9èmes Rencontres de la

SFEDP
Société Française d'Endocrinologie et Diabétologie Pédiatrique

LES RENCONTRES EN ENDOCRINOLOGIE PÉDIATRIQUE

ANNÉE EN ENDOCRINOLOGIE PÉDIATRIQUE 2018 : OBESITE

Maithé Tauber
Hôpital des enfants
CHU Toulouse

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 OCTOBER 4, 2018 VOL. 379 NO. 14

Acceleration of BMI in Early Childhood and Risk of Sustained Obesity

Mandy Geserick, M.Sc., Mandy Vogel, Ph.D., Ruth Gausche, M.B.A., Tobias Lipek, M.D., Ulrike Spielau, M.Sc., Eberhard Keller, M.D., Roland Pfäffle, M.D., Wieland Kiess, M.D., and Antje Körner, M.D.

METHODS

To assess the age at onset of obesity, we performed prospective and retrospective analyses of the course of BMI over time in a population-based sample of 51,505 children for whom sequential anthropometric data were available during childhood (0 to 14 years of age) and adolescence (15 to 18 years of age). In addition, we assessed the dynamics of annual BMI increments, defined as the change in BMI standard-deviation score per year, during childhood in 34,196 children.

CONCLUSIONS

Among obese adolescents, **the most rapid weight gain had occurred between 2 and 6 years of age**; most children who were obese at that age were obese in adolescence.
(Funded by the German Research Council for the Clinical Research Center “Obesity Mechanisms” and others; ClinicalTrials.gov number, NCT03072537.)

Review

Overweight, obesity and adiposity in survivors of childhood brain tumours: a systematic review and meta-analysis

K-W. Wang[1,2,3], A. Fleming[1,3,4], D. L. Johnston[5], S. M. Zelcer[6], S. R. Rassekh[7], S. Ladhani[1,2], A. Socha[1,2], J. Shinuda[1,2], S. Jaber[1,2], S. Burrow[8], S. K. Singh[9,10], L. Banfield[11], R. J. de Souza[3,12], L. Thabane[3,12,13,14,15] and M. C. Samaan[1,2,3,12]

Forty-one studies were included in the meta-analysis. The prevalence of overweight and obesity combined was similar between overall SCBT, SCBT excluding craniopharyngioma and non-cancer controls

We conclude that SCBT have similar overweight and obesity distribution but higher adiposity than non-cancer controls and higher W/T ratio . More studies were needed to explore the determinants of adiposity and its contribution to cardiometabolic outcomes in SCBT

Clinical Obesity 8, 55–67, February 2018

ADCY3, neuronal primary cilia and obesity

Two new studies identify rare homozygous variants in *ADCY3* that are causal for monogenic obesity in consanguineous families of Pakistani origin and are associated with increased risk of obesity in Greenlandic individuals. Greenlandic carriers of homozygous loss-of-function variants in *ADCY3*, and individuals from trans-ancestry studies with a burden of rare *ADCY3* loss-of-function variants, also have increased risk of type 2 diabetes

nature genetics

LETTERS

https://doi.org/10.1038/s41588-017-0023-6

Loss-of-function mutations in *ADCY3* cause monogenic severe obesity

NATURE GENETICS | VOL 50 | FEBRUARY 2018

Sadia Saeed[1,2], Amélie Bonnefond[1], Filippo Tamanini[2], Muhammad Usman Mirza[3], Jaida Manzoor[4], Qasim M. Janjua[5], Sadia M. Din[6], Julien Gaitan[7,8], Alexandra Milochau[7,8], Emmanuelle Durand[1], Emmanuel Vaillant[1], Attiya Haseeb[6], Franck De Graeve[1], Iandry Rabearivelo[1], Olivier Sand[1], Gurvan Queniat[1], Raphaël Boutry[1], Dina A. Schott[9], Hina Ayesha[10], Muhammad Ali[11], Waqas I. Khan[12], Taeed A. Butt[13], Tuula Rinne[14], Connie Stumpel[15], Amar Abderrahmani[1,2], Jochen Lang[7,8], Muhammad Arslan[5,6] and Philippe Froguel[1,2*]

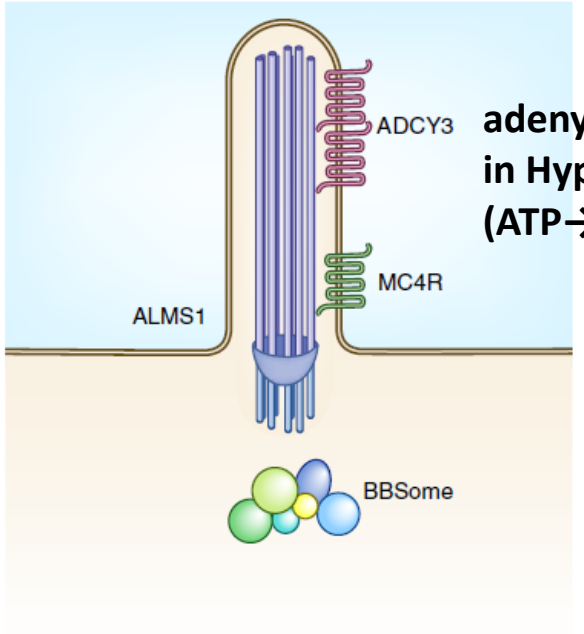
LETTERS

https://doi.org/10.1038/s41588-017-0020-9

nature genetics

Subcellular localization of MC4R with ADCY3 at neuronal primary cilia underlies a common pathway for genetic predisposition to obesity

Jacqueline E. Siljee[1], Yi Wang[1], Adelaide A. Bernard[1], Baran A. Ersoy[1,4], Sumei Zhang[1], Aaron Marley[2], Mark Von Zastrow[2], Jeremy F. Reiter[3] and Christian Vaisse[1*]



adenylate cyclase 3 in Hypothalamus (ATP→AMPc)

Fig. 1 | ADCY3 and MC4R colocalize to primary neuronal cilia where other ciliopathy-related proteins also function, including the Alström syndrome (ALMS1) and Bardet–Biedl proteins (collectively knows as the BBSome). Credit: Marina Corral Spence/Springer Nature.

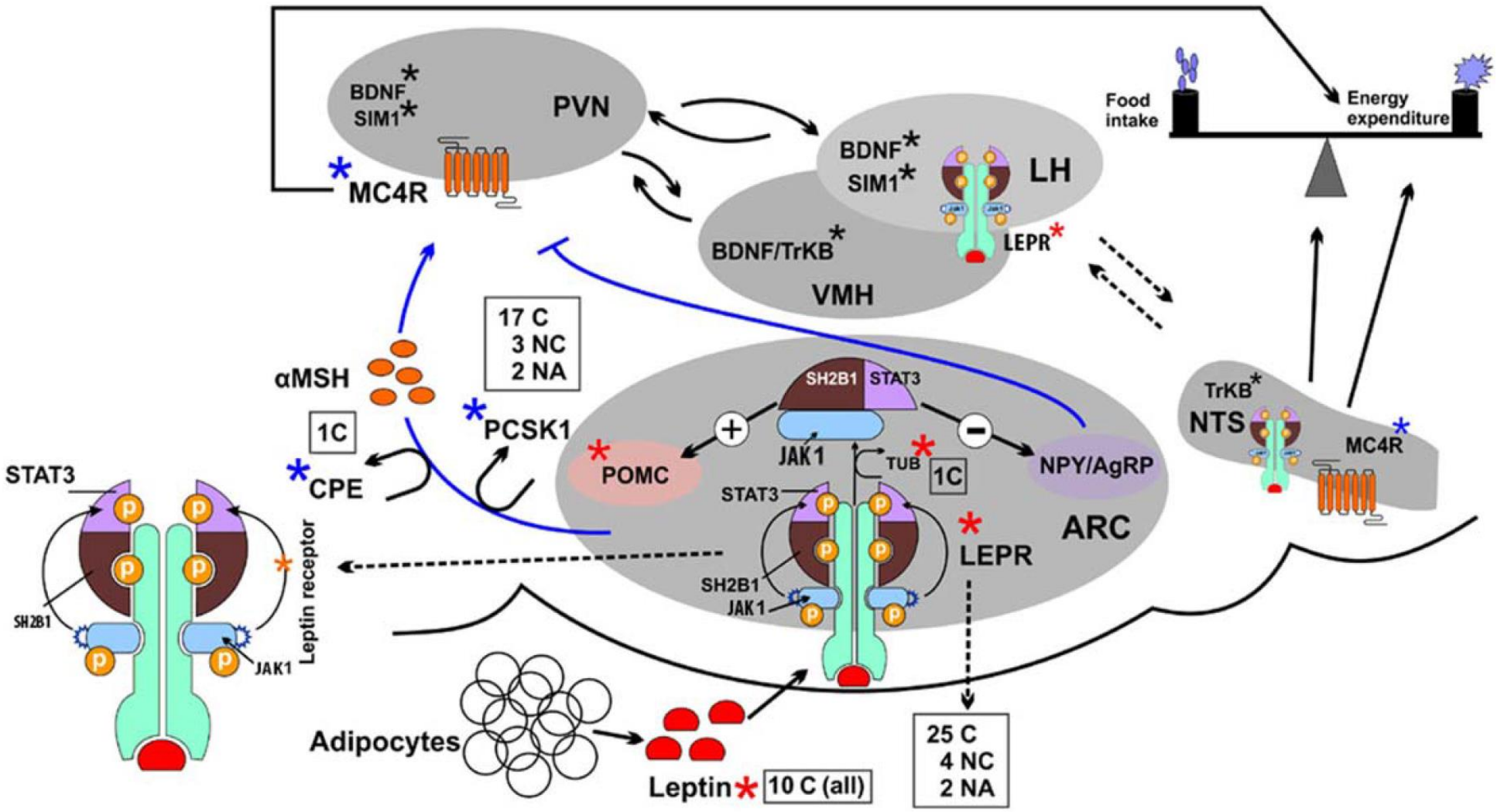
nature genetics

Loss-of-function variants in *ADCY3* increase risk of obesity and type 2 diabetes

Niels Grarup#1, **Ida Moltke**#2, **Mette K. Andersen**1, **Maria Dalby**2, **Kristoffer Vitting-Seerup**2,3, **Timo Kern**1, **Yuvaraj Mahendran**1, **Emil Jørsboe**2, **Christina V. L. Larsen**4,5, **Inger K. Dahl-Petersen**4, **Arthur Gilly**6, **Daniel Suveges**6, **George Dedoussis**7, **Eleftheria Zeggini**6, **Oluf Pedersen**1, **Robin Andersson**2, **Peter Bjerregaard**4,5, **Marit E. Jørgensen**4,5,8, **Anders Albrechtsen**2, and **Torben Hansen**1

Genetics of Obesity in Consanguineous Populations

Sadia Saeed , Muhammad Arslan and Philippe Froguel



Mutations identified in coding genes are marked with a colored asterisk (*); red: autosomal **recessive** inheritance; blue: autosomal **dominant**/ recessive inheritance; black: autosomal **dominant** inheritance. Obesity | VOLUME 26 | NUMBER 3 | MARCH 2018

Diabetes 2019;68:724–732

A Candidate-Gene Approach Identifies Novel Associations Between Common Variants in/Near Syndromic Obesity Genes and BMI in Pediatric and Adult European Populations

Wang DX, Kaur Y , Alyass A and Meyre D

Table 1—The top significant SNPs of their associated gene from single-marker analysis ($P < 2.8 \times 10^{-6}$)

SNP	Chr:position	Gene	Alleles	EAF	β	SE	*P*	*N*
rs6265	11:27679916	*BDNF*	T/C	0.1951	−0.0412	0.0021	1.0×10^{-86}	795,458
rs7498665	16:28883241	*SH2B1*	A/G	0.5962	−0.0271	0.0017	5.6×10^{-60}	790,299
rs3814883	16:29994922	*KCTD13*	T/C	0.4764	0.0232	0.0017	1.1×10^{-40}	685,519
rs879620	16:4015729	*CREBBP*	T/C	0.6179	0.0231	0.0018	5.3×10^{-38}	688,377
rs7164727	15:73093991	*BBS4*	T/C	0.681	0.0182	0.0017	3.3×10^{-25}	791,156
rs946824	1:243684019	*SDCCAG8*	T/C	0.141	0.0206	0.0026	1.1×10^{-15}	689,849
rs12448738	16:56489343	*BBS2*	A/C	0.8629	−0.0168	0.0025	2.8×10^{-11}	691,932
rs12206564	6:100987009	*SIM1*	T/C	0.505	−0.0113	0.0017	5.4×10^{-11}	689,836
rs1187352	9:87293457	*NTRK2*	T/C	0.3482	−0.0119	0.0018	6.0×10^{-11}	688,522
rs1260326	2:27730940	*IFT172*	T/C	0.4027	−0.0105	0.0017	3.9×10^{-10}	784,462
rs11792069	9:140646121	*EHMT1*	A/G	0.83	0.0145	0.0024	6.5×10^{-10}	644,252
rs7975791	12:49413486	*KMT2D*	T/C	0.03601	−0.0264	0.0043	1.1×10^{-9}	770,139
rs12628891	22:38317137	*SOX10*	T/C	0.3169	−0.0112	0.0019	3.0×10^{-9}	686,575
rs12478556	2:63341711	*WDPCP*	A/T	0.4177	0.0103	0.0018	4.5×10^{-9}	683,709
rs11649864	17:56093061	*MKS1*	A/G	0.09127	0.0178	0.0031	6.7×10^{-9}	663,638
rs139531	22:41676176	*EP300*	A/G	0.7197	0.0112	0.0019	9.5×10^{-9}	690,810
rs881301	8:38332318	*FGFR1*	T/C	0.5818	−0.0097	0.0017	2.4×10^{-8}	691,753
rs6901944	6:56862805	*RAB23*	A/G	0.8344	−0.0126	0.0023	2.7×10^{-8}	692,035
rs6910117	**6:84971679**	***MRAP2***	**A/T**	**0.9099**	**−0.0157**	**0.0029**	$\mathbf{9.1 \times 10^{-8}}$	**643,225**
rs12805133	**11:66483265**	***BBS1***	**A/G**	**0.5426**	**−0.0091**	**0.0018**	$\mathbf{3.5 \times 10^{-7}}$	**635,477**
rs6026567	**20:57444915**	***GNAS***	**A/G**	**0.571**	**−0.0092**	**0.0018**	$\mathbf{4.4 \times 10^{-7}}$	**659,708**
rs9610560	**22:37064378**	***IFT27***	**A/G**	**0.7375**	**−0.0099**	**0.002**	$\mathbf{5.1 \times 10^{-7}}$	**671,468**
rs17765088	**3:45943595**	***LZTFL1***	**C/G**	**0.8817**	**−0.0136**	**0.0027**	$\mathbf{5.7 \times 10^{-7}}$	**666,309**
rs13171414	**5:37232079**	***NIPBL***	**A/G**	**0.7836**	**0.0103**	**0.0021**	$\mathbf{7.2 \times 10^{-7}}$	**692,376**
rs11161347	**15:23948049**	***MAGEL2/MKRN3/NDN***	**A/G**	**0.4831**	**0.0085**	**0.0018**	$\mathbf{1.1 \times 10^{-6}}$	**672,682**
rs13311608	**7:32999601**	***BBS9***	**A/G**	**0.5147**	**−0.0083**	**0.0017**	$\mathbf{1.3 \times 10^{-6}}$	**688,219**
rs752579	17:17660347	*RAI1*	T/C	0.6077	0.0082	0.0017	1.6×10^{-6}	781,905

Novel SNPs are shown in bold. Chromosome (Chr):position, Alleles (effect/other), effect allele frequency (EAF), beta (β), SE, *P* values, and number of observations (*N*) are reported. SNP positions are reported according to Build 37. See Supplementary Table 2 for the complete results from single-marker analysis.

- Single-marker, tagSNP, and gene-based 14 novel associations at 16 of 54 (29.6%)
- genome-wide significant SNPs were mapped to 19 of 54 (35.2%) syndromic obesity genes.
- A significant association for 17 of 33 (51.5%) loci was also observed with BMI in children.

« This study supports evidence for **a continuum between rare monogenic syndromic and common polygenic forms of obesity** »

Heterozygous rare genetic variants in non-syndromic early-onset obesity

Serra-Juhé C, Martos-Moreno GA , Bou de Pieri F, Flores R, Chowen JA, Pérez-Jurado LA and Argente J

	All RSVs			Likely pathogenic RSVs		
	EOO-Sp (%)	VLF (%)	Controls (%)	EOO-Sp (%)	VLF (%)	Controls (%)
ADRB3	1 (0.22)	1 (0.34)	4 (0.83)	–	–	1 (0.21)
BDNF	4 (0.86)	1 (0.34)	–	3 (0.65)	1 (0.34)	–
FTO	4 (0.86)	4 (1.37)	1 (0.21)	1 (0.22)	–	–
GHSR	2 (0.43)	6 (2.05)	3 (0.63)	–	1 (0.34)	–
LEP	–	–	–	–	–	–
LEPR	7 (1.51)	5 (1.71)	5 (1.04)	2 (0.43)	2 (0.68)	3 (0.63)
MC3R	3 (0.65)	3 (1.02)	–	2 (0.43)	1 (0.34)	–
MC4R	7 (1.51)	6 (2.05)	2 (0.42)	6 (1.30)	4 (1.37)	–
NEGR1	3 (0.65)	–	–	1 (0.22)	–	–
NTRK2	2 (0.43)	3 (1.02)	4 (0.83)	1 (0.22)	2 (0.68)	1 (0.21)
PCSK1	4 (0.86)	–	5 (1.04)	3 (0.65)	–	1 (0.21)
PCSK2	2 (0.43)	–	5 (1.04)	–	–	2 (0.42)
PPARG	3 (0.65)	–	–	2 (0.43)	–	–
SIM1	6 (1.30)	2 (0.68)	1 (0.21)	2 (0.43)	–	–
TMEM18	–	1 (0.34)	1 (0.21)	–	1 (0.34)	–
	48 (10.37)	**32 (10.92)**	**31 (6.46)**	**23 (4.97)**	**12 (4.10)**	**8 (1.67)**
Selected genes	30 (6.48)	16 (5.46)	4 (0.83)	17 (3.67)	6 (2.05)	–
Other genes	18 (3.89)	16 (5.46)	27 (5.63)	6 (1.30)	6 (2.05)	8 (1.67)

463 EOO patients
480 controls.
exome data from 293 EOO

rare single-nucleotide genetic variants (RSVs) in 5% of the EOO
7 of the 15 genes

Int J Obes 2019

REVIEW ARTICLE

Journal of Neuroendocrinology WILEY

Prader-Willi syndrome: A model for understanding the ghrelin system

Maithé Tauber[1,2,3] | Muriel Coupaye[4] | Gwenaelle Diene[1,5] | Catherine Molinas[1,2,3] | Marion Valette[1,2] | Veronique Beauloye[6]

Journal of Neuroendocrinology. 2019;00:e12728.

nature COMMUNICATIONS

MRAP2 regulates ghrelin receptor signaling and hunger sensing

Dollada Srisai[1,2,3], Terry C. Yin[1,2,3], Abigail A. Lee[1,2,3], Alix A.J. Rouault[1,2,3], Nicole A. Pearson[2,4], Justin L. Grobe[2,4] & Julien A. Sebag[1,2,3]

The melanocortin 2receptor accessory protein 2 (MRAP2) was previously shown to regulate energy homeostasis through the modulation of the activity of the **melanocortin-4 receptor and prokineticin receptors**.

In this study we identify MRAP2 as a partner of **ghrelin-GHSR1a signaling**. We show that MRAP2 interacts with GHSR1a and potentiates ghrelin-stimulated signaling both in vitro and in vivo. We demonstrate that in the absence of MRAP2, fasting fails to activate agouti-related protein neurons. In addition, we show that the orexigenic effect of ghrelin is lost in mice lacking MRAP2.

Our results suggest that MRAP2 is an **important modulator of the energy homeostasis machinery that operates through the regulation of multiple GPCRs throughout the hypothalamus.**

DOI: 10.1038/s41467-017-00747-

Nat Commun 2017

Loss-of-function mutations in *MRAP2* are pathogenic in hyperphagic obesity with hyperglycemia and hypertension

Morgane Baron[1], Julie Maillet[1], Marlène Huyvaert[1], Aurélie Dechaume[1], Raphaël Boutry[1], Hélène Loiselle[1], Emmanuelle Durand[1], Bénédicte Toussaint[1], Emmanuel Vaillant[1], Julien Philippe[1,19], Jérémy Thomas[2], Amjad Ghulam[2], Sylvia Franc[3,4], Guillaume Charpentier[3,4], Jean-Michel Borys[5], Claire Lévy-Marchal[6], Maïthé Tauber[7], Raphaël Scharfmann[8], Jacques Weill[9], Cécile Aubert[10], Julie Kerr-Conte[11], François Pattou[11], Ronan Roussel[12,13,14], Beverley Balkau[15,16], Michel Marre[13,17], Mathilde Boissel[1], Mehdi Derhourhi[1], Stefan Gaget[1], Mickaël Canouil[1], Philippe Froguel[1,18*] and Amélie Bonnefond[1,18*]

NATURE MEDICINE | VOL 25 | NOVEMBER 2019 | 1733–1738 | www.nature.com/naturemedicine

Overexpression of melanocortin 2 receptor accessory protein 2 (MRAP2) in adult paraventricular MC4R neurons regulates energy intake and expenditure

Giuseppe Bruschetta [1,2], Jung Dae Kim [1,2], Sabrina Diano [1,2,3,4,5,**], Li F. Chan [6,*]

Mice with global MRAP2 deletion and conditional MRAP2 deletion in SIM1 expressing neurons developed severe early onset of obesity (without detectable changes in food intake or energy balance) and rare loss-of function or missense heterozygous variants in MRAP2 were identified in humans with severe early-onset obesity

Conclusions: Our data indicate **a site-specific role for MRAP2 in PVN MC4R-expressing neurons** in potentiating MC4R neuronal activation at baseline conditions in the regulation of food intake and energy expenditure.

Mol Metab 2018

Melanocortin Receptor Accessory Protein 2-Induced Adrenocorticotropic Hormone Response of Human Melanocortin 4 Receptor

Lucia Soletto,1* Sergio Hernández-Balfagó,1* Ana Rocha,1 Patrick Scheerer,2
Gunnar Kleinau,2 and José Miguel Cerdá-Reverter1

Melanocortin 4 receptor (MC4R), a canonical melanocyte-stimulating hormone receptor, is the main responsible for monogenic obesity in humans.

Previous studies **in fish and avian species** showed that **MC4R becomes an ACTH receptor after interaction with the melanocortin receptor accessory protein 2 (MRAP2).**

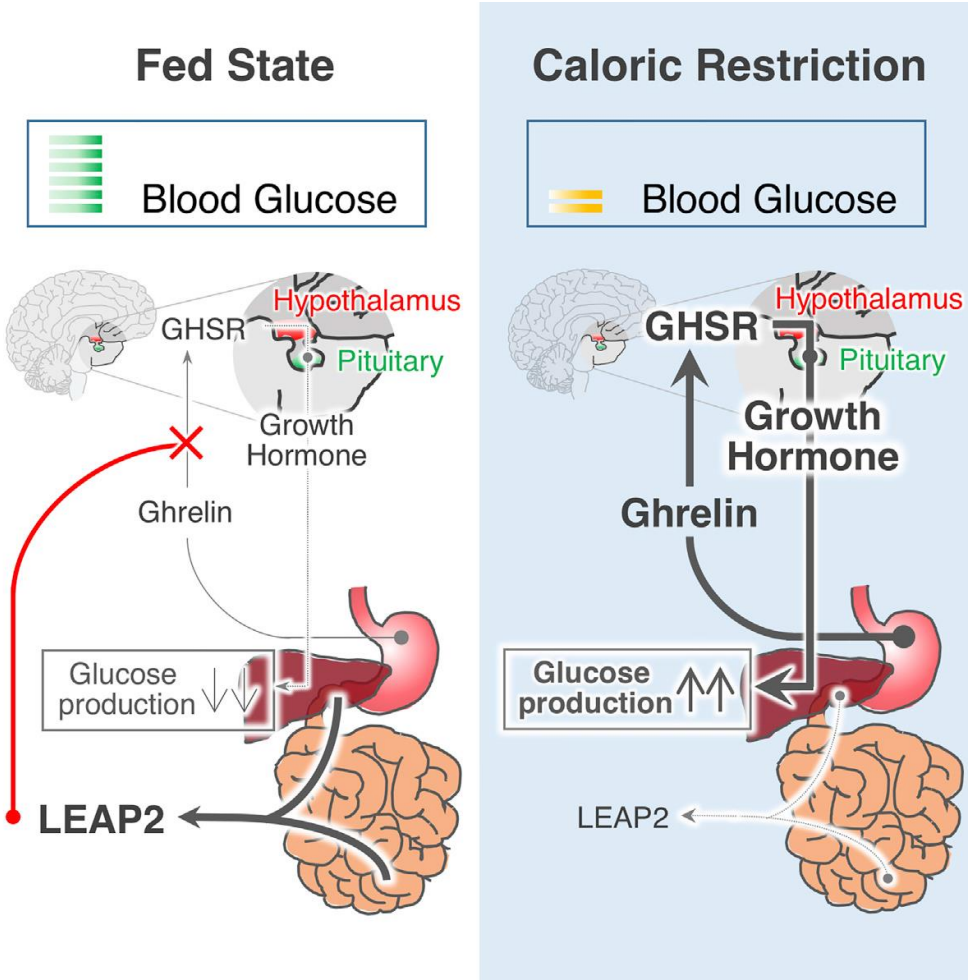
We show that human MC4R behaves in a similar way through its interaction with MRAP2.
This evolutionary conservation of MRAP2-induced ligand selectivity supports a physiological role for the interaction with MC4R. Both proteins are coexpressed in the same hypothalamic neurons

These neurons may link the effects of stress on the energy balance independently of glucocorticoid secretion. The complex MC4R-MRAP2 throws light on the action of ACTH and, by extension, on the relay of stress-related information to additional biological systems.

February 2019 | Vol. 3, Iss. 2
Journal of the Endocrine Society | 314–323

Cell Metabolism

LEAP2 Is an Endogenous Antagonist of the Ghrelin Receptor



LEAP2 : liver expressed antimicrobial peptide 2

Ghrelin maintains blood glucose levels in the face of starvation. Ge et al. identify LEAP2 as an **endogenous antagonist** of the ghrelin receptor that modulates ghrelin function in response to nutrient status, such as fasting. Increasing or suppressing LEAP2 leads to corresponding **counter-regulation of ghrelin action in vivo.**

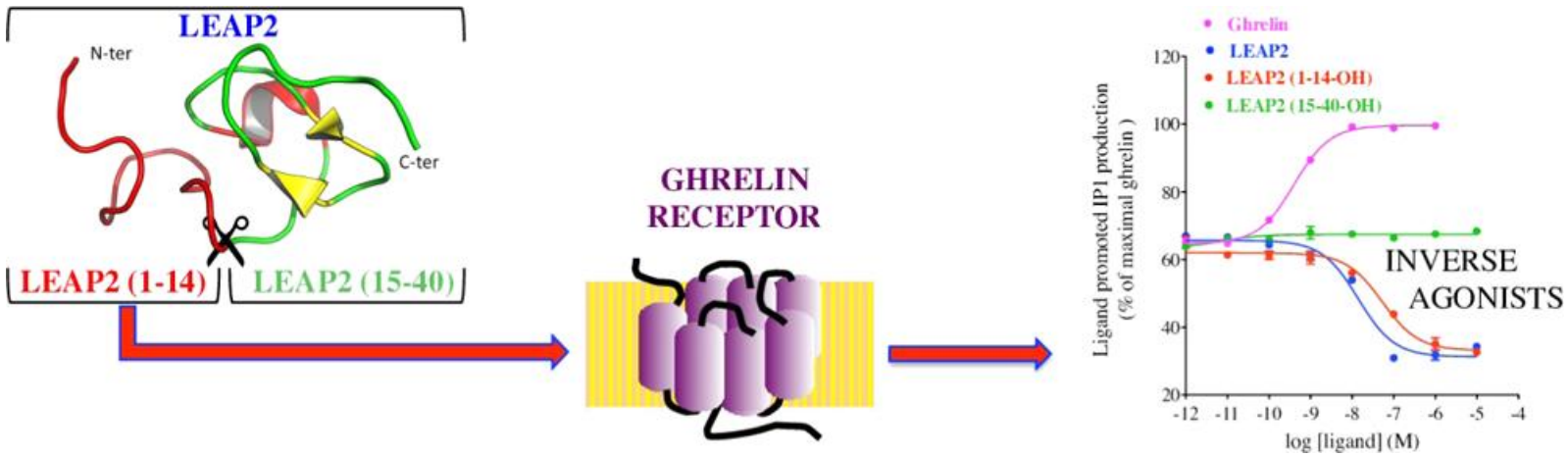
Ge et al., 2018, Cell Metabolism 27, 461–469
February 6, 2018 ª 2017 Elsevier Inc.

N-Terminal Liver-Expressed Antimicrobial Peptide 2 (LEAP2) Region Exhibits Inverse Agonist Activity toward the Ghrelin Receptor

Céline M'Kadmi,[†] Agustina Cabral,[‡] Franco Barrile,[‡] Julien Giribaldi,[†] Sonia Cantel,[†] Marjorie Damian,[†] Sophie Mary,[†] Séverine Denoyelle,[†] Sébastien Dutertre,[†] Sylvie Péraldi-Roux,[†] Jérémie Neasta,[†] Catherine Oiry,[†] Jean-Louis Banères,[†] Jacky Marie,[†] Mario Perello,[‡] and Jean-Alain Fehrentz*[,†]

Liver-expressed antimicrobial peptide 2 (LEAP2) was recently described as an endogenous antagonist of GHSR.
Both LEAP2 and its N-terminal part behave as inverse agonists of GHSR and as competitive antagonists of ghrelin-induced inositol phosphate production and calcium mobilization.
The N-terminal region of LEAP2 is able to inhibit ghrelin-induced food intake in mice. LEAP2 tunes the action of ghrelin in vivo
and is likely to have an important role in the control of ghrelin response under normal and pathological conditions.

(J. Med. Chem. 2019, 62, 965−973)

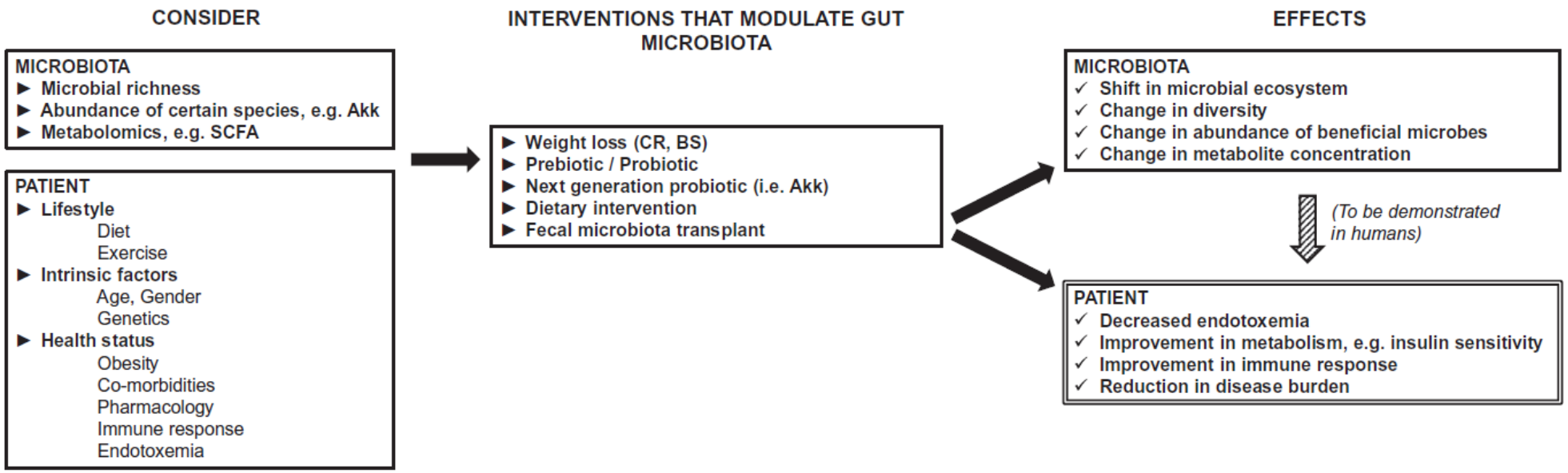


LEAP2 behaved as a competitive antagonist if added at the same time as the agonist and a non-competitive antagonist if added before the agonist. This unusual property of LEAP2 might be caused by its slow dissociation from receptor GHSR1a (Wang et al Febs j 2019)

Review Article

Gut microbiota and obesity: Concepts relevant to clinical care

Maria Carlota Dao*,[1], Karine Clément*



Gut bacteria species and host health: the example of Akkermansia . muciniphila

Currently Dr. Cani's group is conducting a clinical trial of A. muciniphila supplementation in overweight and obeseadults, hypothesizing that it will improve metabolic health (NCT02637115).

International Journal of Obesity (2019) 43:1120–1124
https://doi.org/10.1038/s41366-018-0150-x

BRIEF COMMUNICATION

Clinical Research

Variable oxytocin levels in humans with different degrees of obesity and impact of gastric bypass surgery

Zoltan Pataky[1] · Idris Guessous[2] · Aurélie Caillon[3] · Alain Golay[1] · Françoise Rohner-Jeanrenaud[3] · Jordi Altirriba[3]

Received: 1 February 2018 / Revised: 21 May 2018 / Accepted: 3 June 2018 / Published online: 13 July 2018

Abstract

Exogenous oxytocin administration in obese mice, rats, and monkeys was shown to induce sustained weight loss, mostly due to a decrease in fat mass, accompanied by an improvement of glucose metabolism. A pilot study in obese humans confirmed the weight-reducing effect of oxytocin. Knowledge about circulating oxytocin levels in human obesity might help indicating which obese subjects could potentially benefit from an oxytocin treatment. Conclusive results on this topic are missing. The aim of this study was to measure circulating oxytocin levels in lean ($n = 37$) and obese ($n = 72$) individuals across a wide range of body mass index (BMI) values (18.5–60 kg/m^2) and to determine the impact of pronounced body weight loss following gastric bypass surgery in 12 morbidly obese patients. We observed that oxytocin levels were unchanged in overweight and in class I and II obese subjects and only morbidly obese patients (obesity class III, BMI >40 kg/m^2) exhibited significantly higher levels than lean individuals, with no modification 1 year after gastric bypass surgery, despite substantial body weight loss. In conclusion, morbidly obese subjects present elevated oxytocin levels which were unaltered following pronounced weight loss.

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Current Neuropharmacology, 2018, *16*, 1111-1121

REVIEW ARTICLE

Oxytocin and Eating Disorders: A Narrative Review on Emerging Findings and Perspectives

Katrin Giel[a], Stephan Zipfel[a] and Manfred Hallschmid[b,c,d,*]

[a]*University Hospital Tübingen, Department of Psychosomatic Medicine and Psychotherapy, Tübingen, Germany;* [b]*Institute of Medical Psychology and Behavioral Neurobiology, University of Tübingen, Tübingen, Germany;* [c]*German Center for Diabetes Research (DZD), Tübingen, Germany;* [d]*Institute for Diabetes Research and Metabolic Diseases of the Helmholtz Center Munich at the University of Tübingen, Tübingen, Germany*

Obesity-Diabetes Management/Etiology and Pathophysiology

Oxytocin in metabolic homeostasis: implications for obesity and diabetes management

C. Ding[1], M. K.-S. Leow[1,2,3,5] and F. Magkos[1,4]

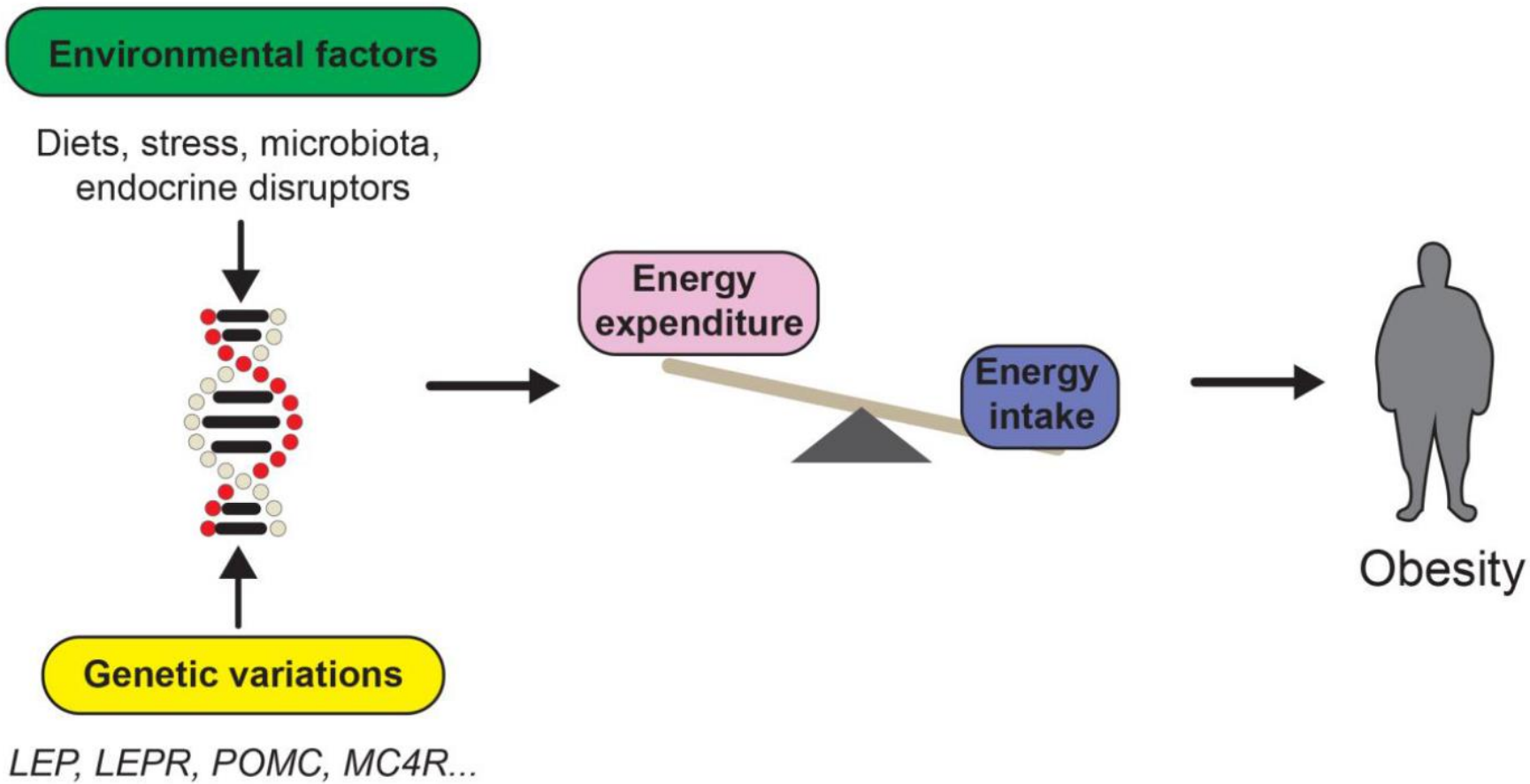
cell stress

Review

www.cell-stress.com

The role of epigenetics in hypothalamic energy balance control: implications for obesity

Arnaud Obri[1,*] and Marc Claret[1,2]



Evaluation of the Genetic Association Between Adult Obesity and Neuropsychiatric Disease

Priska Stahel,[1] Avital Nahmias,[1] Shawn K. Sud,[1] So Jeong Lee,[1] Andrea Pucci,[2,3,4] Ahmed Yousseif,[2,3,4] Alaa Youseff,[5] Timothy Jackson,[5,6] David R. Urbach,[6] Allan Okrainec,[6,7] Johane P. Allard,[8,9] Sanjeev Sockalingam,[7,10,11] Tony Yao,[12] Moumita Barua,[12] Hong Jiao,[13] Reedik Magi,[14] Anne S. Bassett,[15,16,17,18,19,20] Andrew D. Paterson,[10,21] Ingrid Dahlman,[12] Rachel L. Batterham,[2,3,4] and Satya Dash[1]

Diabetes 2019;68:2235–2246 | https://doi.org/10.2337/db18-1254

Received: 21 September 2018 | Accepted: 17 February 2019

DOI: 10.1111/ijpo.12522

ORIGINAL RESEARCH

WILEY pediatricobesity

Short-term and long-term positive outcomes of the multidisciplinary care implemented by the French health networks for the prevention and care of paediatric overweight and obesity

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