Appendix 1 to Lee KWK, Abrahamowicz M, Leonard GT, et al. Prenatal exposure to cigarette smoke interacts with *OPRM1* to modulate dietary preference for fat. *J Psychiatry Neurosci* 2014.

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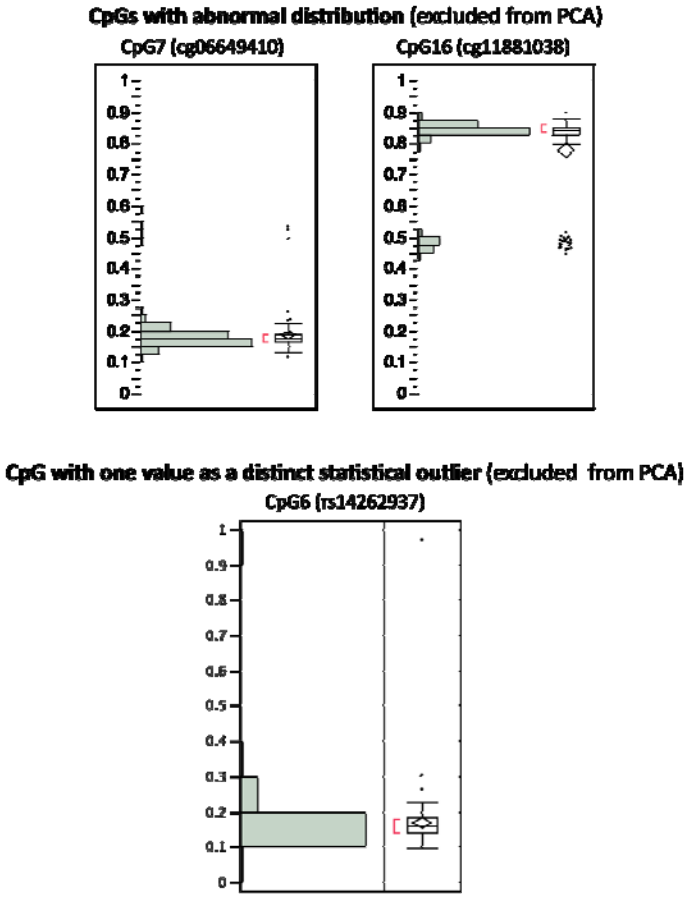

Fig. S1: CpGs with abnormal distribution of DNA methylation- (DNAm-) values (excluded from principal component analysis [PCA]). Distributions of unadjusted DNAm- values from 132 tested adolescents are shown. CpG7 and CpG16 were excluded from PCA. One outlier value from CpG6 was excluded from PCA.

Table S1: *OPRM1* × PEMCS interaction: additional models

	p value*								
	Model 1†			Model 2‡			Model 3§		
24-hour food intake	*OPRM1*	PEMCS	*OPRM1*× PEMCS	*OPRM1*	PEMCS	*OPRM1*× PEMCS	*OPRM1*	PEMCS	*OPRM1*× PEMCS
Fat, % energy	0.11	0.001	0.003	0.08	< 0.001	0.003	0.07	< 0.001	0.003
Carbohydrate, % energy	0.29	0.005	0.003	0.19	< 0.001	0.004	0.18	< 0.001	0.003
Protein, % energy	0.74	0.60	0.64	0.90	0.97	0.64	0.92	0.93	0.59
Energy, kcal	0.28	0.13	0.11	0.32	0.08	0.09	0.29	0.10	0.07

*p values of the linear regression models between *OPRM1* rs2281617 genotype, prenatal exposure to maternal cigarette smoking (PEMCS), or their interaction, and each of the 24-hour food intake outcomes (fat, carbohydrate, protein, and energy intake) are presented.
†Model 1: age + sex
‡Model 2: age + sex + birth weight (adjusted for gestation duration) + breastfeeding duration
§Model 3: age + sex + birth weight (adjusted for gestation duration) + breastfeeding duration + current smoking (defined as smoking at least 1 cigarette in the last 30 days)

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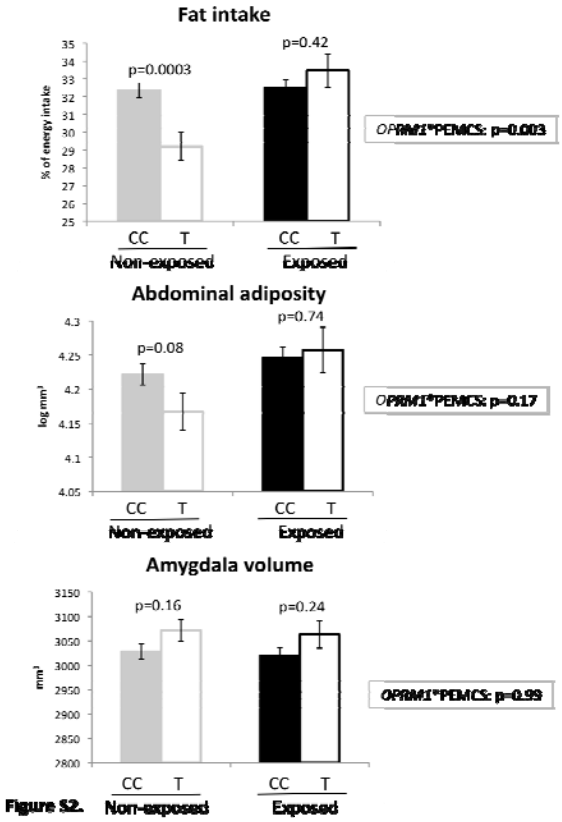

Figure S2.

Fig. S2: Association of *OPRM1* (rs2281617) and PEMCS with fat intake, abdominal adiposity and amygdala volume. Means ± standard errors of the mean, adjusted for age, sex and perinatal variables (birth weight, adjusted for gestation duration, and breastfeeding duration) are shown. Abdominal adiposity was assessed as a volume of visceral fat at the level of the umbilicus measured with magnetic resonance imaging, as described previously.[1] Amygdala volume was also measured with MRI, as described previously.[2]

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Table S2: Basic characteristics of epigenotyped adolescents

Characteristic	Group; mean ± SD*		*p* value†
	Nonexposed	Exposed	
Sex, males:females	33:33	33:33	—
Current			
Age, yr	15.6 1.5	15.5 1.4	0.95
Family income, $CAD/yr	60 384 24 387	58 939 25 548	0.74
Current smoking, yes:no‡	2:61	5:60	0.26
Perinatal			
PECMS, cigarettes/d	0	8.9 3.9	—
Gestational duration, wk	39.6 1.5	39.6 1.6	0.83
Birth weight, g	3442 406	3275 501	0.042
Breastfeeding duration, wk	11.5 14.8	6.7 12.0	0.039

PEMCS = prenatal exposure to maternal cigarette smoking; SD = standard deviation.
*Unless otherwise indicated.
†We used the chi-square test for categorical variables and the *t* test for continuous variables.
‡Adolescents were classified as currently smoking if they reported to have smoked at least 1 cigarette in the last 30 days. Data were missing from 4 adolescents.

References

1. Syme C, Abrahamowicz M, Mahboubi A, et al. Prenatal exposure to maternal cigarette smoking and accumulation of intra-abdominal fat during adolescence. *Obesity (Silver Spring)* 2010;18:1021-5.
2. Haghighi A, Melka MG, Bernard M, et al. Opioid receptor mu 1 gene, fat intake and obesity in adolescence. *Mol Psychiatry* 2014;19:63-8.