

Supplementary Table 1

Mouse Model	Body Weight/Length/Composition	Food Intake/Energy Expenditure	Reference
Paternal <i>Snord116</i> deletion (Brosius)	Growth retardation Small body size <ul style="list-style-type: none"> •Growth retardation from P5 •Low BW into adulthood •No obesity 	N/A	Skryabin et al. 2007 (1) Lassi et al. 2015 (2)
Paternal <i>Snord116</i> deletion (Francke)	Growth retardation Small body size <ul style="list-style-type: none"> •Growth retardation from P2; growth rate normalizes post-weaning, but weight differences remain. •Smaller BW (40% smaller than WT) through adulthood •Decreased body length •Lower fat mass (DEXA) •Low liver/stomach weight postnatally. 	↑ Food intake* ↑ Energy expenditure* Resistant to HFD and calorie restriction <ul style="list-style-type: none"> •Ding: No difference in fast-refeeding; males 48 hours from food reintroduction have slightly higher cumulative FI •Lin: Higher fast-refeeding FI from 100mins post food re-introduction 	Ding et al. 2008 (3) Lin et al. 2014 (4)
Paternal <i>Snord116</i> deletion (Francke)	Small body size All mass components ↓ <ul style="list-style-type: none"> •Low BW through 22 weeks of age. •Weight lower across fat, lean and total mass. 	No diffs in food intake (12h average) ↑ Energy expenditure* <ul style="list-style-type: none"> •Increased day and night EE, but not independent of BW via ANCOVA. •Decreased RER in light hours, normal RER cycling. 	Powell et al. 2013 (5)
Biallelic <i>Snord116</i> deletion (Francke)	Small body size ↓ fat mass; ↑ lean mass <ul style="list-style-type: none"> •Low BW through 33 weeks of age. •Altered body composition in late adulthood (lower fat mass; slightly increased lean mass) <p><u>At 30°C:</u> Low body weight remains No difference in body length</p>	↑ Food intake* ↑ Energy expenditure* Resistant to HFD and calorie restriction <ul style="list-style-type: none"> •Increased EE (light hours) only in early adulthood. •Increased fast-refeeding food intake only 48hrs from food re-introduction. <p><u>At 30°C:</u> No differences in food intake* No differences in energy expenditure*</p>	Qi et al. 2016 (6) Qi et al. 2017 (7)
Biallelic <i>Snord116</i> deletion (Francke) in NPY neurons	Small body size ↓ fat mass <ul style="list-style-type: none"> •Lower BW through 17 weeks of age. •Shorter body length <p><u>At 30°C:</u> No difference in body length</p>	↑ Food intake* ↑ Energy expenditure* <ul style="list-style-type: none"> •Increased daytime RER <p><u>At 30°C:</u> No differences in food intake* No differences in energy expenditure* <ul style="list-style-type: none"> •Lower RER and increase physical activity </p>	Qi et al. 2016 (6) Qi et al. 2017 (7)
<i>Snord116</i> deletion (Brosius); HPRT knock-in upstream maternal <i>Snord116</i>	“Rescue” growth delay <ul style="list-style-type: none"> •Slight growth delay between P7-P19. •No difference in body weight compared to WT pups from 3 weeks old. 	N/A	Rozhdestvensky et al. 2016 (8)
Tamoxifen-induced <i>Snord116</i> deletion	No difference in total body weight ↑ White adipose tissue	↓ Food intake* ↓ Refeeding food intake*	Purtell et al. 2017 (9)
Hypothalamic re-expression of <i>Snord116</i> in biallelic <i>Snord116</i> deletion	↓ Body weight gain ↓ Body length <ul style="list-style-type: none"> •No difference in fat or lean mass 	<u>At room temperature:</u> No difference in food intake* No difference in refeeding food intake* ↑ EE* <u>At 30°C:</u> ↑Refeeding food intake at 48 hours* ↑ EE*	Qi et al. 2017 (10)

Supplementary References

1. Skryabin BV et al. Deletion of the MBII-85 snoRNA gene cluster in mice results in postnatal growth retardation. *PLoS Genet.* 2007;3(12):e235.
2. Lassi G et al. Deletion of the Snord116/SNORD116 Alters Sleep in Mice and Patients with Prader-Willi Syndrome. *Sleep* 2016;39(3):637–644.
3. Ding F et al. SnoRNA Snord116 (Pwcr1/MBII-85) deletion causes growth deficiency and hyperphagia in mice. *PLoS ONE* 2008;3(3):e1709.
4. Lin D et al. Abnormal response to the anorexic effect of GHS-R inhibitors and exenatide in male Snord116 deletion mouse model for Prader-Willi syndrome. *Endocrinology* 2014;155(7):2355–2362.
5. Powell WT et al. A Prader-Willi locus lncRNA cloud modulates diurnal genes and energy expenditure. *Hum. Mol. Genet.* 2013;22(21):4318–4328.
6. Qi Y et al. Snord116 is critical in the regulation of food intake and body weight. *Sci Rep* 2016;6:18614.
7. Qi Y et al. Ambient temperature modulates the effects of the Prader-Willi syndrome candidate gene Snord116 on energy homeostasis. *Neuropeptides* 2017;61:87–93.
8. Rozhdestvensky TS et al. Maternal transcription of non-protein coding RNAs from the PWS-critical region rescues growth retardation in mice. *Sci Rep* 2016;6:20398.
9. Purtell L et al. Adult-onset deletion of the Prader-Willi syndrome susceptibility gene Snord116 in mice results in reduced feeding and increased fat mass. *Transl Pediatr* 2017;6(2):88–97.
10. Qi Y et al. Hypothalamus specific re-introduction of Snord116 into otherwise Snord116 deficient mice increased energy expenditure. *Journal of Neuroendocrinology* [published online ahead of print: January 17, 2017]; doi:10.1111/jne.12457