Figure S1

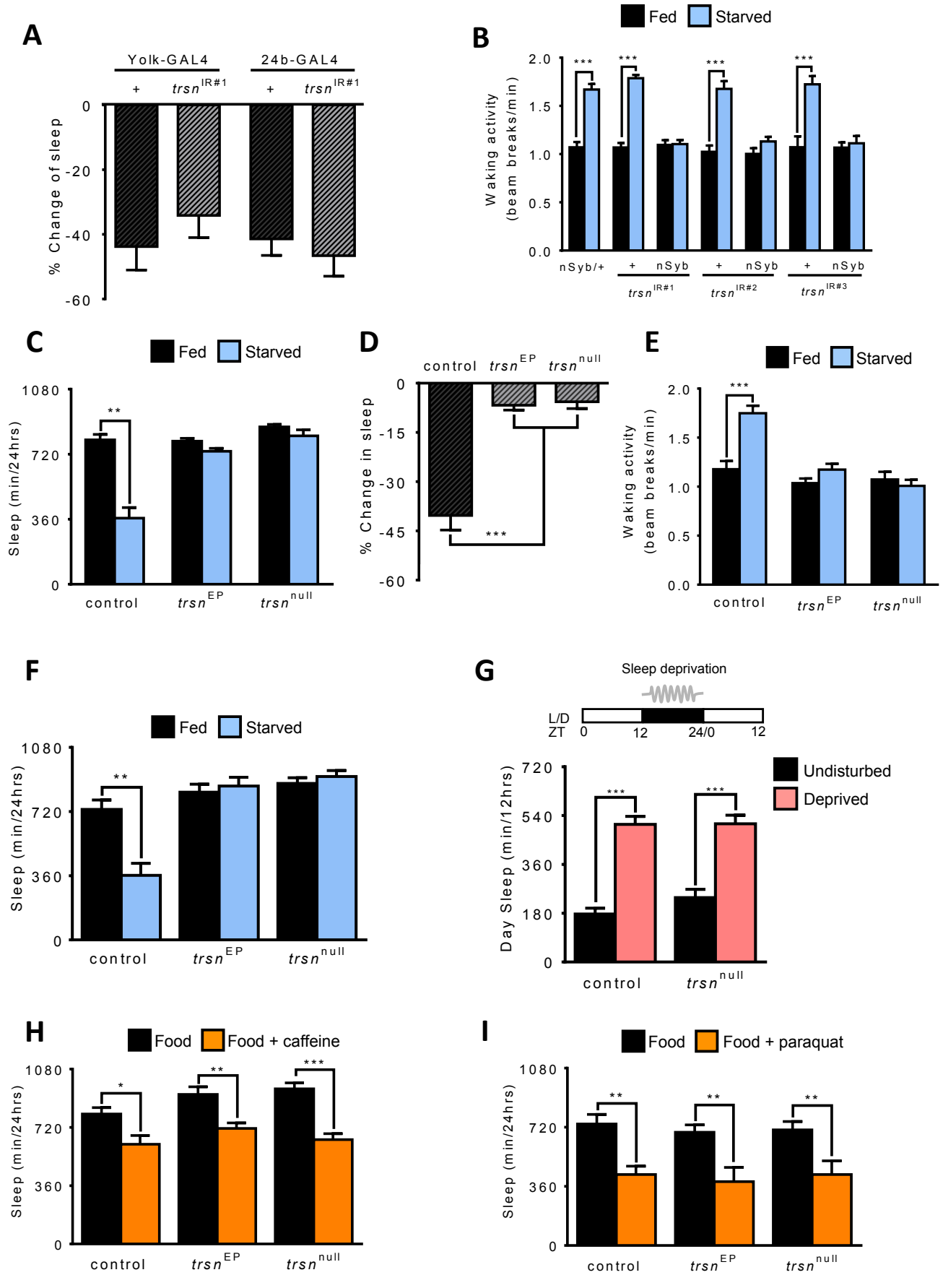
A
Yolk-GAL4
24b-GAL4
+
trsn IR#1
% Change of sleep
B
Fed
Starved
Waking activity (beam breaks/min)
nSyb/+
nSyb
trsn IR#1
trsn IR#2
trsn IR#3
C
Sleep (min/24hrs)
control
trsn EP
trsn null
D
% Change in sleep
E
F
G
Sleep deprivation
L/D
ZT
Undisturbed
Deprived
Day Sleep (min/12hrs)
H
Food
Food + caffeine
I
Food + paraquat

Figure S2

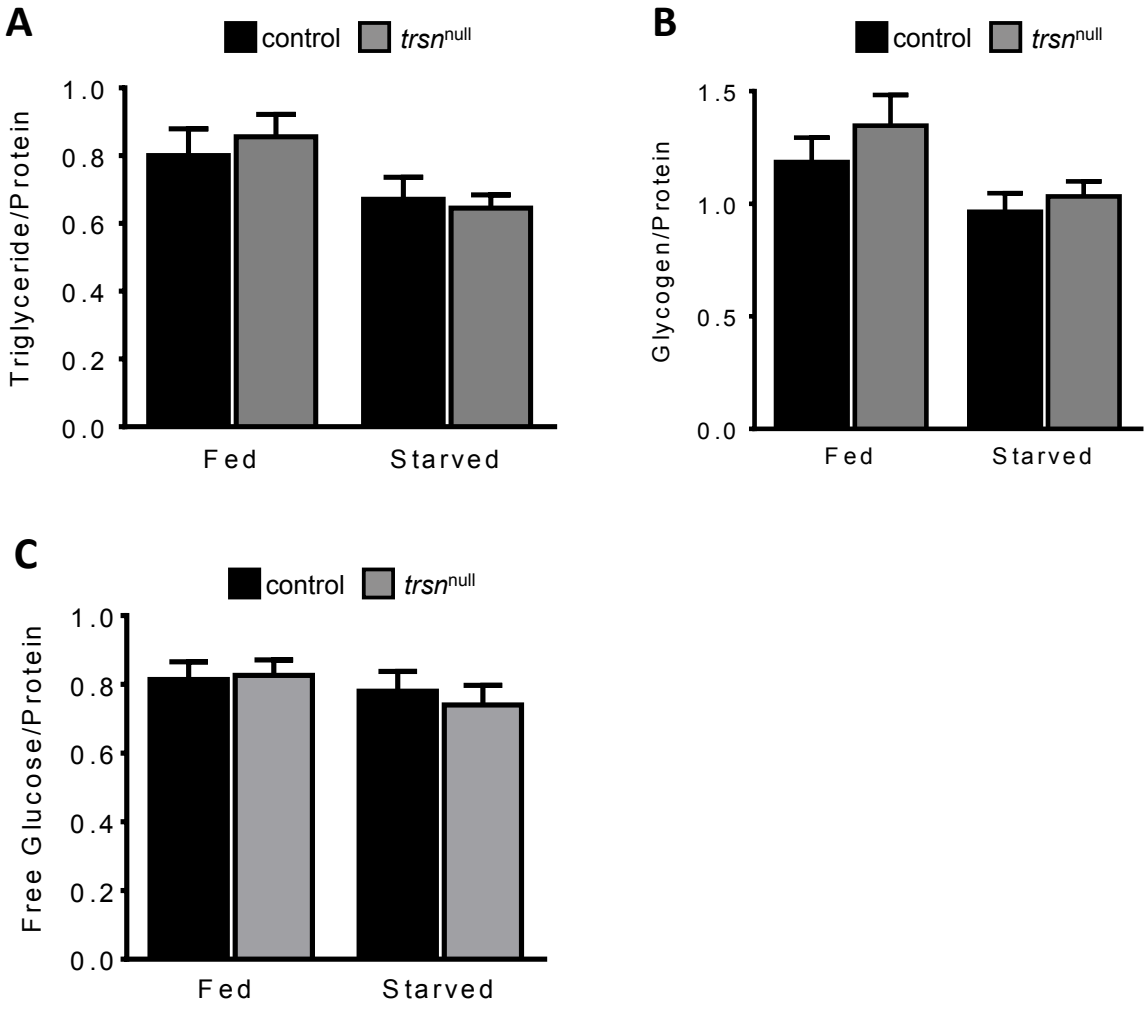
A
control
trsn^null
Triglyceride/Protein
1.0
0.8
0.6
0.4
0.2
0.0
Fed
Starved
B
control
trsn^null
Glycogen/Protein
1.5
1.0
0.5
0.0
Fed
Starved
C
control
trsn^null
Free Glucose/Protein
1.0
0.8
0.6
0.4
0.2
0.0
Fed
Starved

Figure S3

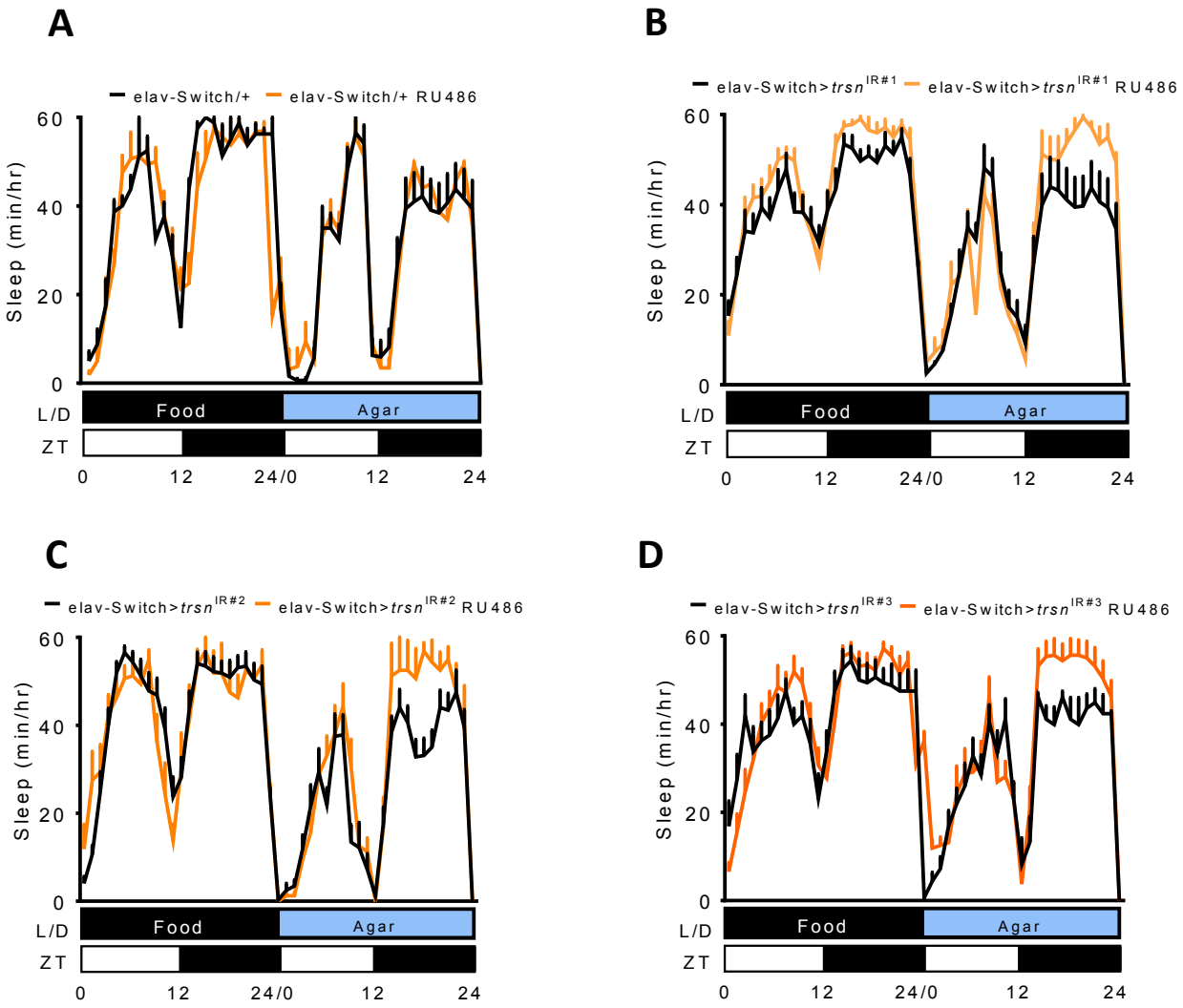
A
elav-Switch/+
elav-Switch/+ RU486
Sleep (min/hr)
60
40
20
0
L/D
Food
Agar
ZT
0
12
24/0
12
24
B
elav-Switch>trsn IR#1
elav-Switch>trsn IR#1 RU486
Sleep (min/hr)
60
40
20
0
L/D
Food
Agar
ZT
0
12
24/0
12
24
C
elav-Switch>trsn IR#2
elav-Switch>trsn IR#2 RU486
Sleep (min/hr)
60
40
20
0
L/D
Food
Agar
ZT
0
12
24/0
12
24
D
elav-Switch>trsn IR#3
elav-Switch>trsn IR#3 RU486
Sleep (min/hr)
60
40
20
0
L/D
Food
Agar
ZT
0
12
24/0
12
24

Figure S4

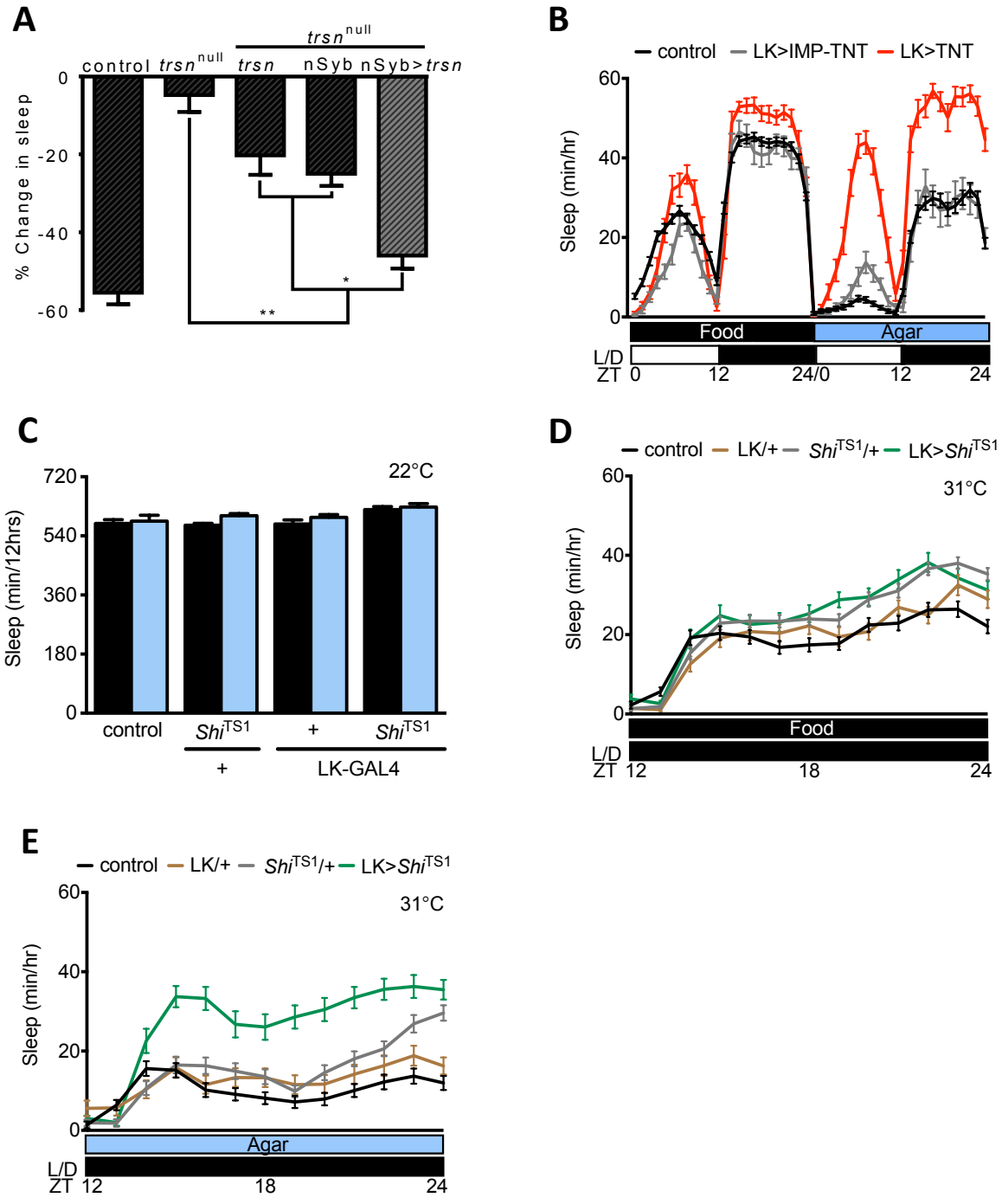
A
trsn null
control
trsn null
trsn
nSyb
nSyb>trsn
% Change in sleep
0
-20
-40
-60
*
**
B
control
LK>IMP-TNT
LK>TNT
Sleep (min/hr)
60
40
20
0
Food
Agar
L/D
ZT 0
12
24/0
12
24
C
22°C
Sleep (min/12hrs)
720
540
360
180
0
control
Shi TS1
+
Shi TS1
+
LK-GAL4
D
control
LK/+
Shi TS1/+
LK>Shi TS1
31°C
Sleep (min/hr)
60
40
20
0
Food
L/D
ZT 12
18
24
E
control
LK/+
Shi TS1/+
LK>Shi TS1
31°C
Sleep (min/hr)
60
40
20
0
Agar
L/D
ZT 12
18
24

Figure S1 related to Figure 1: Characterization of sleep in *trsn* deficient flies.

A. Percentage sleep loss in flies expressing *trsn*^{IR} in the fat body (yolk-GAL4) or muscle (24b-GAL4). No significant differences are observed between *trsn* knockdown and control flies harboring GAL4 alone (N≥11; P>0.34 for both groups). **B.** Average waking activity (beam breaks/waking minute) in fed (black) and starved (blue) flies over 24 hours. Waking activity in fed flies does not differ between any genotypes (N≥36; P>0.98). Under starved conditions, waking activity is increased in *nSyb*-GAL4/+ and *trsn*^{IR}/+ control flies (N≥36; P<0.001), while no change in waking activity is detected in each of the three *nSyb*-GAL4>*trsn*^{IR} knockdown lines (N≥36; P>0.91). **C.** In male flies, sleep is significantly reduced in starved w^{1118} controls (N=39; P<0.01), while sleep duration of *trsn*^{EP} (N=45; P<0.76) and *trsn*^{null} flies (N=48; P>0.82) does not significantly differ on food and agar. **D.** Percentage change in sleep from fed to starved conditions in male flies show sleep loss is significantly greater in control flies compared to *trsn*^{EP} and *trsn*^{null} flies (N≥39, P<0.001). **E.** Average waking activity in fed (black) and starved (blue) flies over 24 hours. Waking activity in fed flies does not differ between any genotypes (N≥54; P<0.66). Waking activity during starvation is increased in control (N=54; P<0.001) and *trsn*^{EP} flies (N=69; P>0.66), while there is no effect of starvation on waking activity in *trsn*^{null} flies (N=68; P>0.98). Waking activity of starved *trsn*^{EP} flies is reduced compared to controls suggesting a blunted locomotor response to starvation. **F.** Video tracking analysis of sleep in fed and starved flies. In control flies, sleep is significantly reduced in fed control (black) compared to starved control (blue, N≥37; P<0.001), while no significant differences are observed in fed *trsn*^{EP} or *trsn*^{null} mutant flies (N≥39; P>0.99). **G**. Daytime sleep from ZT0-ZT12 is significantly greater following mechanical sleep deprivation for 12 hours from ZT12-ZT24 (pink) compared to undisturbed flies (black) for w^{1118} control (N=32; P<0.001) and *trsn*^{null} genotypes (N=32; P<0.001). Total sleep does not differ between sleep-deprived control and *trsn*^{null} (N=32; P>0.43) flies or undisrupted

control and *trsn*^{null} (N=32, P>0.23) flies from ZT0-ZT12. **H.** Sleep is reduced in control (N=32; P<0.05), *trsn*^{EP} (N=32; P<0.01) and *trsn*^{null} (N=32; P<0.001) flies fed food containing caffeine (orange) compared to flies fed standard fly food (black). **HI** Sleep is reduced in control (N=32; P<0.01), *trsn*^{EP} (N=32; P<0.01), and *trsn*^{null} (N=32; P<0.01) fed paraquat (orange) compared to flies fed standard fly food (black). All error bars are mean ± SEM. * denotes P<0.05*, ** denotes P<0.01, *** dentotes P<0.001,*** by two-way ANOVA.

Figure S2 related to Figure 2: Energy stores and free glucose are normal in *trsn* mutant flies.

A. Triglyceride levels did not differ between control (black) and *trsn*^{null} (grey) in the fed (N=20; P>0.92) or starved state (N=20; P>0.99). **B.** Glycogen levels did not differ between control (black) and *trsn*^{null} (grey) in the fed (N=16; P>0.67) or starved state (N=16; P<0.96). **C.** Free glucose did not differ between control (black) and *trsn*^{null} (grey) in the fed (N≥16; P>0.75) nor starved state (N≥13; P>0.81). All bars are mean ± SEM by two-way ANOVA.

Figure S3 related to Figure 3: Adult-specific knockdown of *trsn* disrupts sleep suppression.

A. Sleep profiles depicting hourly sleep averages over a 48 hour experiment. Flies are placed on food for day 1, then transferred to agar for day 2. Flies harboring elav-Switch alone with RU486 treatment (orange) or elav-Switch alone without treatment (black). **B-D.** Sleep on agar is greater in expreimental flies (elav-Switch>*trsn*^{IR}; orange) fed RU486 compared to genotype-matched controls without drug treatment (black).

Figure S4, related to Figure 4. LK neurons are acutely required for starvation-induced sleep suppression.

A. Expression of UAS-*trsn* under control *nSyb*-GAL4 in the background of a *trsn*^{null} mutation restores starvation-induced sleep suppression compared to flies harboring either UAS-*trsn* (N≥37; P<0.05) or the GAL4 line alone (N≥82;P<0.01). No significant difference was detected between *nSyb* rescue and control flies (N≥37; P>0.66) . **B.** Sleep profile over 48 hours reveals that sleep in LK-GAL4>UAS-TNT (red) flies is moderately increased compared to *w*^{1118} control flies (black) or flies expressing inactive IMP-TNT (grey) for day one on food. Sleep in LK-GAL4>UAS-TNT is significantly greater for day two on agar compared to control and IMP-TNT-expressing flies. **C.** No significant differences for sleep duration on food (black) or agar (blue) were detected for any of the genotypes tested when flies were housed at 22°C (Fed vs Starved: control, N≥38, P=0.99; UAS- *Shi*^{TS}/+; N≥87, P=0.83, LK-GAL4/+, N=26, P=0.97, LK-GAL4>UAS- *Shi*^{TS}; N≥23, P=0.99). **D.** Sleep profile over 12 hours on food at 31°C reveals that sleep in LK-GAL4>UAS-*Shi*^{TS} (green) flies does not differ from *w*^{1118} controls (black) or respective heterozygote controls (brown/grey). **E.** Sleep profile over 12 hours on agar at 31°C reveals that sleep suppression in LK-GAL4>UAS-*Shi*^{TS} (green) sleep significantly more than *w*^{1118} controls (black) and heterozygote controls (brown/grey). All columns are mean ± SEM; P<0.01,**; P<0.001,*** by 2-way ANOVA.

Supplemental Experimental Procedures

Drosophila maintenance and Fly Stocks

The *trsn*-RNAi lines are from the Vienna *Drosophila* Resource Center [S1]. The RNAi lines have been renamed from original transformant identifiers as follows: *trsn*-IR$^{\#1}$ (GD9963), *trsn*-IR$^{\#2}$ (GD9964) and *trsn*-IR$^{\#3}$ (108456). The *trsn*^{EP} line is the EPgy2 insertion *trsn*^{EY06981} and has previously been characterized [S2–S4]. The *trsn*^{null} allele is an excision of the *trsn*^{EY06981} locus derived from mobilizing the EPgy2 insertion in the *w*^{1118} background that has been previously described [S3]. This allele removes the entire coding region of the gene and likely represents a null mutation. It has previously been described as Δ*trsn* [S3]. The *LK*-GAL4 line is a promoter fusion of 3.6 kb upstream of LK, cloned in the laboratory of YJK with a similar expression pattern to a previously described line [5].. The lines UAS-TNT and UAS-Shi^{TS1} have previously been described [7, 8]. The UAS-mCD8::GFP (32184; [S6]) and UAS-GFP.nls (32184; [S9]) transgenes have previously been described and were obtained from Bloomington. The UAS-*trsn* transgene was generated by amplifying from GM27569 clone into a PhiC31 vector at the attP86Fb docking site on the 3rd chromosome by Zoltan Astolos (Aktogen, Cambridge, UK). Three to five day old mated female flies were used for all experiments in this study, except when noted.

Behavioral Analysis

The DAM system detects activity by monitoring infrared beam crossings for each animal. These data were used to calculate sleep information by extracting immobility bouts of 5 minutes using the *Drosophila* Sleep Counting Macro [S10]. For experiments examining the effects of starvation on sleep, activity was recorded for one day on food, prior to transferring flies into tubes containing 1% agar (Fisher Scientific) at ZT0 and activity was

monitored for an additional 24 hours. Change in sleep during starvation or dietary manipulation was calculated as ((sleep duration (mins) experimental-sleep duration (mins) baseline)/(sleep duration (mins) baseline))*100 as previously described [S11]. For experiments employing thermogenetic manipulation of LK neurons, only nighttime sleep was analyzed because flies were unable to survive 24 hours of starvation at elevated temperatures. Following 24 hours of acclimation, baseline sleep was measured on food at 22°C from ZT12-ZT24. On the following day at ZT8 flies were transferred to new tubes containing either standard fly food (control) or 1% agar. The temperature was increased to 31°C at ZT12 and activity was recorded through ZT24.

For tracking analysis, fly activity was recorded using a custom video acquisition system [S12]. Flies were anesthetized using cold-shock and loaded into standard 24-well tissue culture plates (BD Biosciences 351147), with each well containing either 5% sucrose dissolved in 1% agar (fed group) or 1% agar alone (starved group). The sucrose diet was required as standard fly food is opaque and prevents efficient tracking. The plates were placed in a chamber illuminated with white (6500K) LED lights (Environmental Lights Inc. product no. dlrf3528-120-8-kit) on a 12:12 LD cycle, and with constant illumination from 850-880nm infra-red (IR) lights (Environmental Lights Inc., product no. irrf850-390). Video was recorded using an ICD-49 camera (Ikegami Tsushinki Co., Japan) fitted with an IR-transmitting lens (Computar Inc., Vari Focal H3Z4512 CS-IR 4.5-12.5 mm F 1.2 TV lens). An IR high-pass filter (Edmund Optics Worldwide, filter optcast IR 5x7 in. part no. 46,620) was placed between the camera and the lens to block visible light. Video was recorded at a resolution of 525 lines at 59.94 Hz, 2:1 interlace. Fly activity was analyzed using Ethovision XT 9.0 video tracking software (Noldus Inc.). Sleep was calculated by

measuring bouts of inactivity ≥5 minutes using a previously described Microsoft Excel macro [S12].

For sleep deprivation experiments, flies were shaken in DAM2 monitors every 3-4 minutes for 12 hours from ZT12 (onset of darkness) through ZT0 (onset of light) as previously described [S13]. Stimulus was applied using a vortexer (Fisher Scientific, MultiTube Vortexer) with a custom milled plate to hold DAM2 monitors and a repeat cycle relay switch (Macromatic, TR63122). Sleep rebound was measured the following day from ZT0-ZT12.

Proboscis Extension Reflex (PER)

Three to five day old flies were collected and placed on fresh food for 24 hours, then starved for the designated period of time in vials containing wet Kimwipe paper (Kimberly-Clark Corporation). Flies were then anaesthetized under CO_2, and their thorax and wings were glued with nail polish (Electron Microscopy Science) to a microscopy slide, leaving heads and legs unconstrained. Following 3-6 hours recovery in a humidified chamber, the slide was mounted vertically under the dissecting microscope (Leica, S6E) and PER was observed. PER induction was performed as described previously [S14]. Briefly, flies were satiated with water before and during experiments. Flies that did not water satiate within 5 minutes were excluded from the experiment. A 1 ml syringe (Tuberculin, BD&C) with an attached pipette tip (TipOne) was used for tastant presentation. Tastant was manually applied to tarsi for 2-3 seconds 3 times with 10 second inter-trial intervals, and the number of full proboscis extensions was recorded. Tarsi were then washed with distilled water between applications of different tastants and flies were allowed to drink water during the experiment *ad libitum*. Each fly was assayed for response to multiple tastants. PER response was calculated as a percentage of proboscis extensions to total number of tastant stimulations to tarsi.

Blue dye assay

Short-term food intake was measured as previously described [S15]. Briefly, flies were starved for 24 or 48 hours on wet Kimwipes or maintained on standard fly food. At ZT0 flies were then transferred to food vials containing 1% agar, 5% sucrose, and 2.5% blue dye (FD&C Blue Dye No. 1). Following 30 minutes of feeding flies were flash frozen on dry ice and individually homogenized in 400 µL PBS (pH 7.4, Ambion). Color spectrophotometry was then used to measure absorbance at 655 nm in a 96-well plate reader (Millipore, iMark). Baseline absorbance was determined by subtracting the absorbance measured in non-dye fed flies from each experimental sample.

Capillary Feeder assay (CAFE)

A modified volumetric drinking assay was used to test food consumption [S16] as previously described [S13]. Female flies were allowed to feed on a tube containing 100mM sucrose or 5% yeast extract in water, while a second capillary tube provided access to water alone (WPI, #1B150F-4 ID 1mm, OD 1.5mm, with filament). The capillary tubes were inserted into an empty food vial at a 90° angle and vials were placed at a 45° angle. The openings of the capillaries were aligned with the ceiling of the vial. Following 24 hours of fasting, 30-60 female flies were placed into a vial and food consumption was measured. The volume consumed was calculated as the length of liquid missing from the capillary multiplied by the cross-section of the inner diameter of the capillary. All measurements were adjusted for missing liquid due to evaporation using control capillary tubes without flies. Consumption was measured every hour following the introduction of flies into the assay. Taste compounds were mixed with Allura red food dye (FD&C red #40) to a concentration of 3µl per 1ml dilution for better

visibility in the capillary tube. Following the conclusion of the assay flies were anaesthetized and the number of flies in each vial was counted. Total consumption per fly was measured as volume consumed in each capillary divided by number of live flies in the vial.

Pharmacological manipulation

Crosses for RU486 experiments were raised at room temperature in normal fly food vials then transferred to individual DAM tubes containing 0.25mM RU486; the flies were acclimated in the DAM monitor for 24 hours. On experimental day 1, sleep was recorded. On day 2, flies were flipped to DAM tubes containing 1% agar and 0.25mM RU486; % sleep was then recorded. RU486 effects during the experiment were calculated by comparing the amount of sleep during the baseline night (without drug) with that during the treatment night. For parquat experiments, DAM tubes were made similarly. Both w^{1118} controls and *trsn* mutant flies were raised at room temperature in normal fly food vials then transferred to individual DAM tubes containing 1mM paraquat dichloride. The flies were acclimated in the DAM monitor for 24 hours before treatment. Sleep was measured for 5 days under standard light/dark cycles and percent sleep was monitored. For caffeine experiments, both w^{1118} and *trsn* mutant flies were raised at room temperature in normal fly food vials and then transferred to individual DAM tubes containing standard food. The flies were acclimated in the DAM monitor for 24 hours. On experimental day 1, sleep was recorded. On day 2, flies were flipped to DAM tubes containing 4mg/mL caffeine and percent sleep was recorded. Caffeine effects during each experiment were calculated by comparing the amount of sleep during the baseline night (without drug) with that during the treatment night.

Protein, glycogen, and triglyceride measurements

Protein glucose and triglyceride measurements were performed as previously described [S17, S18].Two female flies aged 3-5 days were homogenized in 50 mM Tris-HCl, pH 7.4, 140 mM NaCl, 0.1% Triton-X, and 1X protease inhibitor cocktail (Sigma Aldrich, P8340). Triglyceride concentration was measured using the Stanbio Liquicolor Kit (Boerne, TX), and protein concentrations were measuring using a BCA Protein Assay Kit (Pierce Scientific). Total glucose levels were determined using the Glucose Oxidase Reagent (Pointe Scientific) in samples previously treated with 8mg/mL amyloglucosidase in 0.2M Sodium Citrate buffer, pH 5.0 (Boston BioProducts). Free glucose was measured in samples not treated with amyloglucosidase and then glycogen concentrations were determined by subtracting the free glucose from total glucose concentration. Both glycogen and triglyceride concentrations were standardized to the total protein content of each sample containing two flies.

Quantitative RT-PCR

For qPCR experiments 3-5 day old female flies were sacrificed at ZT0 and flash-frozen on dry ice. Heads and bodies were separated by vortexing and manually isolated. The primers used were: *trsn* (F-5'GCTCCGCCTTCTCCAGATACT3' and R-5'CCGCCTCCAGGTAAATAACCA3'), actin 5C (F-5'AGCGCGGTTACTCTTTCACCAC3') and R-5'GTGGCCATCTCCTGCTCAAAGT3'), and β-tubulin (F-5'GCAGTTCACCGCTATGTTCA3' and R-5'CGGACACCAGATCGTTCAT3'). Triplicate measurements were conducted for each sample. Primers were purchased from IDT technologies.

Immunohistochemistry

Fly brains were dissected in ice-cold PBS and fixed in 4% formaldehyde, PBS, 0.2% Triton-X 100 for 30 minutes. Brains were rinsed 3X with PBS, Triton-X for 10 minutes

and incubated overnight at 4°C in 1:4 anti-ELAV*, 1*:20 NC82 ([S19] Iowa Hybridoma Bank) and 1:1000 anti-TRSN [3]. The brains were rinsed again in PBS-Triton X, 3X for 10 minutes and placed in secondary antibodies (Goat anti-Mouse 555, and Goat anti-rabbit 488; Life Technologies) for 90 minutes at room temperature. The brains were mounted in Vectashield (VectorLabs) and imaged on a Leica SP8 confocal microscope. Brains were imaged in 2μm sections and are presented as the Z-stack projection through the entire brain. For quantification of whole-brain TRSN levels, the entire brain was imaged in 2μm sections, merged into a single Z-stack as maximum fluorescence, and the total brain fluorescence was determined. For experiments examining co-localization, each channel was imaged separately, and the absence of bleed through was validated.

Supplemental References

S1. Dietzl, G., Chen, D., Schnorrer, F., Su, K.-C., Barinova, Y., Fellner, M., Gasser, B., Kinsey, K., Oppel, S., Scheiblauer, S., et al. (2007). A genome-wide transgenic RNAi library for conditional gene inactivation in Drosophila. Nature *448*, 151–156.

S2. Bellen, H. J., Levis, R. W., Liao, G., He, Y., Carlson, J. W., Tsang, G., Evans-Holm, M., Hiesinger, P. R., Schulze, K. L., Rubin, G. M., et al. (2004). The BDGP gene disruption project: Single transposon insertions associated with 40% of Drosophila genes. Genetics *167*, 761–781.

S3. Claußen, M., Koch, R., Jin, Z. Y., and Suter, B. (2006). Functional characterization of Drosophila Translin and Trax. Genetics *174*, 1337–1347.

S4. Suseendranathan, K., Sengupta, K., Rikhy, R., D'Souza, J. S., Kokkanti, M., Kulkarni, M. G., Kamdar, R., Changede, R., Sinha, R., Subramanian, L., et al. (2007). Expression pattern of Drosophila translin and behavioral analyses of the mutant. Eur. J. Cell Biol. *86*, 173–186.

S5. Al-Anzi, B., Armand, E., Nagamei, P., Olszewski, M., Sapin, V., Waters, C., Zinn, K., Wyman, R. J., and Benzer, S. (2010). The leucokinin pathway and its neurons regulate meal size in Drosophila. Curr. Biol. *20*, 969–978.

S6. Pfeiffer, B. D., Jenett, A., Hammonds, A. S., Ngo, T.-T. B., Misra, S., Murphy, C., Scully, A., Carlson, J. W., Wan, K. H., Laverty, T. R., et al. (2008). Tools for neuroanatomy and neurogenetics in Drosophila. Proc. Natl. Acad. Sci. U. S. A. *105*, 9715–9720.

S7. Sweeney, S. T., Broadie, K., Keane, J., Niemann, H., and O'Kane, C. J. (1995). Targeted expression of tetanus toxin light chain in Drosophila specifically eliminates synaptic transmission and causes behavioral defects. Neuron *14*, 341–351.

S8. Kitamoto, T. (2001). Conditional modification of behavior in drosophila by targeted expression of a temperature-sensitive shibire allele in defined neurons. J. Neurobiol. *47*, 81–92.

S9. Yasunaga, K., Saigo, K., and Kojima, T. (2006). Fate map of the distal portion of Drosophila proboscis as inferred from the expression and mutations of basic patterning genes. Mech. Dev. *123*, 893–906. Available at: http://www.ncbi.nlm.nih.gov/pubmed/17027238.

S10. Pfeiffenberger, C., Lear, B. C., Keegan, K. P., and Allada, R. (2010). Processing sleep data created with the Drosophila Activity Monitoring (DAM) System. Cold Spring Harb. Protoc. *2010*, pdb.prot5520.

S11. Keene, A. C., Duboué, E. R., McDonald, D. M., Dus, M., Suh, G. S. B., Waddell, S., and Blau, J. (2010). Clock and cycle limit starvation-induced sleep loss in

drosophila. Curr. Biol. *20*, 1209–1215.

S12. Garbe, D., Bollinger, W., Vigderman, A., Masek, P., Gertowski, J., Sehgal, A., and Keene, A. (2015). Context-specific comparison of sleep acquisition systems in Drosophila. Biol Open *4*, 1558–68.

S13. Masek, P., Reynolds, L. a, Bollinger, W. L., Moody, C., Mehta, A., Murakami, K., Yoshizawa, M., Gibbs, A. G., and Keene, A. C. (2014). Altered regulation of sleep and feeding contribute to starvation resistance in Drosophila. J. Exp. Biol. Available at: http://www.ncbi.nlm.nih.gov/pubmed/24948636.

S14. Masek, P., and Scott, K. (2010). Limited taste discrimination in Drosophila. Proc. Natl. Acad. Sci. U. S. A. *107*, 14833–14838.

S15. Wong, R., Piper, M. D. W., Wertheim, B., and Partridge, L. (2009). Quantification of food intake in Drosophila. PLoS One *4*, e6063.

S16. Ja, W. W., Carvalho, G. B., Mak, E. M., de la Rosa, N. N., Fang, A. Y., Liong, J. C., Brummel, T., and Benzer, S. (2007). Prandiology of Drosophila and the CAFE assay. Proc. Natl. Acad. Sci. U. S. A. *104*, 8253–8256.

S17. Sassu, E. D., McDermott, J. E., Keys, B. J., Esmaeili, M., Keene, A. C., Birnbaum, M. J., and DiAngelo, J. R. (2012). Mio/dChREBP coordinately increases fat mass by regulating lipid synthesis and feeding behavior in Drosophila. Biochem. Biophys. Res. Commun. *426*, 43–48.

S18. Gingras, R. M., Warren, M. E., Nagengast, A. A., and Diangelo, J. R. (2014). The control of lipid metabolism by mRNA splicing in Drosophila. Biochem. Biophys. Res. Commun. *443*, 672–676.

S19. Wagh, D. A., Rasse, T. M., Asan, E., Hofbauer, A., Schwenkert, I., Dürrbeck, H., Buchner, S., Dabauvalle, M. C., Schmidt, M., Qin, G., et al. (2006). Bruchpilot, a protein with homology to ELKS/CAST, is required for structural integrity and function of synaptic active zones in Drosophila. Neuron *49*, 833–844.