1 | Wnt/β-catenin target gene | Hs00221623 |
| DKK1 | Wnt/β-catenin target gene | Hs00183740 |
| MSX2 | Wnt/β-catenin target gene | Hs00427183 |
| cMYC | Wnt/β-catenin target gene | Hs00153408 |
| NKD1 | Wnt/β-catenin target gene | Hs01548773 |
| TCF7 | Wnt/β-catenin target gene | Hs01556515 |
| BAX | p53 target gene | Hs00180269 |
| BAX | p53 target gene | Hs00180269 |
| CDKN1A15 | p53 target gene | Hs00355782 |
| P53AIP1 | p53 target gene | Hs00986095 |
| PTEN | p53 target gene | Hs01628827 |