Supplementary Information

Materials and Methods

Experimental Animals. *Aedes aegypti* mosquitoes were nurtured in an incubator at 27 ℃ and 80% humidity. Larvae were reared in water supplemented with complete larval diet (a mixture of rat chow, yeast and lactalbumin, 1:1:1 ratio), while adults were supplied with unlimited access to water and 10% (wt/vol) sucrose solution. Four-day-old female mosquitoes were blood fed on White Leghorn chickens. The treatment of vertebrate animals was approved by the University of California Riverside Institutional Animal Care and Use Committee.

RNA Extraction and qRT-PCR. The adult female mosquitoes were dissected at different time points. Total RNA was isolated from mosquito tissues using TRIzol (Invitrogen) method. cDNA was produced using the SuperScript IV First-Strand Synthesis System (Invitrogen). The SYBR Green Supermix (Bio-Rad) was used for qRT-PCR. The relative expression was calculated as $2^{-\Delta\Delta Ct}$ and normalized to the housekeeping gene *RPS7*. Each sample was measured in triplicate. All of these were performed according to the manufacturers' instructions.

In Vivo Hormonal Treatment. For JH, a 0.3-μL aliquot of 1 μg/mL JH III (Sigma) or acetone (solvent) alone was applied topically to the abdomens of newly emerged (6 h PE) female mosquitoes. For 20E hormone treatment, a 0.3-μL aliquot of 500 ng/mL 20E (Sigma) or ethanol (solvent) alone was injected into female mosquitoes at 72 h PE. Samples were collected and dissected 24 h post-treatments, then subjected to qPCR analyses.

dsRNA-Mediated RNAi. dsRNA was synthesized using the MEGAscript RNAi Kit (Ambion). The *luciferase* gene was used to generate control dsRNA (*dsLuc*). A 0.3-μL aliquot of 4 μg/μL dsRNA was injected into the thorax of cold-anesthetized female mosquitoes at 6 h

PE using Picospritzer II (General Valve).

ChIP Analysis. A Magna ChIP™ G Tissue Kit (Millipore) was used to obtain genomic interactions of transcription factors with chromosomal DNA. Briefly, tissues were dissected and sheared in Tissue Stabilizing Solution with Protease Inhibitor, fixed using 1% formaldehyde, treated with Tissue Lysis Buffer/ Protease Inhibitor and sonicated on wet ice. The targeted chromatin was pulled down by G bead-antibody of interest. The protein-DNA crosslinks were reverse transcribed, and the DNA was purified for downstream applications (qPCR). IgG of the same species as the antibody of interest was used as a negative control. RNAi-mediated disruption of the protein of interest was used as a control. The fold enrichment represents three biological replicates with 30 females in each.

Luciferase Reporter Assay. The promoter regions were PCR amplified and subcloned into reporter vector pGL3-Basic (Promega). The putative binding sites were mutated by incorporating restriction sites and In-Fusion HD Cloning (TaKaRa) in reporter plasmids. The coding sequence of interest was inserted into the expression plasmid pAc5.1 with Myc or FLAG tag. 200 ng of desired reporter plasmid and 50 ng of the control Renilla luciferase reporter vector, with or without 200 ng of expression plasmid, were co-transfected into *Drosophila* S2 cells using FuGENE HD Transfection Reagent (Promega). JH-III (final concentration 20 μM) was added at 42 h post-transfection. Luciferase determinations were performed at 48 h post-transfection using the Dual Luciferase Assay kit (Promega) according to the manufacturer's instructions.

Measurements of ILP Levels Using ELISA. For circulating ILP levels, hemolymph was collected on ice from each female mosquito using Picospritzer II (General Valve). 20 μL of pooled hemolymph in phosphate buffered saline (PBS) was used for ELISA (five females per sample). For total ILP levels, tissues were homogenized in PBS containing 1% Triton X-100 (200 μL, three females per sample), lysed for 0.5 h with rotary shaking, then centrifuged and the supernatant used for ELISA. Immunoplates were coated overnight (4 ℃) with anti-FLAG antibody (Sigma-Aldrich) diluted in 0.2M sodium carbonate/bicarbonate buffer (pH 9.4) to 5

μg/mL. The plates were washed twice with PBS containing 0.2% Tween 20 (PBS-T) and blocked with 2% bovine serum albumin (BSA) in PBS for 3 h at room temperature. After a triple wash with PBS-T, a sample (5 μL of hemolymph or 50 μL of supernatant) or equal volume of FLAG(GS)HA peptide standard (LifeTein LLC) was added to each well. Anti-HA-Peroxidase 3F10 antibody (Sigma-Aldrich), diluted to 15 ng/mL in PBS with 1% Triton X-100, was added to each well (50 μL/well). Plates were sealed and incubated in a humidity chamber overnight at 4 ℃. The wells were washed six times with PBS-T. 1-Step Ultra TMB ELISA Substrate (Thermo Fisher Scientific) was added to each well and incubated for 20 minutes at room temperature. The reactions were stopped by adding 2 M sulfuric acid. Absorbance was measured at 450 nm using a plate reader (Wallac). To convert molar concentrations, we used the molecular weights of 10.12 (ILP1-HA/FLAG), 9.55 (ILP2-HA/FLAG), 8.26 (ILP3-HA/FLAG), 8.17 (ILP4-HA/FLAG), 13.16 (ILP5-HA/FLAG), 17.92 (ILP6-HA/FLAG), 10.23 (ILP7-HA/FLAG) and 7.82 (ILP8-HA/FLAG) kilodaltons (kDa) for each mature protein. Three biological replicates were determined for each group.

Immunofluorescence. Tissues were fixed in 4% (vol/vol) paraformaldehyde, rinsed with PBS-T [0.3% (vol/vol) Triton X-100 in PBS], kept in 3% (wt/vol) BSA-PBS-T for 1 h, incubated overnight with primary antibody mouse anti-HA (1:1,000; Abcam), incubated 2 h with Alex Fluor Plus 488-conjugated secondary antibody (goat anti-mouse; Invitrogen), stained by ProLong Diamond Antifade Mountain with DAPI (Invitrogen), then imaged under a confocal laser scanning microscope (Leica SP5). The mean fluorescence intensity was analyzed using *ImageJ* software (https://imagej.nih.gov/ij/docs/index.html).

Statistical Analysis. All values are presented as mean ± SEM. Mean values were compared using the Student's t-test at the following significance levels: *P < 0.05, **P < 0.01, and ***P < 0.001. Statistical analyses were performed using GraphPad Prism 6.

Supplementary Information

Figure legends

Fig. S1. Temporal distribution of *ILP* transcription in female *Ae. aegypti* mosquitoes. Relative expression profiles of *ilps* 1, 3, 4, 7 and 8 in heads, *ilp2* in ovaries, *ilp5* in carcasses and *ilp6* in fat bodies at 12 and 48 h PE, and at 12, 24 and 48 h PBM. Data represent three biological replicates, with 30 mosquitoes in head replication and 10 mosquitoes in other tissue replication and are shown as mean ± SEM. *P < 0.05, **P < 0.01, and ***P < 0.001.

Fig. S2. The absence of the binding of Met and Kr-h1 to the promoters of several metabolic enzyme genes. ChIP analysis of 3-kb regulatory regions showing no enrichment from anti-Met and anti-Kr-h1 antibodies. The X-axis numbers (base pair, bp) indicate the 3-kb of 5' upstream regulatory regions with ten 300-bp units for the assay. Data represent three biological replicates with 30 individuals in each and are shown as mean ± SEM.

Fig. S3. There is no binding of EcR to the promoters of several metabolic enzyme genes. ChIP analysis of 3-kb regulatory regions showing the absence of enrichment from anti-EcR antibodies. X-axis: base pair (bp). Data represent three biological replicates with 30 individuals in each and are shown as mean ± SEM.

Fig. S4. The absence of the binding of E74A and E74B with the promoters of several metabolic enzyme genes. ChIP analysis of potential binding motifs showing no enrichment from anti-E74A and anti-E74B antibodies. X-axis: base pair (bp). Data represent three biological replicates with 30 individuals in each and are shown as mean ± SEM.

Fig. S5. Comparative analysis of *ILP* transcript levels in JH- and 20E-treated female mosquitoes. (*A*) Topical application of JH elevated transcript levels of *ILPs 2*, *6* and *7*, while repressing those of other *ILPs*. (*B*) The effects of 20E injection on *ILP* transcript levels. *ILPs*

4 and *5* increased, *ILP2* decreased, and others showed no change. Data represent three biological replicates with 30 individuals in each and are shown as mean ± SEM. *P < 0.05, **P < 0.01, and ***P < 0.001.

Fig. S6. Insulin pathway factors are not dependent on the JH and 20E pathways. (*A*) The efficiency of RNAi for *Met, Kr-h1, EcR* and *E74*. (*B*) Analysis of *InR, IRS, PI3K, PTEN, Akt, PDK* and *FoxO* transcripts in *dsMet, dsKr-h1, dsEcR* and *dsE74* RNAi mosquitoes at 72 h PE and 24 h PBM. Data represent three biological replicates with 30 individuals in each and are shown as mean ± SEM. *P < 0.05, **P < 0.01.

Fig. S7. Graphical representation of the *ilp* genomic loci and their motif binding sites. (*A*) The chromosomal location of genomic loci of the *ilp* genes from VectorBase. (*B*) The motif binding sites of Met (green), Kr-h1 (blue) and E74 (red) within the 3-kb 5' upstream regulatory region of each *ilp* gene.

Fig. S8. ChIP-qPCR assay for Met and *ilp* genes. Met genomic binding was found in *ilp6* promoter, but absence in the promoters of *ilp8-ilp1-ilp3* operon, *ilp2, ilp4, ilp5* and *ilp7*. X-axis: base pair (bp). The relative fold enrichment of repeats was obtained from specific antibodies (anti-Met). RNAi silencing of Met diminished the binding enrichment. Data represent three biological replicates with 30 individuals in each and are shown as mean ± SEM. ***P < 0.001.

Fig. S9. Kr-h1 binding of the *ilp* genes. ChIP-qPCR shows the relative fold enrichment of repeats obtained from specific anti-Kr-h1 antibodies. *Kr-h1* RNAi silencing diminished the binding enrichment. X-axis: base pair (bp). Data represent three biological replicates with 30 individuals in each and are shown as mean ± SEM. *P < 0.05, **P < 0.01, ***P < 0.001.

Fig. S10. Kr-h1, E74A and E74B and other *ilp* genes in cell Luciferase reporter assay. (*A*) Luciferase reporter assay after co-transfection of the expression vector *pAc-Kr-h1-Myc* and reporter constructs points out the activation role of Kr-h1 in the transcription of *ilp7*, while the

repression role in the transcription of *ilps 8-1-3*. (*B*) Luciferase reporter assay after co-transfection of expression vectors *pAc-E74A-Myc* and reporter constructs indicates that E74A has no change on *ilp6* promoter. Luciferase reporter assay after co-transfection of expression vectors *pAc-E74B-Myc* and reporter constructs indicates E74B has no change on *ilp5* promoter. Treatments with the empty expression vector and no input DNA and motif mutation served as controls. Data represent six replicates and are shown as mean ± SEM. **P < 0.01, ***P < 0.001.

Fig. S11. The absence of EcR binding to the promoters of *ilp* genes. ChIP analysis of 3-kb regulatory regions (without potential EcR-binding motif) shows no enrichment from anti-EcR antibodies. X-axis: base pair (bp). Data represent three biological replicates with 30 individuals in each and are shown as mean ± SEM.

Fig. S12. ChIP-qPCR analysis of E74A interaction with promoters of the *ilp* genes. (*A*) The relative fold enrichment of repeats on *ilp4* and *ilp5* was obtained with specific antibodies (anti-E74A). RNAi silencing of *E74A* diminished the binding enrichment. (*B*) ChIP analysis of the potential binding motifs shows no enrichment from anti-E74A on *ilp8-ilp1-ilp3*, *ilp2*, *ilp6* and *ilp7*. X-axis: base pair (bp). Data represent three biological replicates with 30 individuals in each and are shown as mean ± SEM. *P < 0.05, **P < 0.01.

Fig. S13. ChIP-qPCR analysis of E74B interaction with promoters of the *ilp* genes. (*A*) The relative fold enrichment of repeats on *ilp2* and *ilp6* was obtained with specific antibodies (anti-E74B). RNAi silencing of *E74B* diminished the binding enrichment. (*B*) ChIP analysis of the potential binding motif shows no enrichment from anti-E74B on *ilp8-ilp1-ilp3*, *ilp4*, *ilp5* and *ilp7*. X-axis: base pair (bp). Data represent three biological replicates with 30 individuals in each and are shown as mean ± SEM. *P < 0.05, **P < 0.01.

Fig. S14. The insertion events verified by PCR of genomic DNA and DNA sequencing. (*A*) The primers F1 and R1 were used for HA tag with R1 harboring the HA sequence. The primers F2 and R2 were used for FLAG tag, with F2 harboring the FLAG sequence. The

ilp-HF females were positive in both PCR products. Wild-type (WT) females were used as controls. M, DNA marker; numbers 1~8, analysis of *ilps 1~8* in their related mosquito lines and WT control. (*B*) Sanger sequencing of the PCR products for *ilp-HA/FLAG* (*ilp-HF*). Green, HA-tag DNA sequence; Red, FLAG-tag DNA sequence; Black, genomic DNA sequence in *ilp* gene.

Fig. S15. The rates of *ilp* gene tagging. The injected egg numbers (injected-G0/G1), survival-to-adult rates (G0/G1-adults), survived female rates (G0/G1-female), and insertion rates in females (G0/G1-F-HA/HF). Data represent mean value in groups.

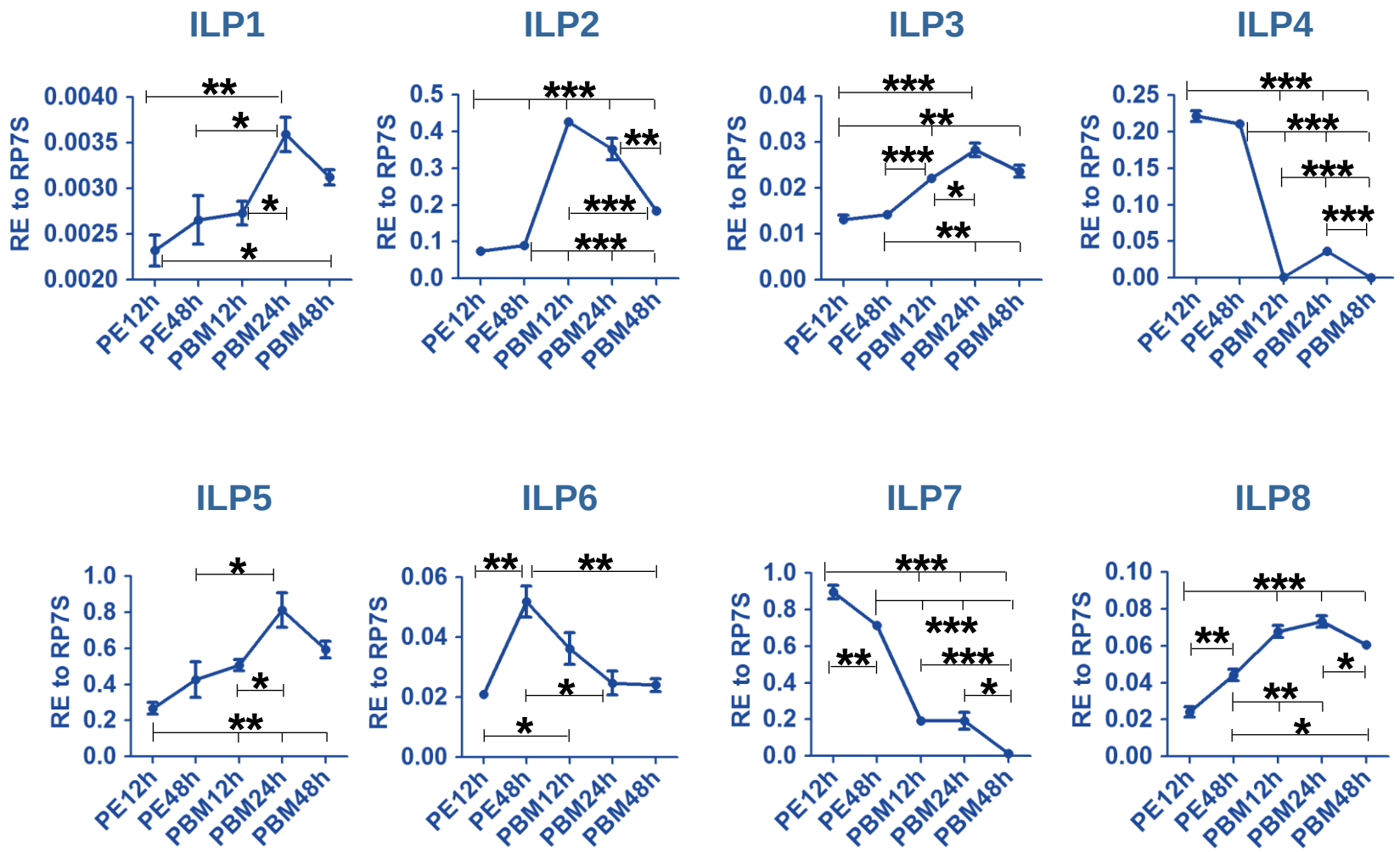
Fig. S16. The response of tissue ILP proteins on JH and 20E signaling. (*A*) The protein level of each ILP-HA/FLAG (pg/mg) was determined using ELISA in tagged females with RNAi *Met* or RNAi *Kr-h1* treatments and their respective controls. (*B*) The protein level of each ILP-HA/FLAG (pg/mg) was determined using ELISA in tagged females with RNAi *EcR* or RNAi *E74* treatments and their respective controls. Data represent three biological replicates with 3 individuals in each and are shown as mean ± SEM. *P < 0.05, **P < 0.01, ***P < 0.001.

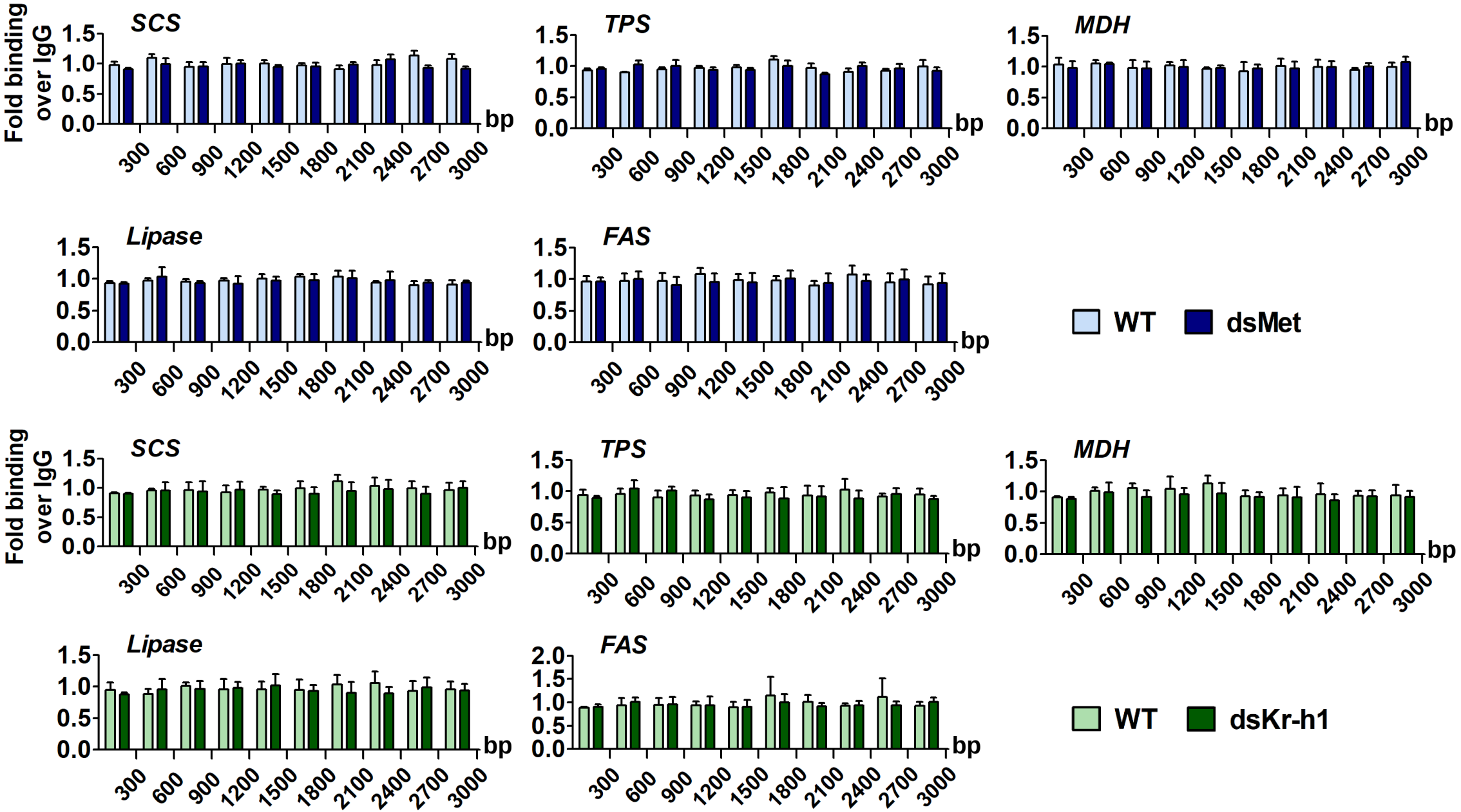
Fig. S17. Hemolymph levels of other ILPs in response to JH and 20E signaling. (*A*) Hemolymph ILP-HA/FLAG content (pg/μL) (ILPs 1, 3, 7, 8) was determined using ELISA in tagged females after RNAi *Met* or RNAi *Kr-h1* treatments and respective controls. (*B*) Hemolymph ILP-HA/FLAG content (pg/μL) (ILPs 1, 3, 7, 8) was determined using ELISA in tagged females after RNAi *EcR* or RNAi *E74* treatments and respective controls. Data represent three biological replicates with 5 individuals in each and are shown as mean ± SEM. *P < 0.05, **P < 0.01.

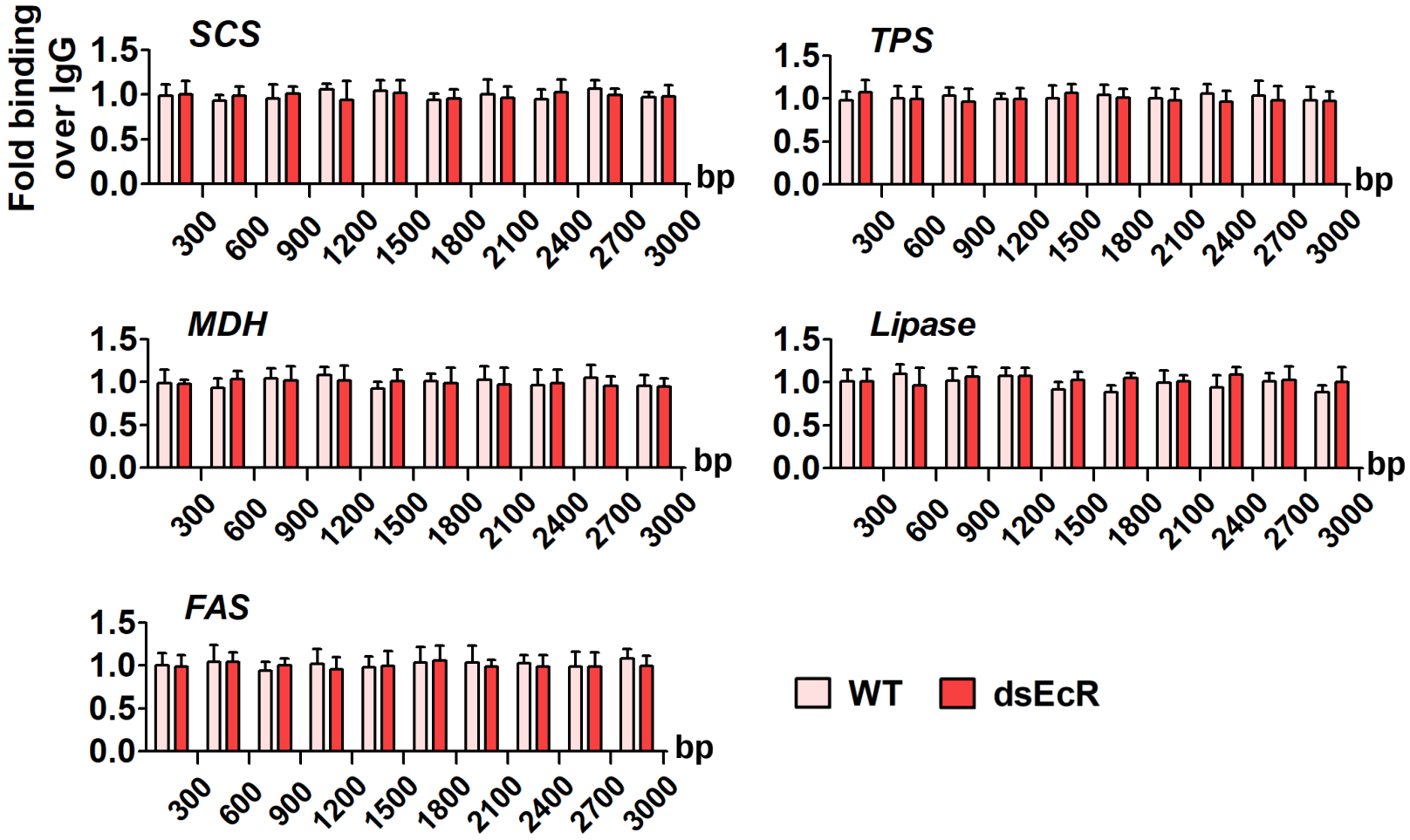
Fig. S18. RNAi efficiency of *dsMet*, *dsKr-h1*, *dsEcR*, *dsE74* and *dsFoxO* in *FoxO-HA*-tagged mosquitoes. (*A*) The efficiency of RNAi- *Met*, *Kr-h1*, *EcR* and *E74* at 72 h PE and 24 h PBM for FoxO localization. (*B*) The efficiency of RNAi-*FoxO* at 72 h PE for the ChIP-binding analysis of FoxO and subsequent experiment. Data represent three biological replicates and

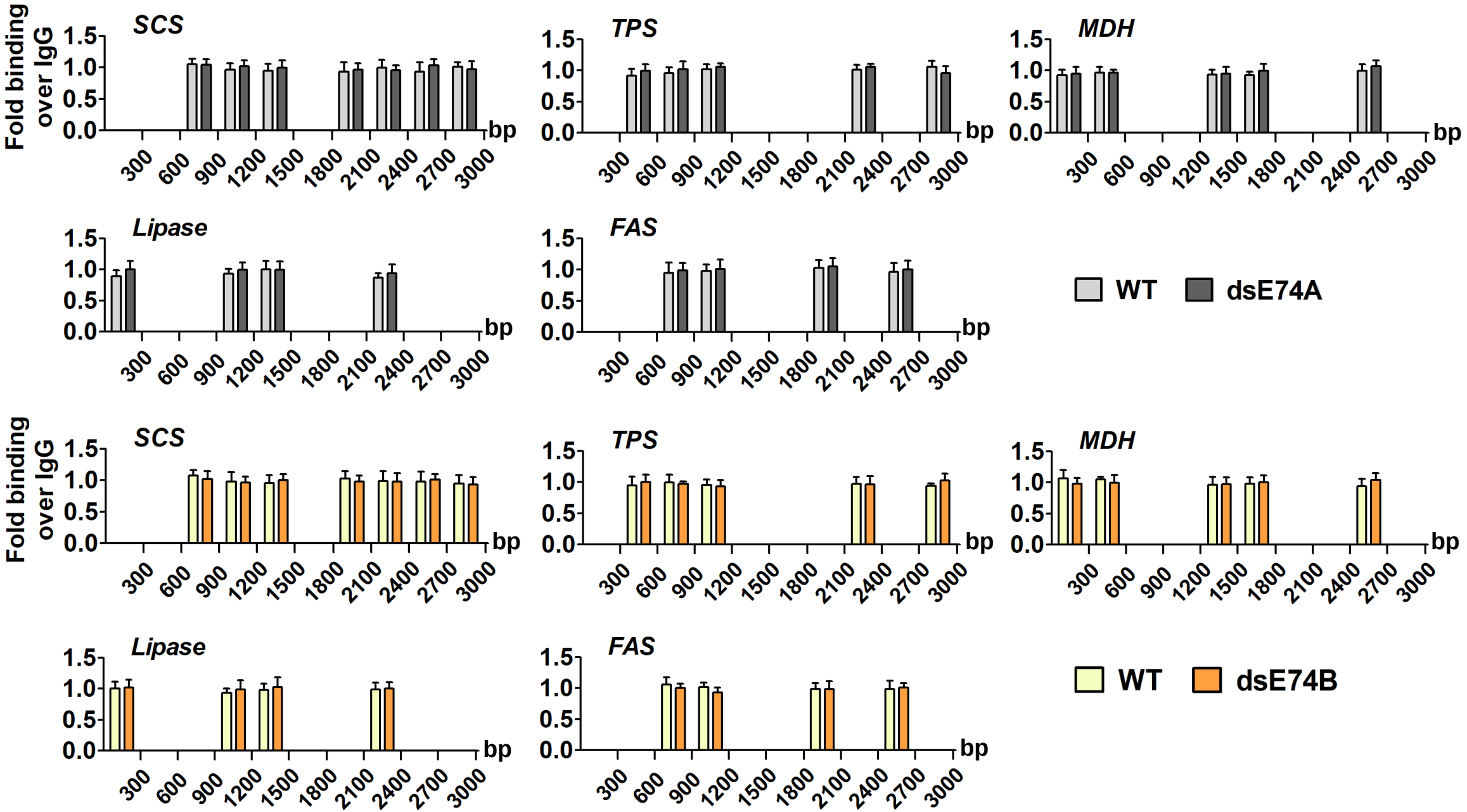
are shown as mean ± SEM. **P < 0.01.

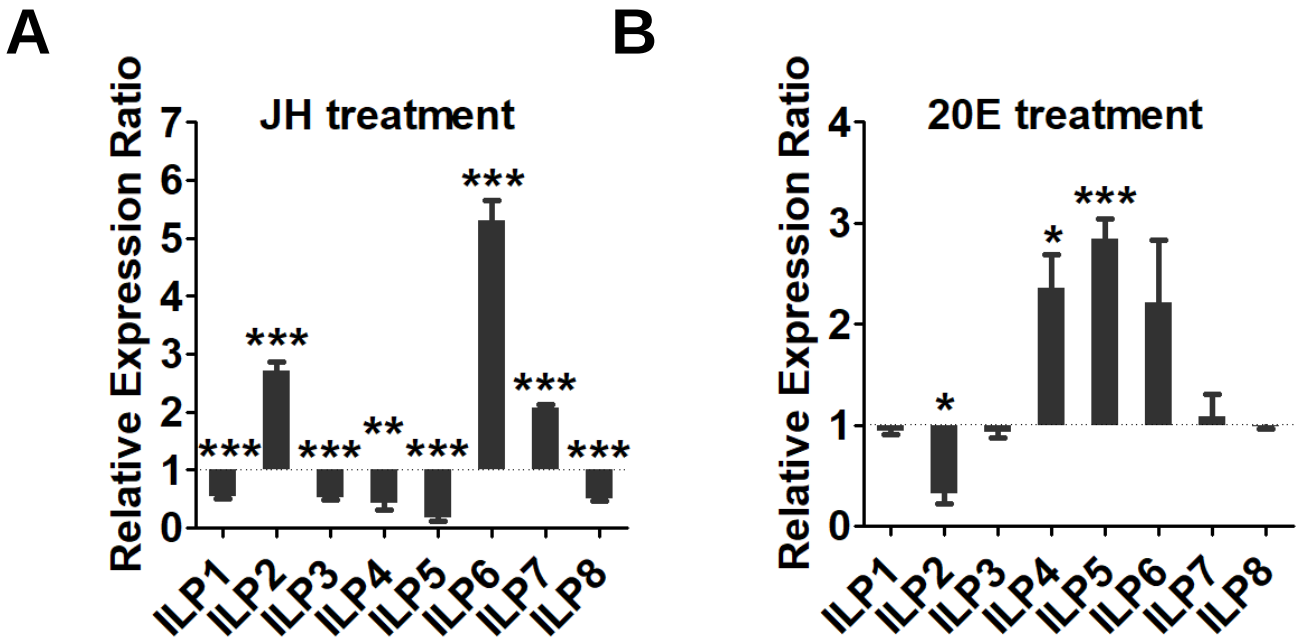
Fig. S19. ChIP analysis demonstrating FoxO direct binding to the promoters of metabolic enzyme genes: *succinyl-coA synthetase* (*SCS*), *trehalose-6-phosphate synthase* (*TPS*), *malate dehydrogenase* (*MDH*), *lipase* and *fatty acid synthases* (*FAS*). Their FoxO motif binding sites (blue string) were marked within the 3-kb 5' upstream regulatory region. ChIP-qPCR analysis in *FoxO-HA* tagged mosquito females, showing the relative fold enrichment of repeats obtained from anti-HA antibody, indicates the interactions of the transcription factor FoxO with the chromosomal DNA of metabolic genes. Data represent three biological replicates with 30 individuals in each and are shown as mean ± SEM. *P < 0.05, **P < 0.01, ***P < 0.001.

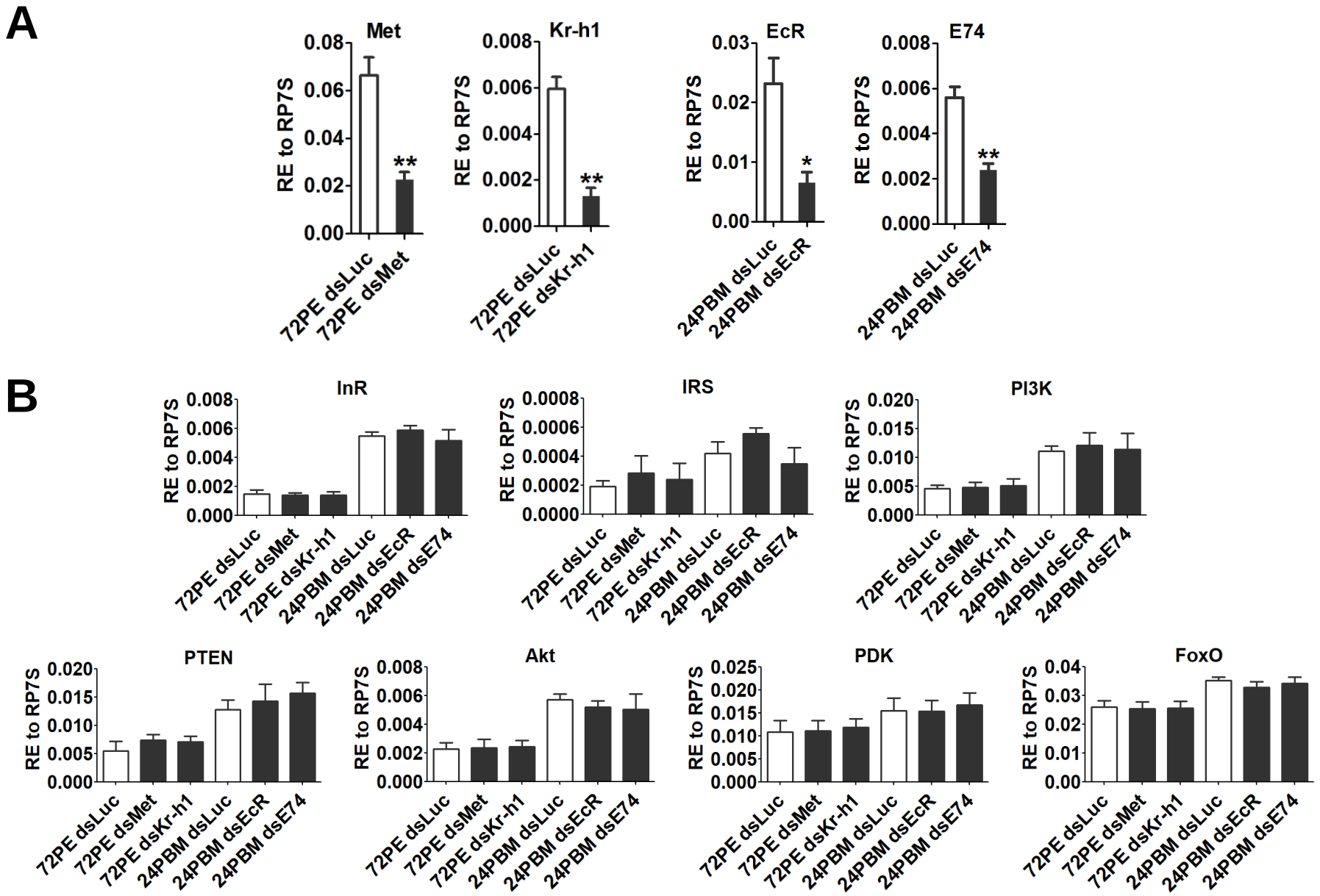

ILP1
ILP2
ILP3
ILP4
ILP5
ILP6
ILP7
ILP8
RE to RP7S
PE12h
PE48h
PBM12h
PBM24h
PBM48h

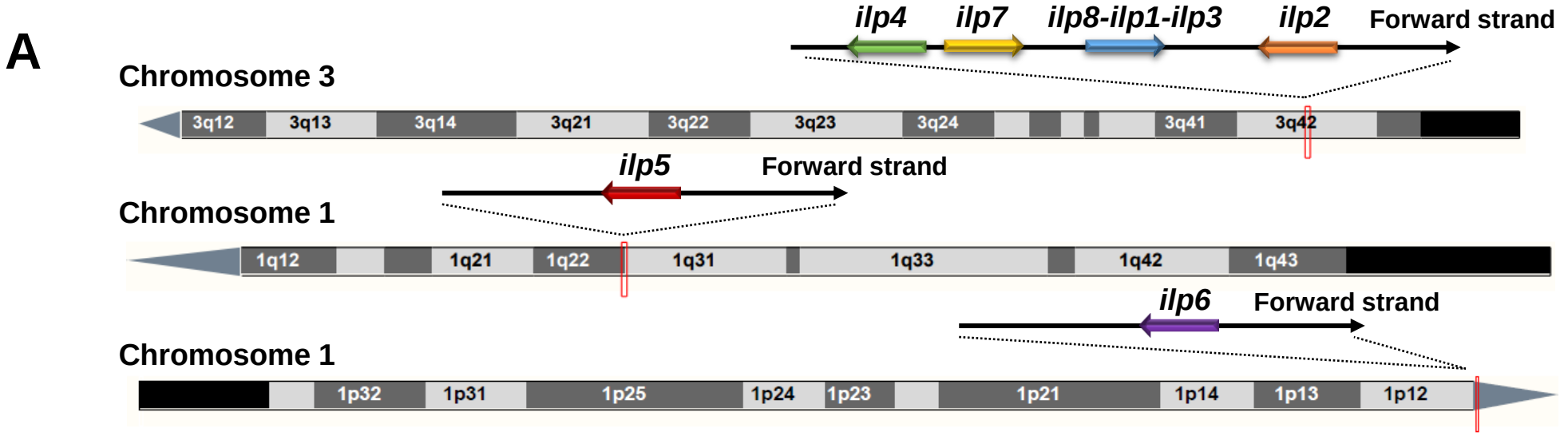

Fold binding over IgG
SCS
TPS
MDH
Lipase
FAS
bp
300
600
900
1200
1500
1800
2100
2400
2700
3000
WT
dsMet
WT
dsKr-h1

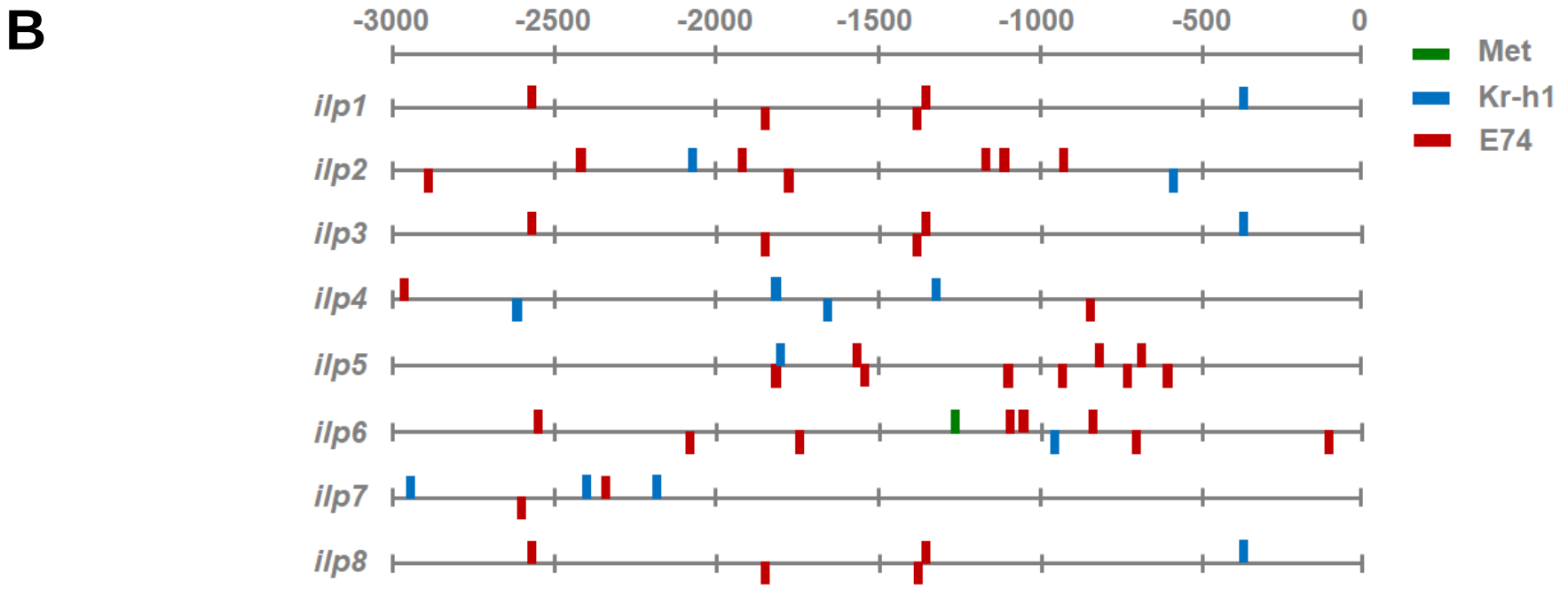

Fold binding over IgG
SCS
TPS
MDH
Lipase
FAS
1.5
1.0
0.5
0.0
bp
300
600
900
1200
1500
1800
2100
2400
2700
3000
WT
dsEcR

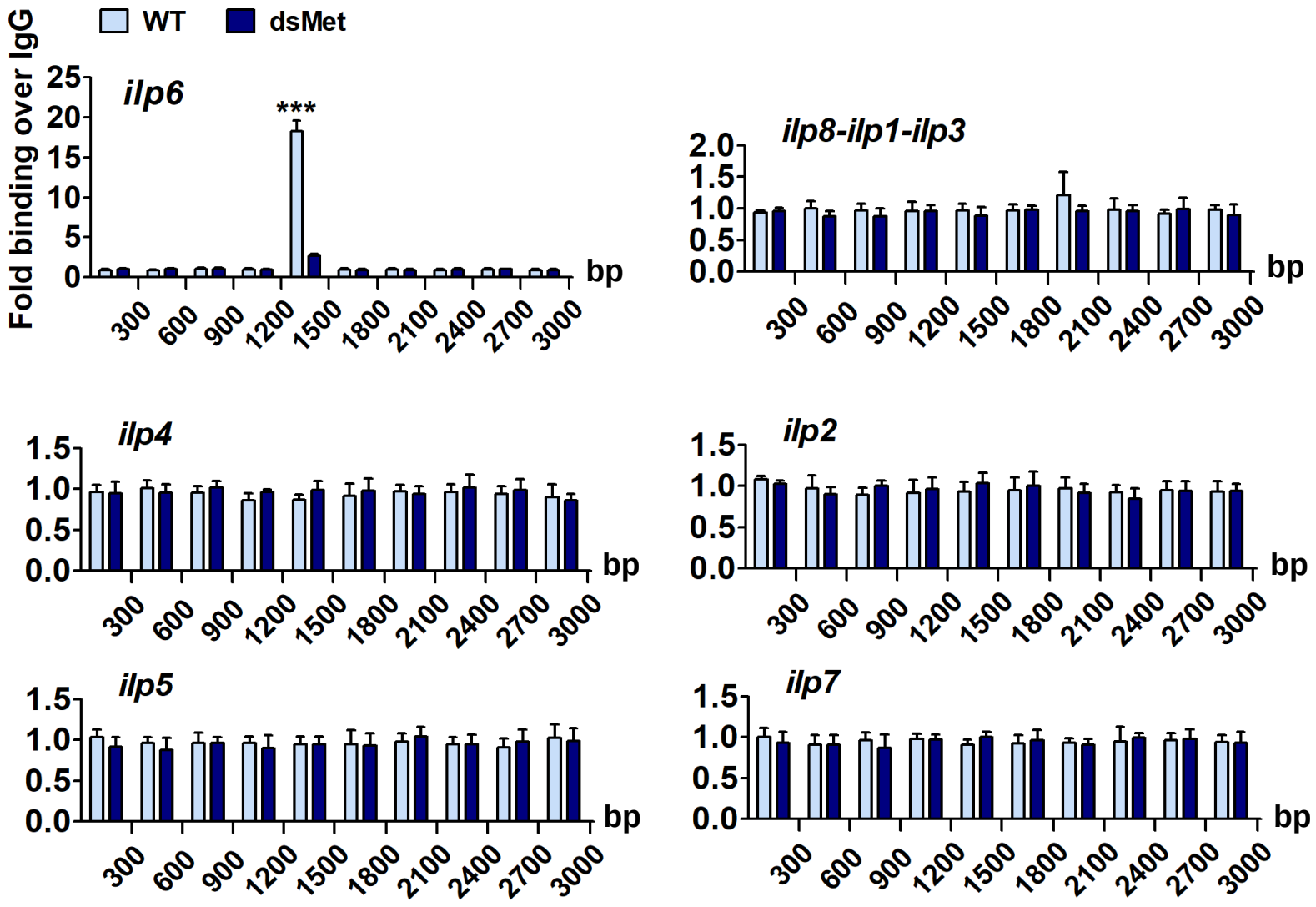

Fold binding over IgG
SCS
TPS
MDH
Lipase
FAS
1.5
1.0
0.5
0.0
300
600
900
1200
1500
1800
2100
2400
2700
3000
bp
WT
dsE74A
dsE74B

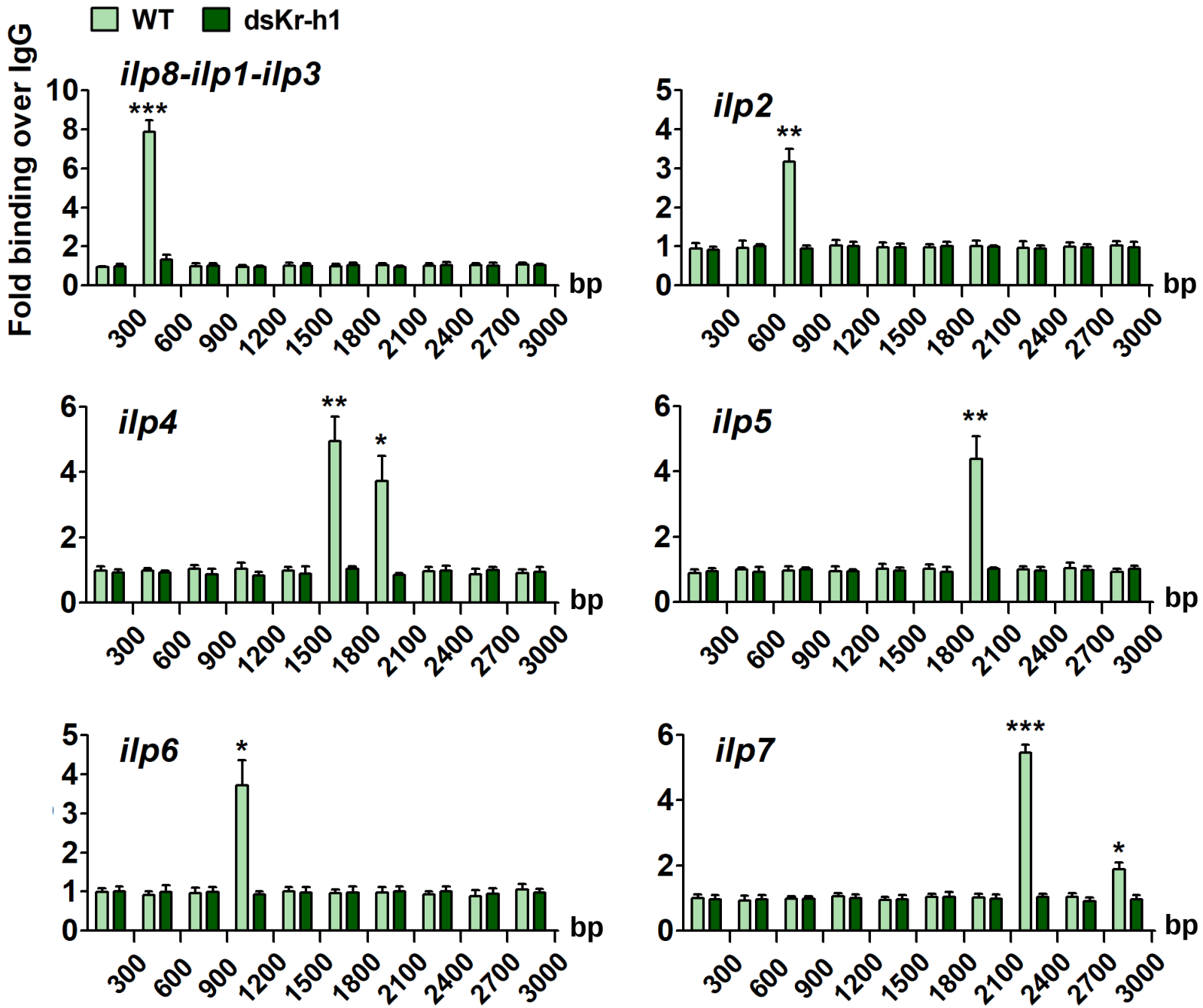

A
JH treatment
Relative Expression Ratio
0
1
2
3
4
5
6
7
***
***
***
**
***
***
***
ILP1
ILP2
ILP3
ILP4
ILP5
ILP6
ILP7
ILP8
B
20E treatment
Relative Expression Ratio
0
1
2
3
4
*
*
***
ILP1
ILP2
ILP3
ILP4
ILP5
ILP6
ILP7
ILP8

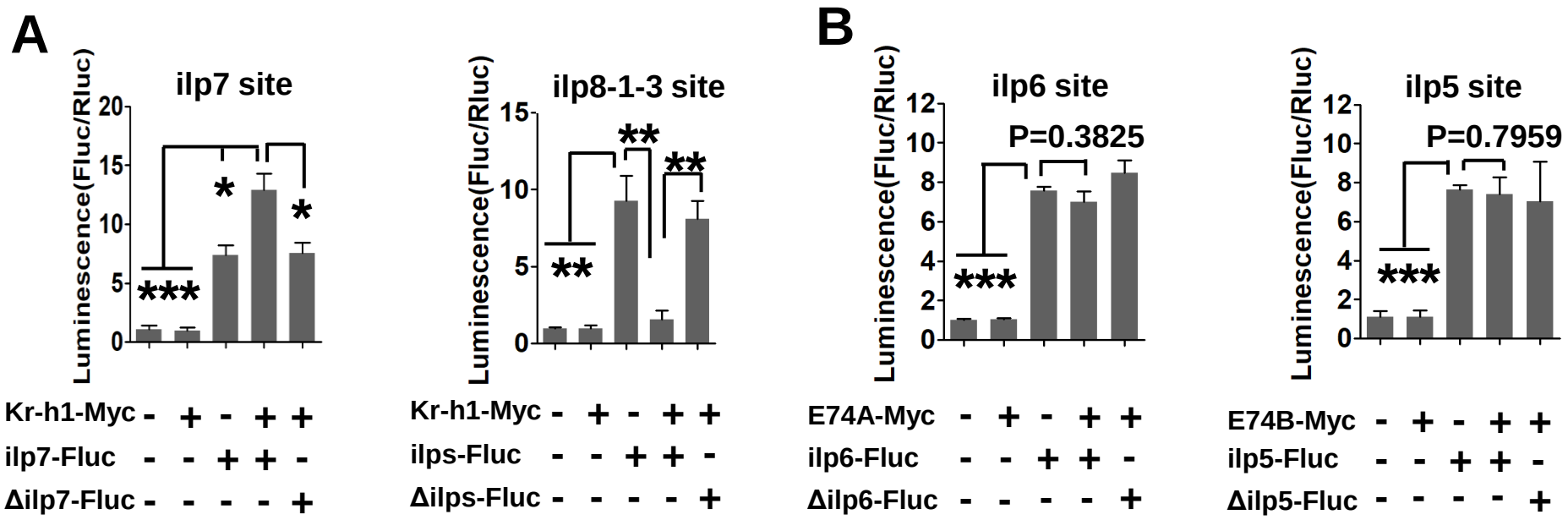

A
Met
Kr-h1
EcR
E74
RE to RP7S
72PE dsLuc
72PE dsMet
72PE dsKr-h1
24PBM dsLuc
24PBM dsEcR
24PBM dsE74
B
InR
IRS
PI3K
PTEN
Akt
PDK
FoxO

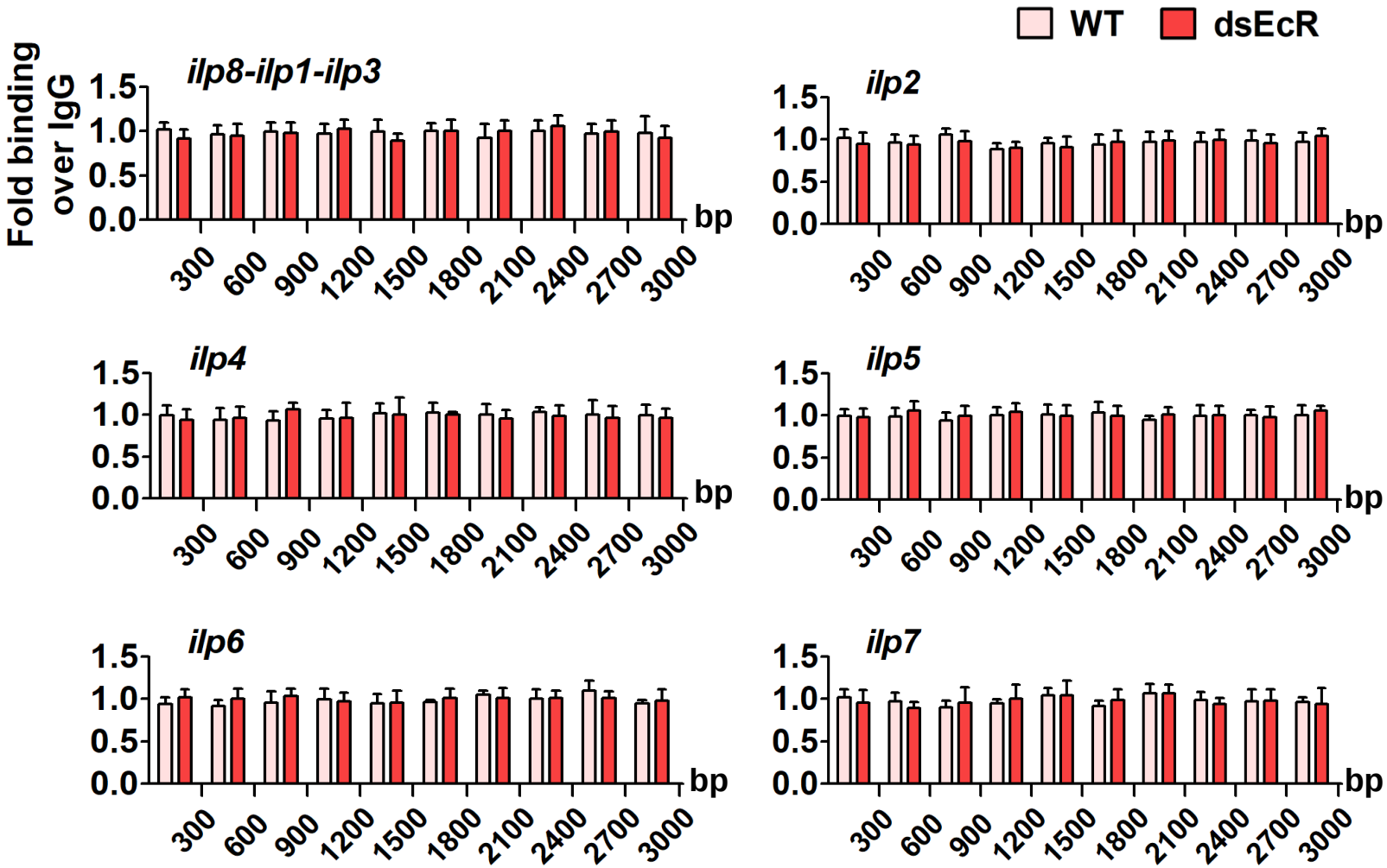

A
Chromosome 3
ilp4
ilp7
ilp8-ilp1-ilp3
ilp2
Forward strand
3q12
3q13
3q14
3q21
3q22
3q23
3q24
3q41
3q42
Chromosome 1
ilp5
Forward strand
1q12
1q21
1q22
1q31
1q33
1q42
1q43
Chromosome 1
ilp6
Forward strand
1p32
1p31
1p25
1p24
1p23
1p21
1p14
1p13
1p12

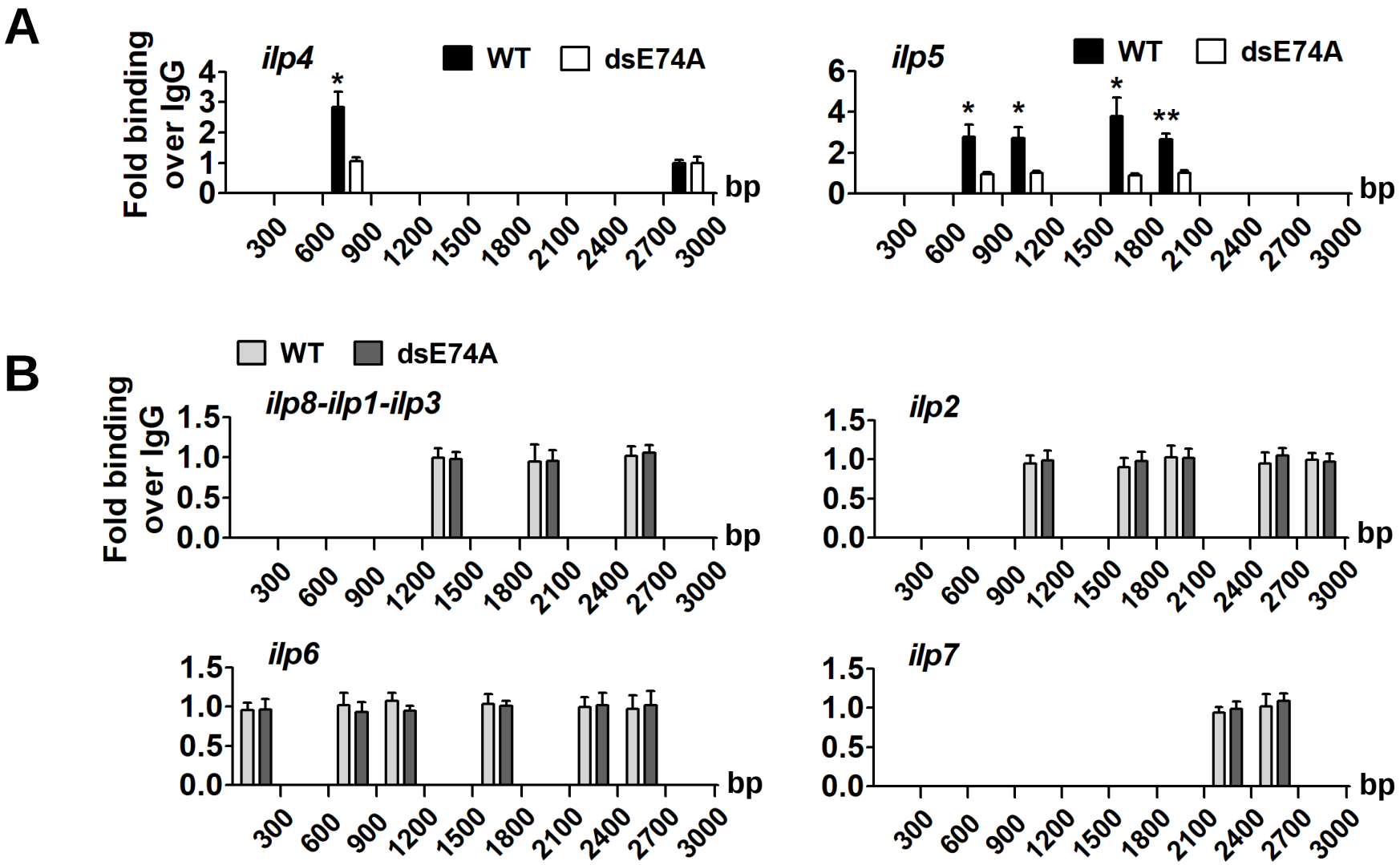

B
-3000
-2500
-2000
-1500
-1000
-500
0
ilp1
ilp2
ilp3
ilp4
ilp5
ilp6
ilp7
ilp8
Met
Kr-h1
E74

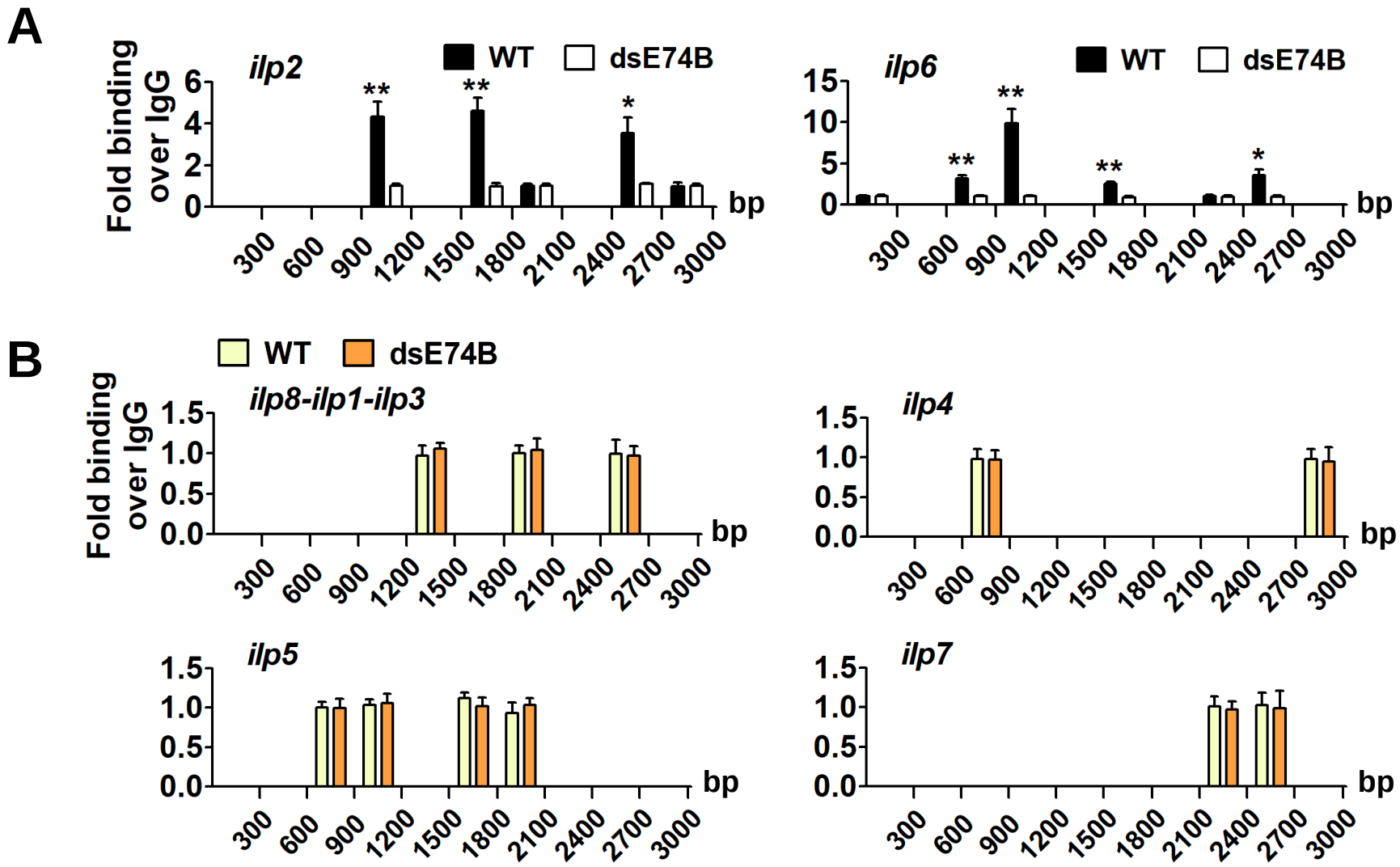

WT
dsMet
Fold binding over IgG
ilp6
***
ilp8-ilp1-ilp3
ilp4
ilp2
ilp5
ilp7
bp
300
600
900
1200
1500
1800
2100
2400
2700
3000


WT
dsKr-h1
Fold binding over IgG
ilp8-ilp1-ilp3
ilp2
ilp4
ilp5
ilp6
ilp7
bp
300
600
900
1200
1500
1800
2100
2400
2700
3000

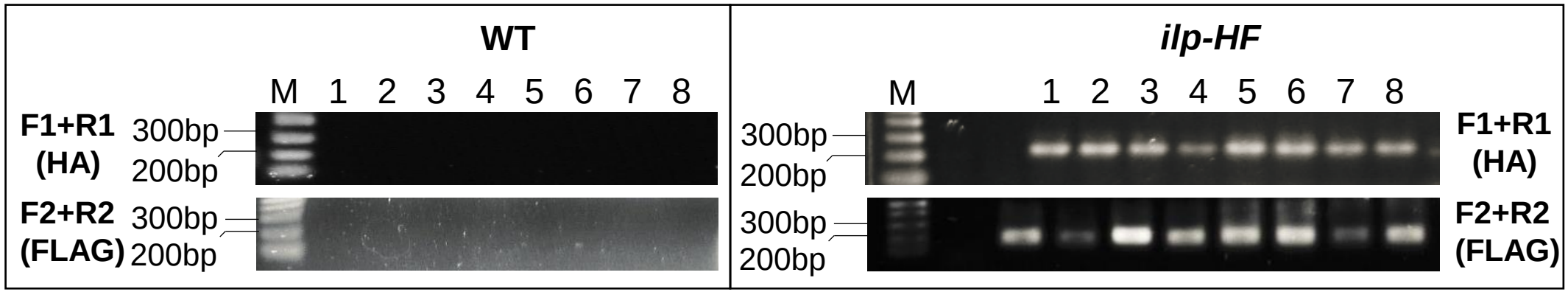

A
ilp7 site
Luminescence(Fluc/Rluc)
20
15
10
5
0
***
*
*
Kr-h1-Myc - + - + +
ilp7-Fluc - - + + -
Δilp7-Fluc - - - - +
ilp8-1-3 site
Luminescence(Fluc/Rluc)
15
10
5
0
**
**
**
Kr-h1-Myc - + - + +
ilps-Fluc - - + + -
Δilps-Fluc - - - - +
B
ilp6 site
P=0.3825
Luminescence(Fluc/Rluc)
12
10
8
6
4
2
0
***
E74A-Myc - + - + +
ilp6-Fluc - - + + -
Δilp6-Fluc - - - - +
ilp5 site
P=0.7959
Luminescence(Fluc/Rluc)
12
10
8
6
4
2
0
***
E74B-Myc - + - + +
ilp5-Fluc - - + + -
Δilp5-Fluc - - - - +


WT
dsEcR
Fold binding over IgG
ilp8-ilp1-ilp3
ilp2
ilp4
ilp5
ilp6
ilp7
1.5
1.0
0.5
0.0
bp
300
600
900
1200
1500
1800
2100
2400
2700
3000


A
ilp4
WT
dsE74A
Fold binding over IgG
bp
ilp5
B
ilp8-ilp1-ilp3
ilp2
ilp6
ilp7


A
ilp2
WT
dsE74B
Fold binding over IgG
bp
ilp6
B
ilp8-ilp1-ilp3
ilp4
ilp5
ilp7
300
600
900
1200
1500
1800
2100
2400
2700
3000

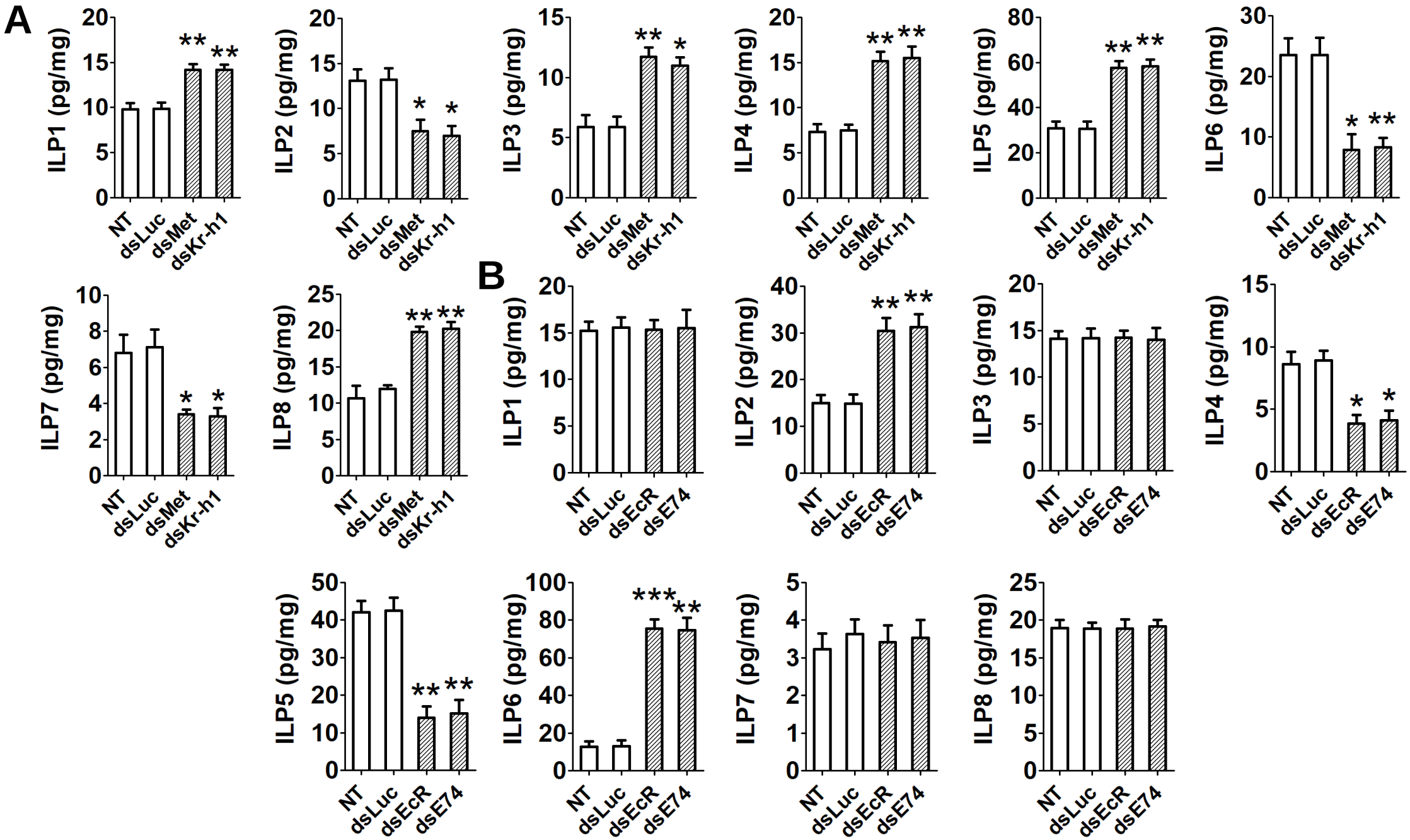
A

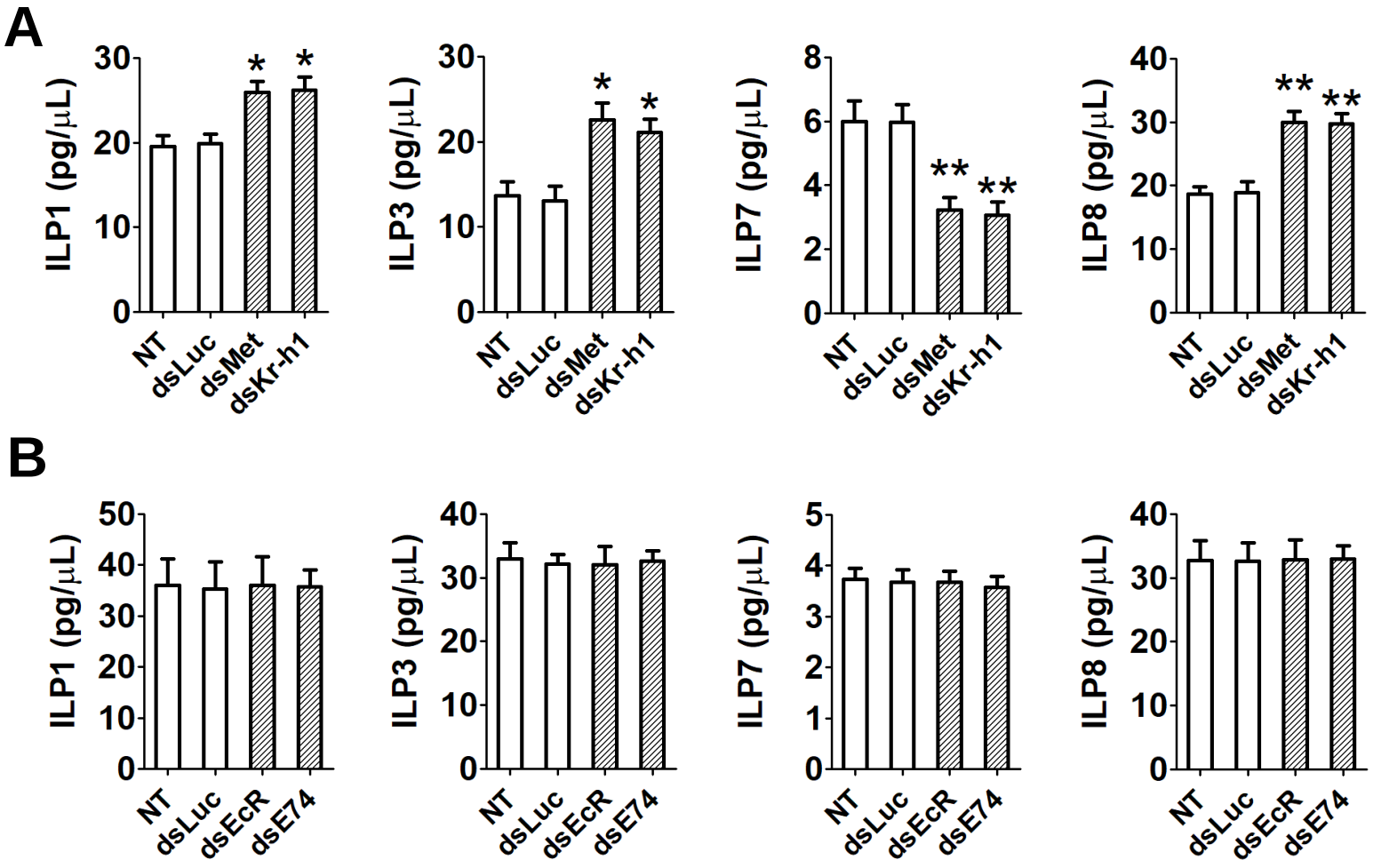


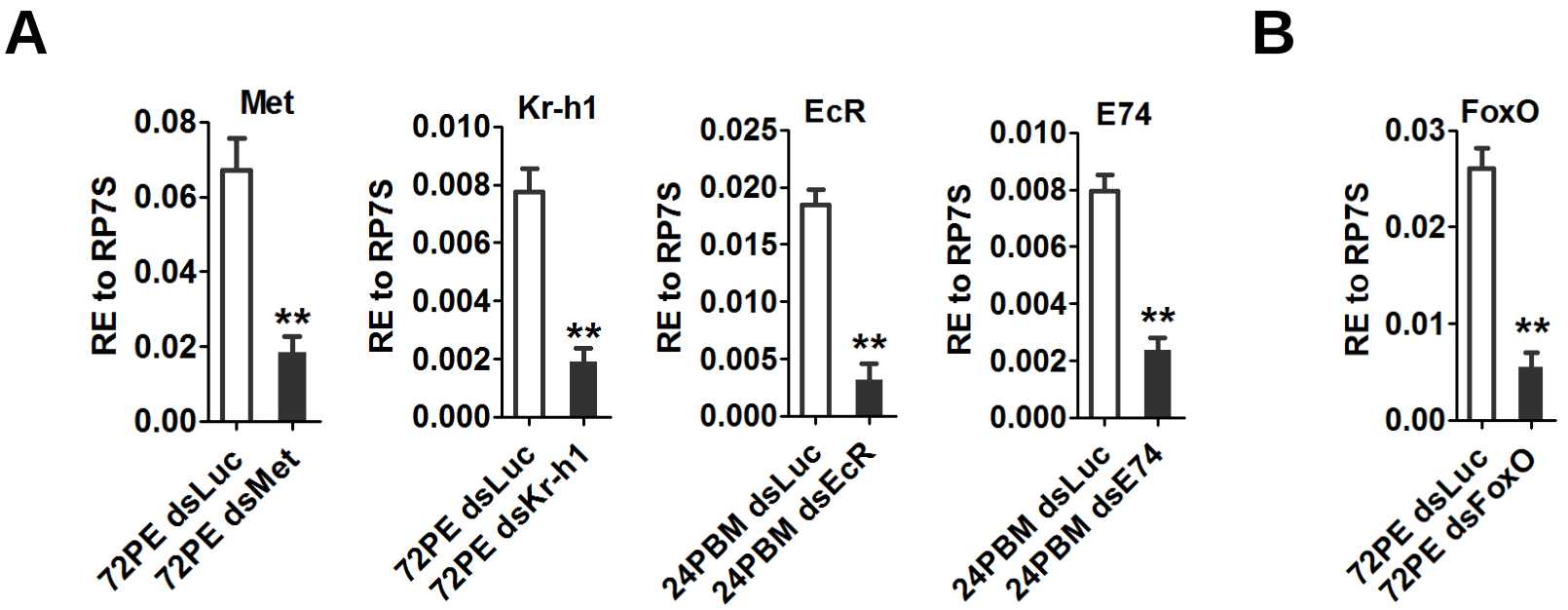
B

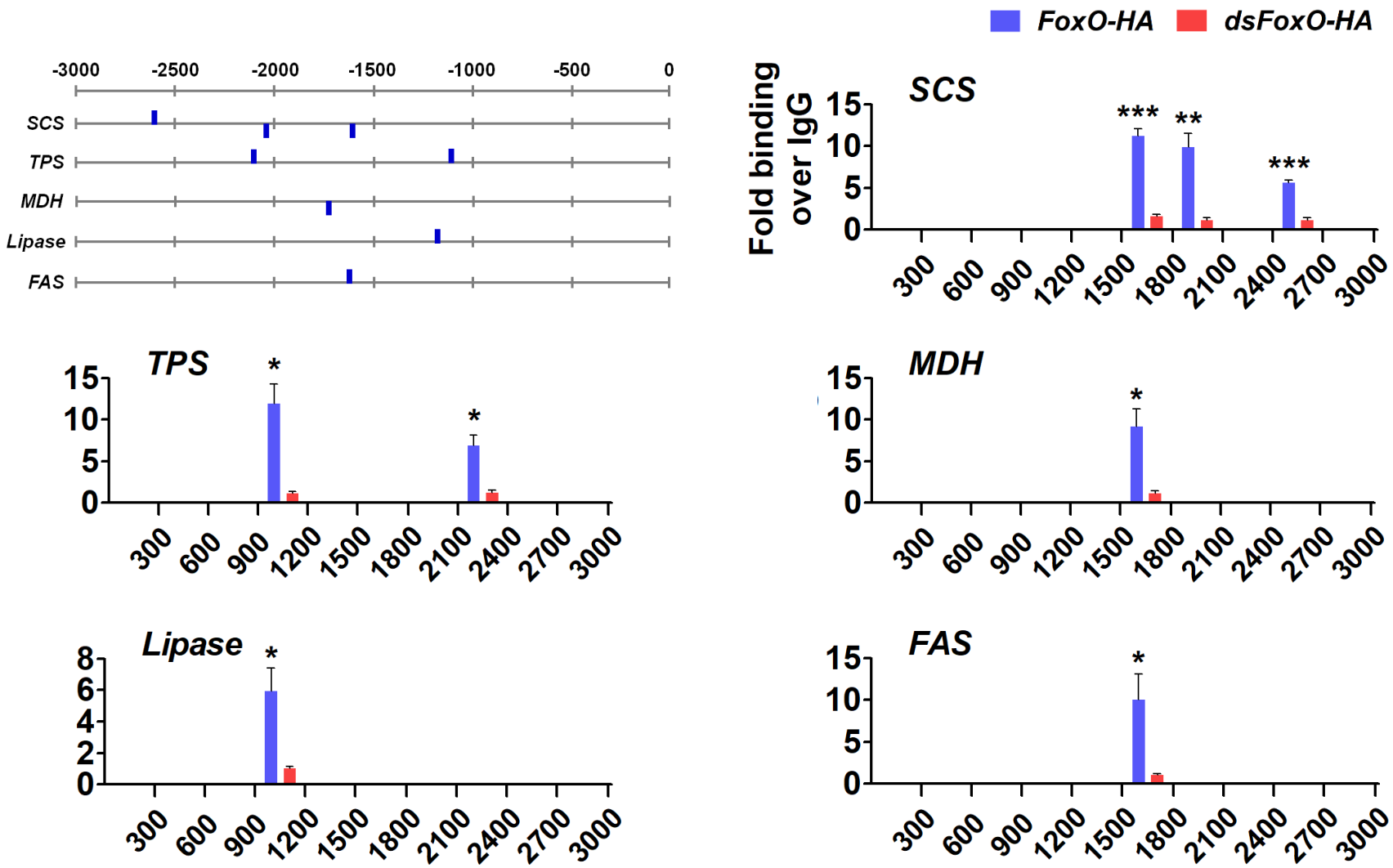
ilp1-HF …CCGTGGTACCCATACGATGTTCCAGATTACGCTAGAAAG……CGACGCGACTACAAGGACGACGATGACAAGCAGATC…
ilp2-HF …CATCACTACCCATACGATGTTCCAGATTACGCTCGCTCA……CGCCGAGACTACAAGGACGACGATGACAAGGGCGG…
ilp3-HF …GGTGCTTACCCATACGATGTTCCAGATTACGCTAAAAAA……CGCAAAGACTACAAGGACGACGATGACAAGAGCCCC…
ilp4-HF …CCAATGTACCCATACGATGTTCCAGATTACGCTAAACGA……CGTAGAGACTACAAGGACGACGATGACAAGCCAGGG…
ilp5-HF …ATTTCATACCCATACGATGTTCCAGATTACGCTAGAAGG……CGACGCGACTACAAGGACGACGATGACAAGACCGGG…
ilp6-HF …AGCGAATACCCATACGATGTTCCAGATTACGCTCGACGA……AAACGAGACTACAAGGACGACGATGACAAGGGGATA…
ilp7-HF …CCCAGCTACCCATACGATGTTCCAGATTACGCTAAGCGT……AAACGAGACTACAAGGACGACGATGACAAGGGAGTT…
ilp8-HF …CAAACGTACCCATACGATGTTCCAGATTACGCTAAACGA……CGAAGAGACTACAAGGACGACGATGACAAGAACATT…

	Injected-G0	G0-adult	G0-female	G0-F-HA	Injected-G1	G1-adult	G1-female	G1-F-HA	G1-F-HF
ilp1	1500	58%	30%	17%	2100	64%	32%	2.02%	1.29%
ilp2	1500	52%	27%	17%	2100	67%	36%	2.59%	1.36%
ilp3	1500	56%	28%	16%	2100	66%	32%	2.37%	1.27%
ilp4	1500	67%	32%	18%	2100	75%	37%	2.17%	1.30%
ilp5	1500	55%	28%	16%	2100	60%	31%	2.68%	1.59%
ilp6	1500	59%	31%	18%	2100	68%	35%	1.71%	1.27%
ilp7	1500	66%	32%	17%	2100	62%	32%	1.87%	1.22%
ilp8	1500	60%	31%	16%	2100	69%	36%	2.15%	1.26%


A
ILP1 (pg/mg)
ILP2 (pg/mg)
ILP3 (pg/mg)
ILP4 (pg/mg)
ILP5 (pg/mg)
ILP6 (pg/mg)
ILP7 (pg/mg)
ILP8 (pg/mg)
NT
dsLuc
dsMet
dsKr-h1
B
ILP1 (pg/mg)
ILP2 (pg/mg)
ILP3 (pg/mg)
ILP4 (pg/mg)
ILP5 (pg/mg)
ILP6 (pg/mg)
ILP7 (pg/mg)
ILP8 (pg/mg)
NT
dsLuc
dsEcR
dsE74


A
ILP1 (pg/μL)
ILP3 (pg/μL)
ILP7 (pg/μL)
ILP8 (pg/μL)
NT
dsLuc
dsMet
dsKr-h1
B
ILP1 (pg/μL)
ILP3 (pg/μL)
ILP7 (pg/μL)
ILP8 (pg/μL)
NT
dsLuc
dsEcR
dsE74


A
Met
RE to RP7S
0.08
0.06
0.04
0.02
0.00
**
72PE dsLuc
72PE dsMet
Kr-h1
RE to RP7S
0.010
0.008
0.006
0.004
0.002
0.000
**
72PE dsLuc
72PE dsKr-h1
EcR
RE to RP7S
0.025
0.020
0.015
0.010
0.005
0.000
**
24PBM dsLuc
24PBM dsEcR
E74
RE to RP7S
0.010
0.008
0.006
0.004
0.002
0.000
**
24PBM dsLuc
24PBM dsE74
B
FoxO
RE to RP7S
0.03
0.02
0.01
0.00
**
72PE dsLuc
72PE dsFoxO


-3000
-2500
-2000
-1500
-1000
-500
0
SCS
TPS
MDH
Lipase
FAS
FoxO-HA
dsFoxO-HA
Fold binding over IgG
SCS
TPS
MDH
Lipase
FAS
300
600
900
1200
1500
1800
2100
2400
2700
3000