Adipositas & metabolisches Syndrom

Definition, Epidemiologie und Ursachen

Prof. T. Reinehr

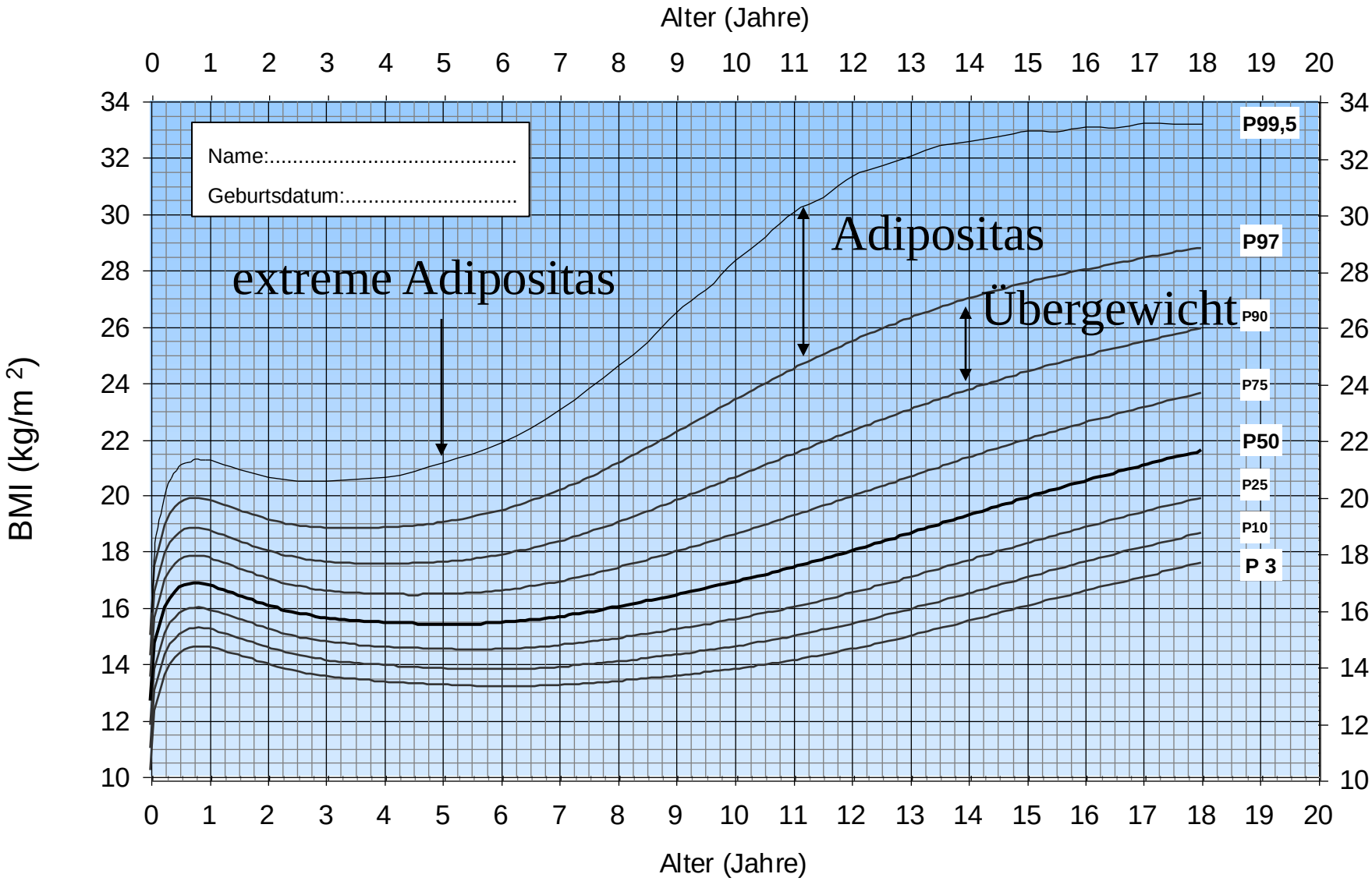
Abteilung für Pädiatrische Endokrinologie, Diabetologie und Ernährungsmedizin

Vestische Kinder- und Jugendklinik Datteln

Universität Witten/Herdecke

Definition und Epidemiologie der Adipositas

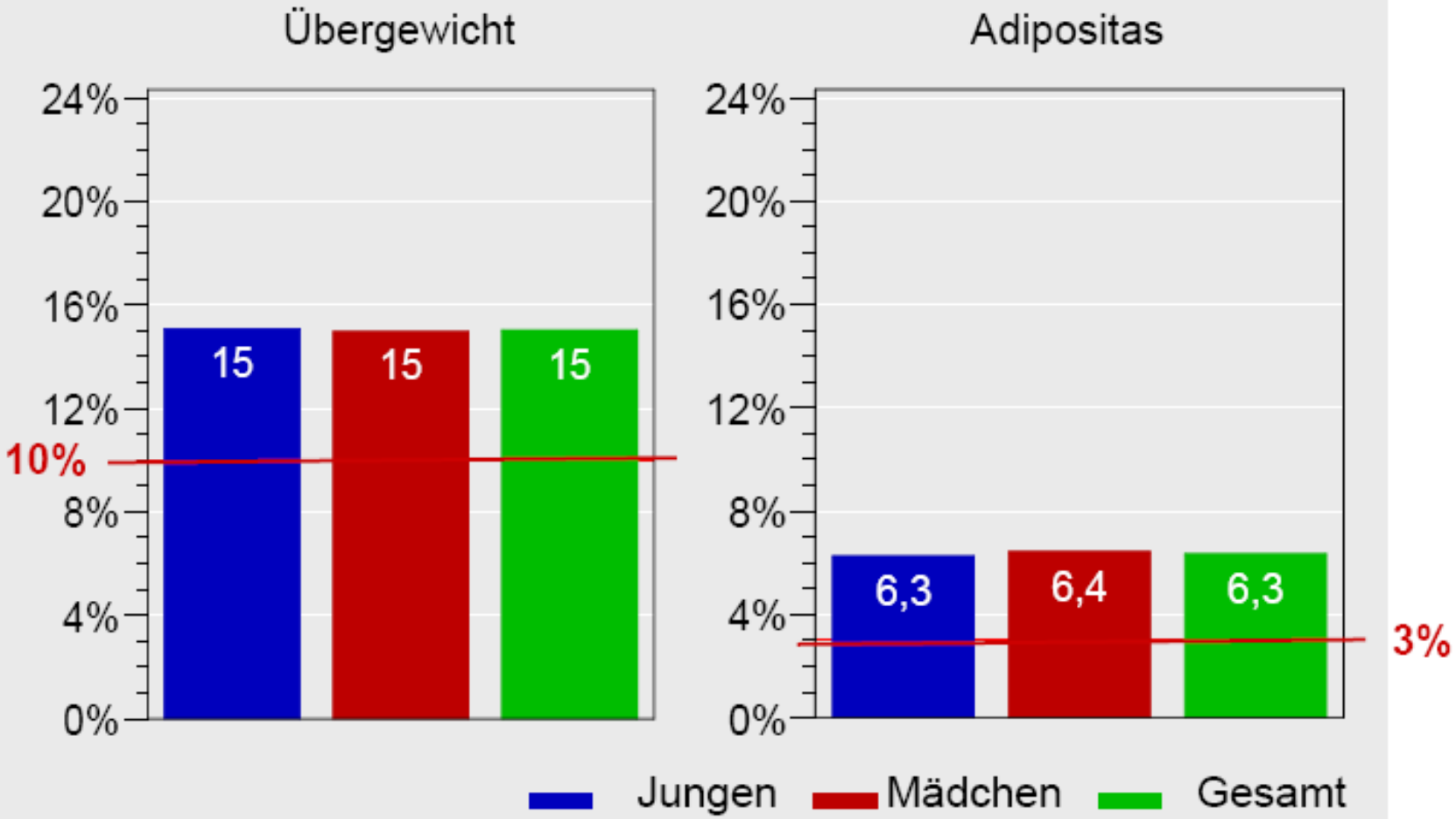
Perzentilkurven für den Body Mass Index (Jungen 0 - 18 Jahre)



K. Kromeyer-Hauschild, M. Wabitsch, D. Kunze et al.: Monatsschr. Kinderheilk. 149 (2001).

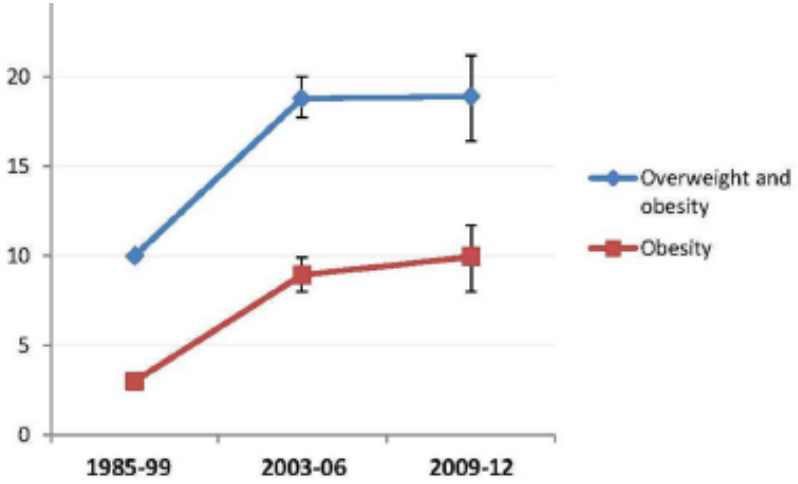
www.a-g-a.de

Ergebnisse: Häufigkeit von Übergewicht und Adipositas
Kinder und Jugendliche von 3-17 Jahren



Deutlicher Anstieg gegenüber Referenzdaten 1985-1999.
Keine Unterschiede zwischen Jungen und Mädchen.

Häufigkeit von Übergewicht und Adipositas bei Kindern und Jugendlichen in Deutschland (-KIGGS Studie-)



Brettschneidera et al.
BMC Public Health 2015;15:1101

Prävalenz der extremen Adipositas ist zunehmend in Europa !

- Nach Definition (>99.5 Perzentile BMI) **erwartet: <0.5%**
- **England** (Ells et al. *Arch Dis Child 2015* doi:10.1136)**: 2012/2013**
  - **2.9% der Mädchen und 3.9% der Jungen**
- **Niederlande** (van Dommelen et al *PLoS One. 2014*;14;9:e94299)**:2009**
  - **1.06% bei Niederländern**
  - **2.11% bei Türken**
  - **1.41% bei Marokkanern**

Die Gruppe extrem adipöser Jugendlicher in BRD (BMI ≥ 35 kg/m²; Alter:14-21 J.) umfasst in Deutschland ca. **182.000 Personen**.

Wann beginnt Adipositas im Kindesalter?

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.

ABSTRACT

BACKGROUND

The dynamics of body-mass index (BMI) in children from birth to adolescence are unclear, and whether susceptibility for the development of sustained obesity occurs at a specific age in children is important to determine.

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.

RESULTS

In retrospective analyses, we found that most of the adolescents with normal weight had always had a normal weight throughout childhood. Approximately half (53%) of the obese adolescents had been overweight or obese from 5 years of age onward, and

From the Center for Pediatric Research, University Hospital for Children and Adolescents (M.G., T.L., U.S., R.P., W.K., A.K.), Leipzig Research Center for Civilization Diseases (LIFE Child) (M.G., M.V., W.K., A.K.), CrescNet, Medical Faculty (R.G., E.K., R.P.), and Integrated Research and Treatment Center (IFB), Adiposity Diseases, University Medical Center (T.L., U.S., A.K.), University of Leipzig, Leipzig, Germany. Address reprint requests to Dr. Körner at the Center for Pediatric Research, University Hospital for Children and Adolescents, University of Leipzig, Liebigstr. 20a 04103 Leipzig, Germany, or at antje.koerner@medizin.uni-leipzig.de.

Drs. Geserick and Vogel contributed equally to this article.

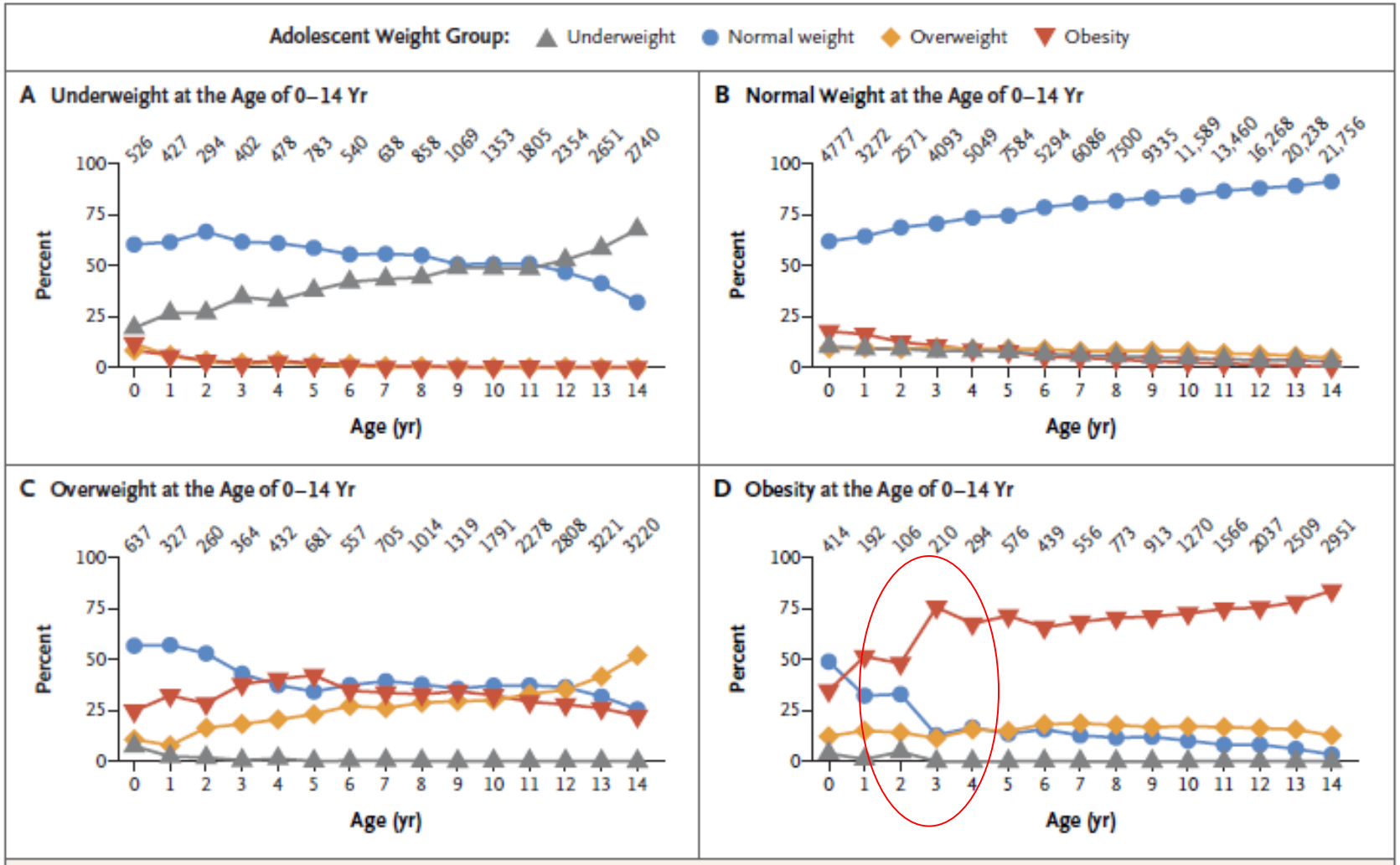
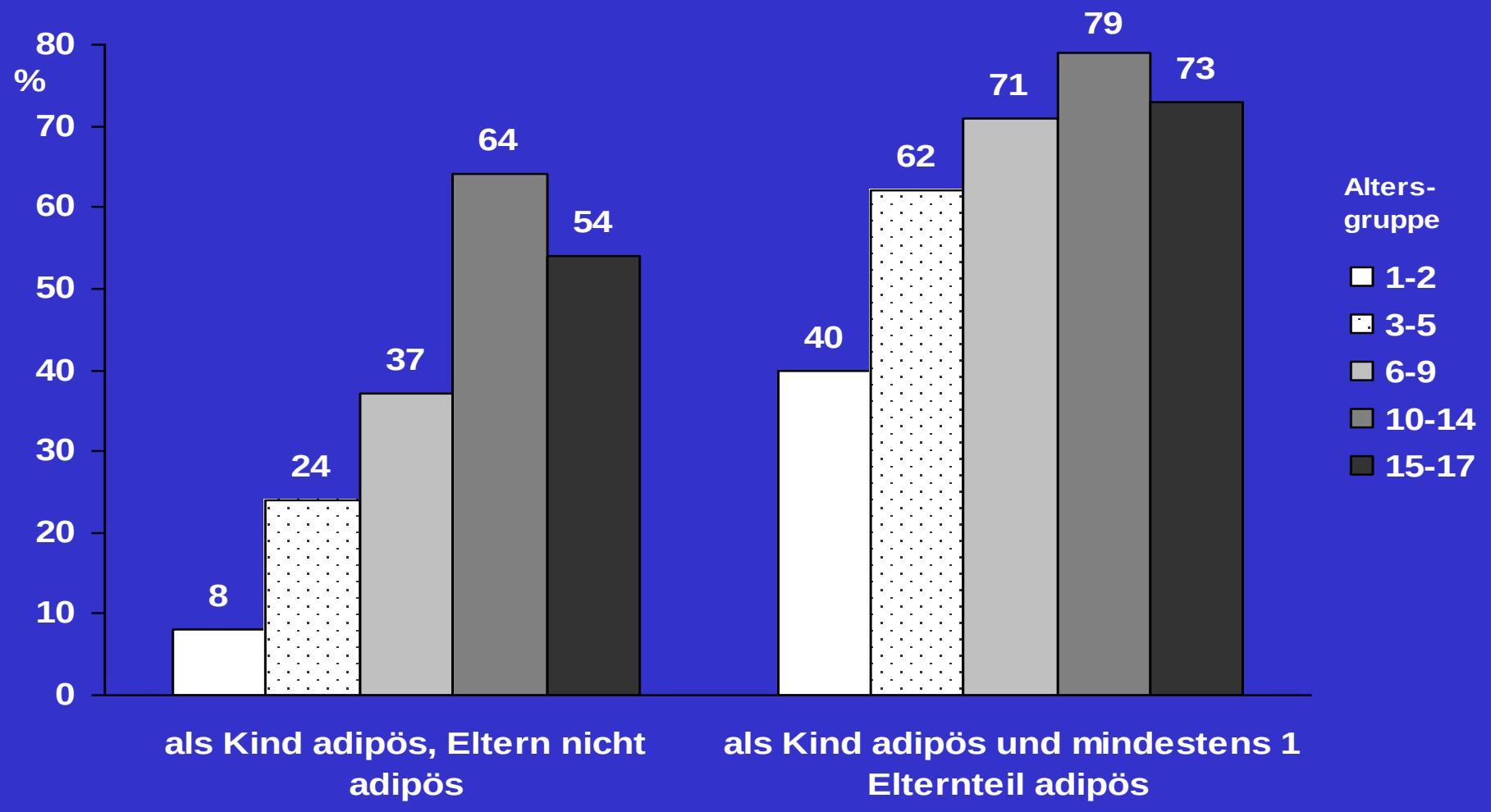


Figure 2. Prospective Tracking of BMI Status of Children into Adolescence.

Shown are the results of the prospective analysis, in which the percentage of adolescents in each weight-group category is shown relative to the weight group — underweight (Panel A), normal weight (Panel B), overweight (Panel C), or obese (Panel D) — during childhood. For example, until the age of 2 years, approximately half the children with overweight returned to a normal weight in adolescence; 62% of the children who were overweight at the age of 4 years remained overweight (21%) or became obese (41%) in adolescence, and only 38% return to a normal weight (Panel C). A total of 83% of the children with obesity at the age of 4 were overweight or obese in adolescence, and only 17% returned to a normal weight (Panel D). The sample size for each year of age is provided above the plots.

Persistenz der kindlichen Adipositas im Erwachsenenalter



Whitaker et al., N Engl J Med 1997; 337:869-873

Ursachen der Adipositas

Primär- Erkrankungen

Cushing Syndrom

- Leitsymptom: Wachstumsstillstand
  + rasche Gewichtszunahme
- Screening: 24h Urin auf freies Cortisol
- Diagnose nur durch niedrig dosierten Dexamethasonhemmtest

- Adipöse Kinder haben erhöhte Cortisolwerte insbesondere bei Insulinresistenz

(Reinehr et al. Exp Clin Endo Diab 2006)

Syndromale Erkrankungen

Mit Kleinwuchs

- Prader-Willi-Syndrom (imprinting Chromosom 15q11-q13)
  - “floppy infant”
  - Trinkschwäche als Säugling,
  - Hyperphagie ab Kleinkindesalter
  - Hodenhochstand
  - z.T. Hypogonadismus

- (Laurence Moon) Bardet-Biedl-Syndrom (auto-rez.): genet nachweisbar aber 15 Gene
  - Postaxiale Hexadaktylie
  - Retinitis pigmentosa (Makuladegeneration)
  - früh Nierenversagen (Zystenniere)
  - früh Diabetes mellitus 2

- Cohen-Syndrom (auto-rez.): 30% genet. nachweisbar
  - Schmale Hände/Füße
  - lange Finger/Zehen
  - offener Mund, vorstehende Schneidezähne
  - Muskuläre Hypotonie
  - Mikrozephalie
  - Mentale Retardierung
  - z.T. Opticusatrophie

Syndromale Erkrankungen

Ohne Kleinwuchs

- Fragiles X-Syndrom
  - Makrozephalie
  - große Hoden
  - große Ohren
  - Intelligenzminderung

- Sotos-Syndrom (2/3 Veränderungen im NSD1-Gen Chromosom 5 (5q35))
  - Makrosomie bei Geburt
  - Makrocephalie
  - Längliches Gesicht
  - Knochenalter akzeleriert
  - große Hände/Füße
  - „Geheimratsecken“
  - Balkonstirn
  - Z.T. mentale Retardierung

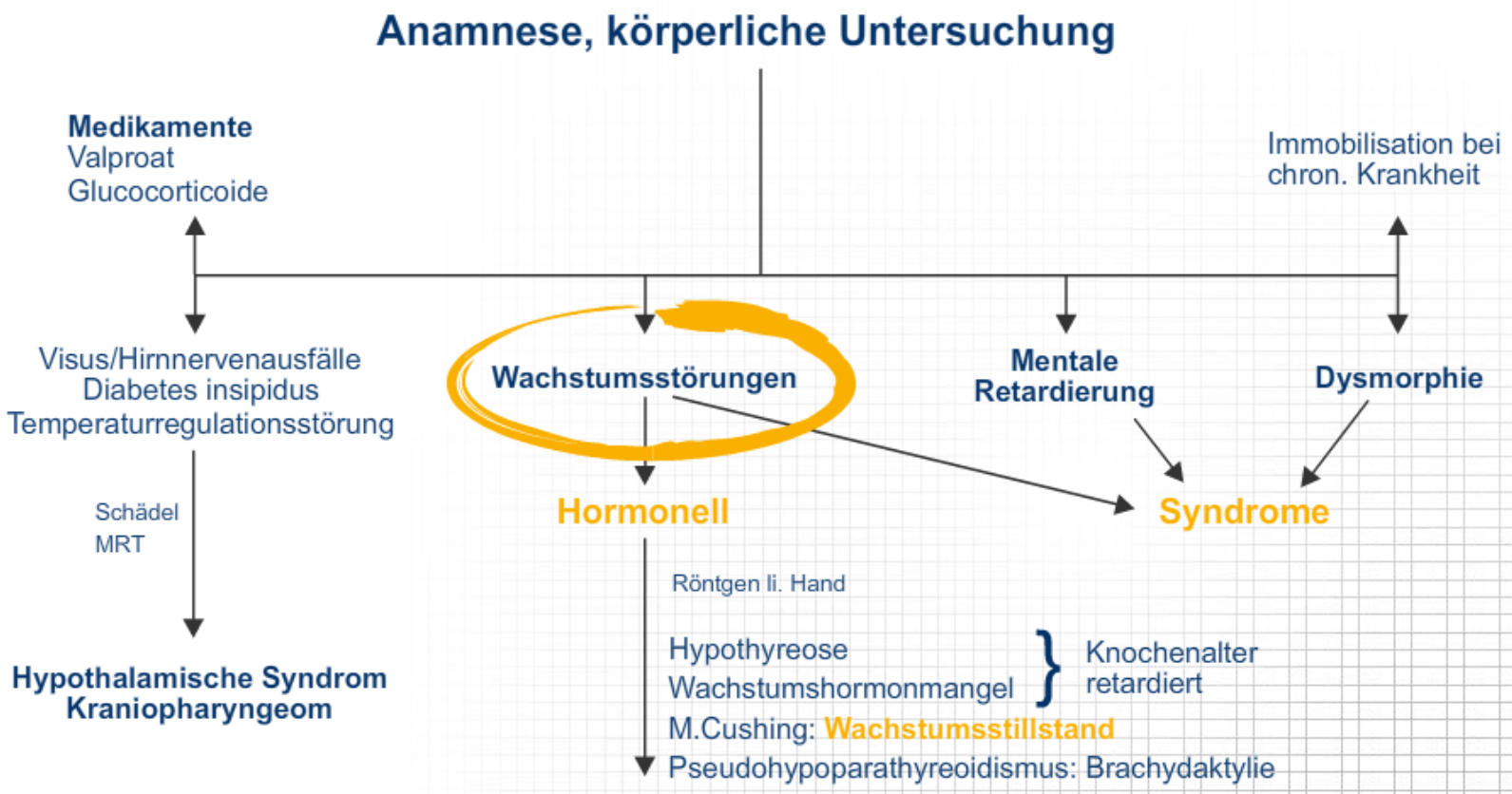
Grunderkrankung bei Adipositas

- Kollektiv: 1400 adipöse Kinder (Alter 4-16 Jahre)

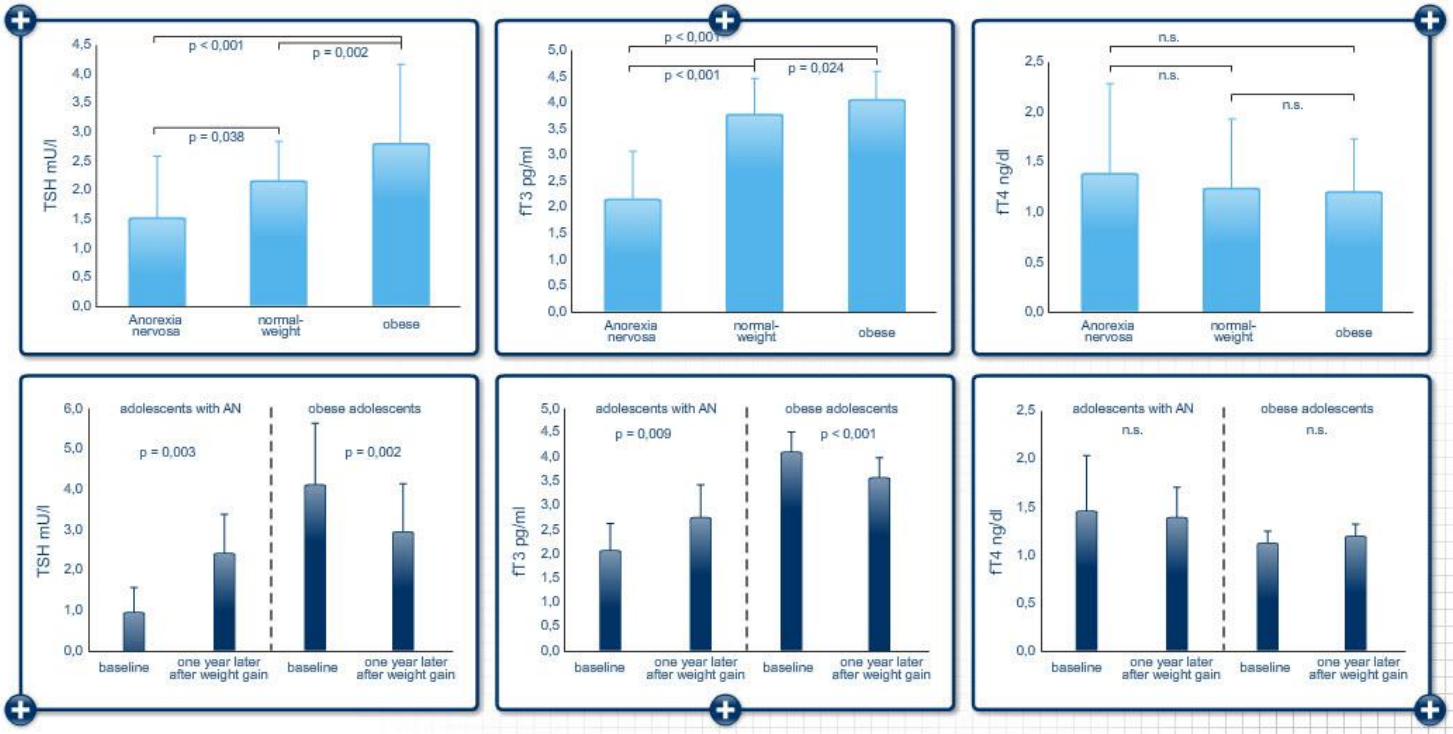
• **endokrinologisch**:	- Autoimmunhypothyreose:	3
	- Wachstumshormonmangel:	1
	- Cushing Syndrom bei NNRtumor:	1
	- Pseudohypoparathyreoidismus:	2
	- Pseudopseudohypoparathyreoidismus:	1
• **syndromal:**	- Prader- Willi- Syndrom:	2
	- Bardet- Biedl- Syndrom:	1
	- Klinefelter -Syndrom:	1
• **Gesamt:**		**12 (<1%)**

Reinehr et al Journal of Ped 2007

Diagnostik Primärerkrankung bei Adipositas im Kindesalter



Schilddrüsenhormone und Gewichtsstatus



Erhöhte TSH und T3 Werte bei Adipositas erhöhen Grundumsatz!

Reinehr et al. Arch Dis Child 2002 + JCEM 2006

Neue Leitlinie in USA

JCEM 2017
102(3): 709-757

Pediatric Obesity—Assessment, Treatment, and Prevention: An Endocrine Society Clinical Practice Guideline

Dennis M. Styne,[1] Silva A. Arslanian,[2] Ellen L. Connor,[3] Ismaa Sadaf Farooqi,[4] M. Hassan Murad,[5] Janet H. Silverstein,[6] and Jack A. Yanovski[7]

[1]University of California Davis, Sacramento, California 95817; [2]University of Pittsburgh, Pittsburgh, Pennsylvania 15224; [3]University of Wisconsin, Madison, Wisconsin 53792; [4]University of Cambridge, Cambridge CB2 0QQ, United Kingdom; [5]Mayo Clinic, Rochester, Minnesota 55905; [6]University of Florida, Gainesville, Florida 32607; and [7]National Institutes of Health, Bethesda, Maryland 20892

Cosponsoring Associations: The European Society of Endocrinology and the Pediatric Endocrine Society. This guideline was funded by the Endocrine Society.

Objective: To formulate clinical practice guidelines for the assessment, treatment, and prevention of pediatric obesity.

Participants: The participants include an Endocrine Society–appointed Task Force of 6 experts, a methodologist, and a medical writer.

Evidence: This evidence-based guideline was developed using the Grading of Recommendations, Assessment, Development, and Evaluation approach to describe the strength of recommendations and the quality of evidence. The Task Force commissioned 2 systematic reviews and used the best available evidence from other published systematic reviews and individual studies.

Consensus Process: One group meeting, several conference calls, and e-mail communications enabled consensus. Endocrine Society committees and members and co-sponsoring organizations reviewed and commented on preliminary drafts of this guideline.

Conclusion: Pediatric obesity remains an ongoing serious international health concern affecting ~17% of US children and adolescents, threatening their adult health and longevity. Pediatric obesity has its basis in genetic susceptibilities influenced by a permissive environment starting *in utero* and extending through childhood and adolescence. Endocrine etiologies for obesity are rare and usually are accompanied by attenuated growth patterns. Pediatric comorbidities are common and long-term health complications often result; screening for comorbidities of obesity should be applied in a hierarchal, logical manner for early identification before more serious complications result. Genetic screening for rare syndromes is indicated only in the presence of specific historical or physical features. The psychological toll of pediatric obesity on the individual and family necessitates screening for mental health issues and counseling as indicated. The prevention of pediatric obesity by promoting healthful diet, activity, and environment should be a primary goal, as achieving effective, long-lasting results with lifestyle modification once obesity occurs is difficult. Although some behavioral and pharmacotherapy studies report modest success, additional research into accessible and effective methods for preventing and treating pediatric obesity is needed. The use of weight loss medications during childhood and adolescence should be restricted to clinical trials. Increasing evidence

ISSN Print 0021-972X ISSN Online 1945-7197
Printed in USA
Copyright © 2017 by the Endocrine Society
Received 30 June 2016. Accepted 10 November 2016.
First Published Online 31 January 2017

Abbreviations: ALT, alanine aminotransferase; BMI, body mass index; CDC, Centers for Disease Control and Prevention; CVD, cardiovascular disease; FDA, Food and Drug Administration; GH, growth hormone; HbA1c, hemoglobin A1c; LAGB, laparoscopic adjustable gastric banding; NAFLD, nonalcoholic fatty liver disease; PCOS, polycystic ovary syndrome; QOL, quality of life; RCT, randomized controlled trial; RYGB, Roux-en-Y gastric bypass; T2DM, type 2 diabetes mellitus; VSG, vertical sleeve gastrectomy; WHO, World Health Organization.

Summary of Recommendations

1.0 Diagnosing overweight and obesity

1.1 We recommend using body mass index (BMI) and the Centers for Disease Control and Prevention (CDC) normative BMI percentiles to diagnose overweight or obesity in children and adolescents ≥2 years of age. (1|⊕⊕⊕○)

1.2 We recommend diagnosing a child or adolescent >2 years of age as overweight if the BMI is ≥85th percentile but <95th percentile for age and sex, as obese if the BMI is ≥95th percentile, and as extremely obese if the BMI is ≥120% of the 95th percentile or ≥35 kg/m^2 (1|⊕⊕○○). We suggest that clinicians take into account that variations in BMI correlate differently to comorbidities according to race/ethnicity and that increased muscle mass increases BMI. (2|⊕○○○)

1.3 We suggest calculating, plotting, and reviewing a child's or adolescent's BMI percentile at least annually during well-child and/or sick-child visits. (Ungraded Good Practice Statement)

1.4 We suggest that a child <2 years of age be diagnosed as obese if the sex-specific weight for recumbent length is ≥97.7th percentile on the World Health Organization (WHO) charts, as US and international pediatric groups accept this method as valid. (2|⊕○○○)

1.5 We recommend against routine laboratory evaluations for endocrine etiologies of pediatric obesity unless the patient's stature and/or height velocity are attenuated (assessed in relationship to genetic/familial potential and pubertal stage). (1|⊕⊕⊕○)

1.6 We recommend that children or adolescents with a BMI of ≥85th percentile be evaluated for potential comorbidities (see Table 2 and Fig. 1). (1|⊕⊕⊕○)

Keine Blutentnahme für Diagnostik Primär-erkrankung bei normaler Wachstumsgeschwindig-keit notwendig

Ungleich BRD: TSH Bestimmung empfohlen

-> In USA keine Verwirrung durch erhöhte TSH Werte

Verzeichnis von Leitsymptomen

Table 3. Genetic Obesity Syndromes With and Without Developmental Delay

Genetic Obesity Syndrome	Clinical Features
Obesity with developmental delay	
Dominant	
Prader-Willi syndrome	Hypotonia, failure to thrive in infancy followed by weight gain, short stature (due to GH deficiency), hyperphagia, hypogonadotropic hypogonadism, sleep disturbance, obsessive behaviors
Albright hereditary osteodystrophy	Short stature in some but not all patients, skeletal defects, impaired olfaction, and hormone resistance (*e.g.*, parathyroid hormone) if a mutation is maternally inherited
SIM1 deficiency	Hyperphagia with autonomic dysfunction (characterized by low systolic blood pressure), speech and language delay, neurobehavioral abnormalities, including autistic type behaviors
BDNF/TrkB deficiency	Hyperactivity, impaired concentration, limited attention span, impaired short-term memory and pain sensation
Recessive	
Bardet-Biedl syndrome	Dysmorphic extremities (syndactyly/brachydactyly/polydactyly), retinal dystrophy or pigmentary retinopathy, hypogonadism, renal abnormalities/impairment
TUB deficiency	Retinal dystrophy, deafness
Obesity without developmental delay	
Dominant	
Alström syndrome	Retinal dystrophy; extreme insulin resistance; deafness; dilated cardiomyopathy; progressive pulmonary, hepatic, and renal dysfunction
MC4R deficiency	Hyperphagia, accelerated linear growth, disproportionate hyperinsulinemia, low/normal blood pressure
SH2B1 deficiency	Hyperphagia, disproportionate hyperinsulinemia, early speech and language delay that often resolves, behavioral problems including aggression
KSR2 deficiency	Mild hyperphagia and reduced basal metabolic rate, insulin resistance often with acanthosis nigricans, irregular menses, early development of T2DM
Recessive	
Leptin deficiency	Extreme hyperphagia, frequent infections, hypogonadotropic hypogonadism, mild hypothyroidism
Leptin receptor deficiency	Extreme hyperphagia, frequent infections, hypogonadotropic hypogonadism, mild hypothyroidism
POMC deficiency	Hyperphagia, cholestatic jaundice or adrenal crisis due to ACTH deficiency, pale skin, and red hair in whites
PCSK1 deficiency	Small bowel enteropathy, hypoglycemia, hypothyroidism, ACTH deficiency, diabetes insipidus

MC4R Mutation

- **innerhalb von Familien haben Mutationsträger ein um durchschnittlich <u>15 - 30 kg</u> höheres Körpergewicht als Wildtypen**
- Häufigkeit: 3 - 5 % bei adipösen Kindern
- Autosomal-dominant vererbt

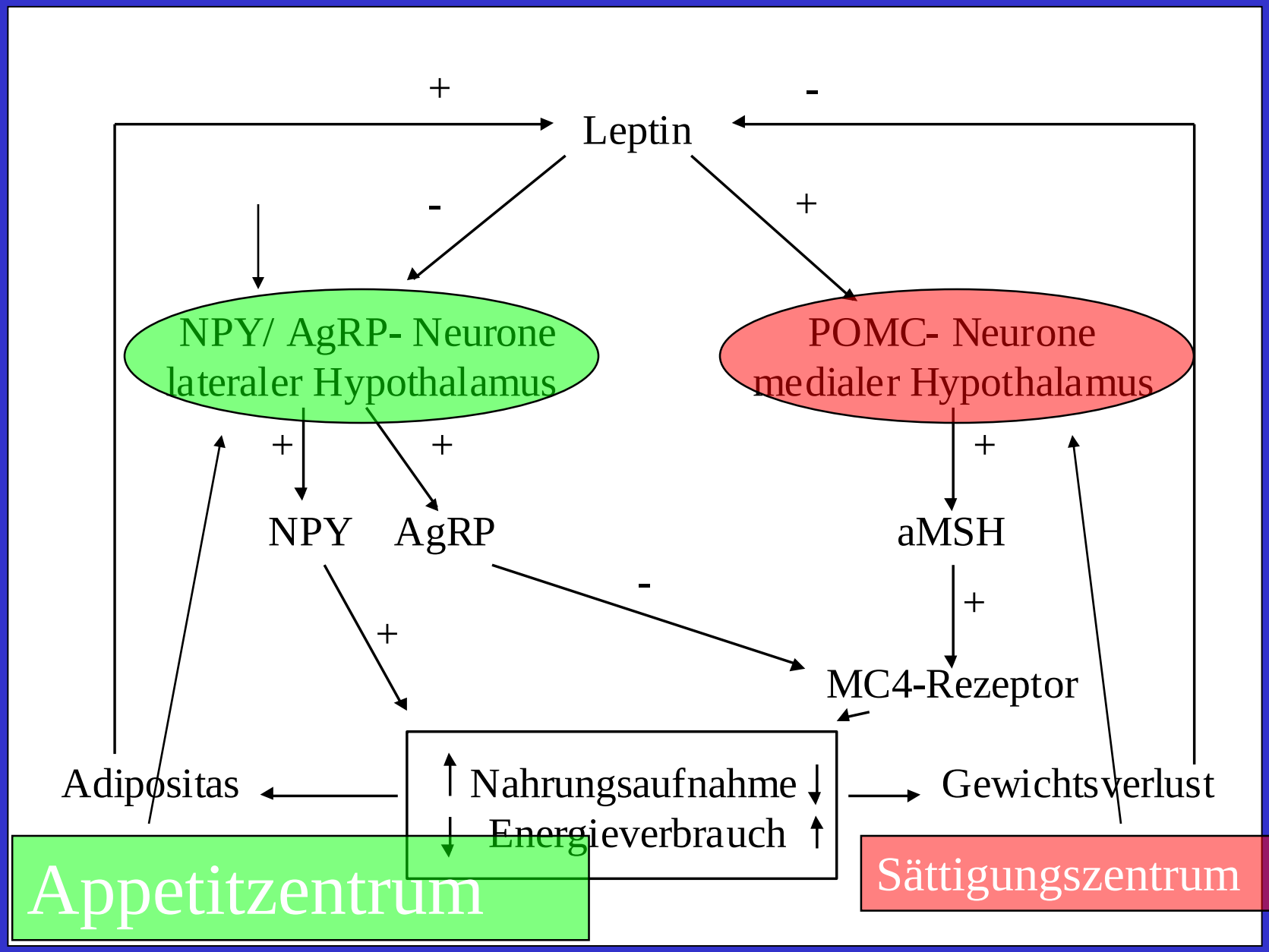
Dempfle et al., J Med Genet. 2004 Oct;41(10):795-800

Variants with a polygenic effect on the human body weight (Speliotes et al Nat Genetics 2010; n=250.000)

Chromosome nearest gene	SNP	approx. frequency of the risk allele	effect on BMI
INSIG2	rs7566605	37% (C)	+ 1.0 kg/m² for CC genotype
FTO	rs9939609	40 % (A)	+ 0.40 kg/m² per A allele
MC4R	rs571312	24 % (A)	+ 0.23 kg/m² per A allele
TMEM 18	rs11127485	83% (T)	+0.37 kg/m² per T allele
GNPDA2	rs10938397	43% (G)	+ 0.18 kg/m² per G allele
BDNF	rs10767664	78% (A)	+ 0.19 kg/m² per A allele
NEGR1	rs2815752	61% (A)	+ 0.13 kg/m² per A allele
SH2B1	rs7359397	40% (T)	+ 0.15 kg/m² per T allele
ETV5	rs9816226	82% (T)	+ 0.14 kg/m² per T allele
MTCH2	rs[illegible]	[illegible]	[illegible]
KCTD15	rs29941	67% (G)	+ 0.06 kg/m² per G allele
[illegible]	[illegible]	19% (G)	+ 0.22 kg/m² per G allele
TFAP2B	rs987237	18% (G)	+ 0.13 kg/m² per G allele
FAIM2	rs7138803	38% (A)	+ 0.12 kg/m² per T allele
NRXN3	rs710150332	21% (C)	+ 0.13 kg/m² per T allele
RBJ	rs713586	47% (C)	+ 0.14 kg/m² per C allele
GPRC5B	rs12444979	87% (C)	+ 0.17 kg/m² per C allele
MAP2K5	rs2241423	78% (G)	+ 0.13 kg/m² per G allele
QPCTL	rs2287017	80% (C)	+ 0.15 kg/m² per C allele

Diese Polymorphismen (n=34) erklären <5% der BMI Varianz

Regelkreis Hypothalamus



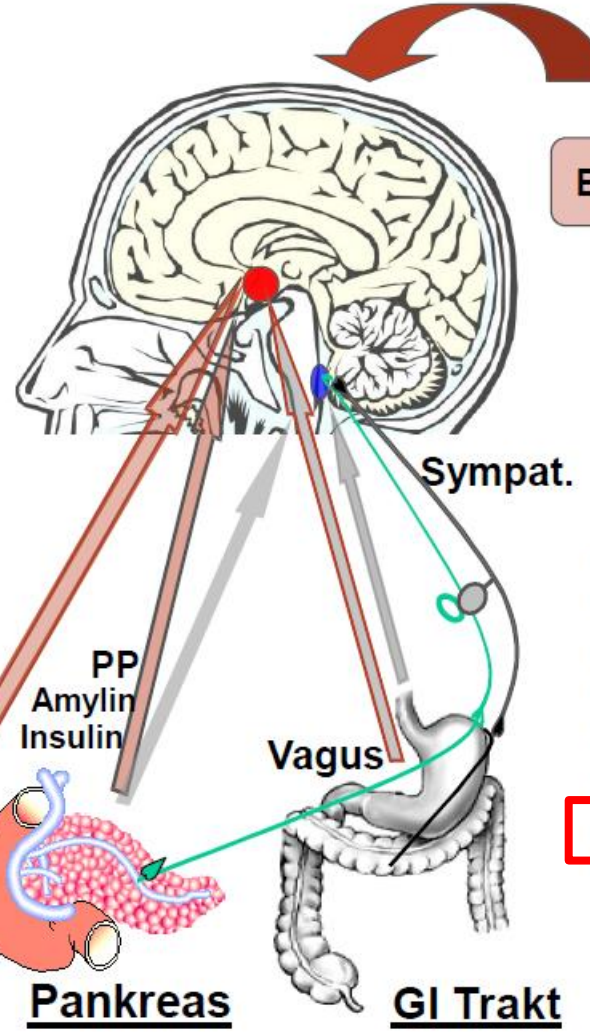
Zentrale und periphere Regulatoren der Energiehomöostase

Roth & Reinehr, Arch Pediatr Adolesc Med. 2010

Höhere kortikale Regionen
-Orbitofrontaler Kortex
Mittelhirn/limbisches System
-Nucleus accumbens
-Corpus striatum
-Globus pallidum
-Ventrotegmentale Region

Emotion (Stress)

Externe Nahrungsreize



Langzeitsignale:
- *Leptin*
- *Insulin*
- *Adiponektin*

Kurzzeitsignale:
- *Pankreat. Polypeptid (PP)*
- *Peptide-YY (PYY)*
- *Ghrelin*
- *Cholecystokinin (CCK)*
- *Amylin*
- *Glucagon-like peptide*
- *Glucagon*

Indikation für genetische Untersuchung: BMI > 25kg/m² mit 2 Lebensjahren

Wabitsch et al Int J Obes. 2019

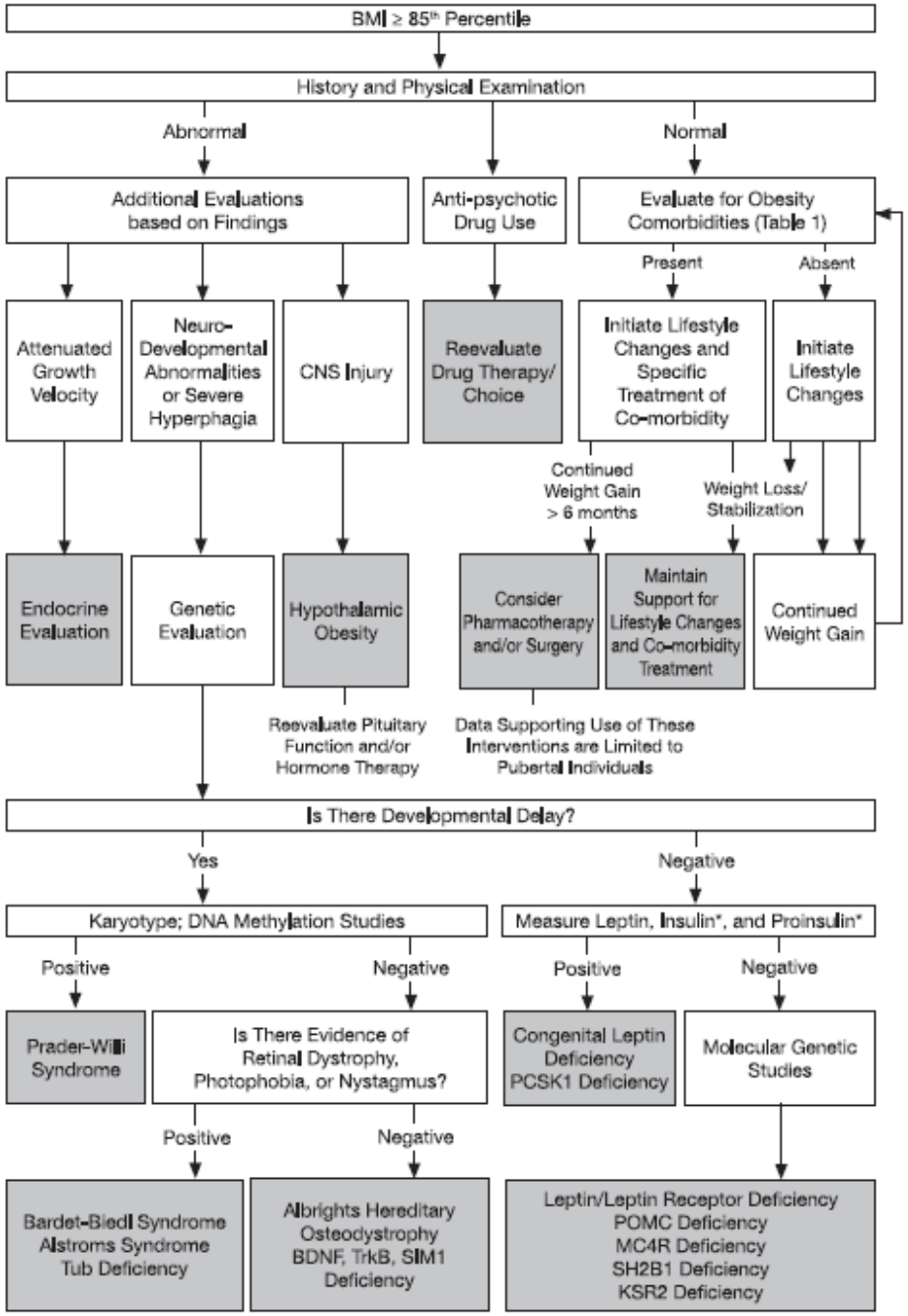
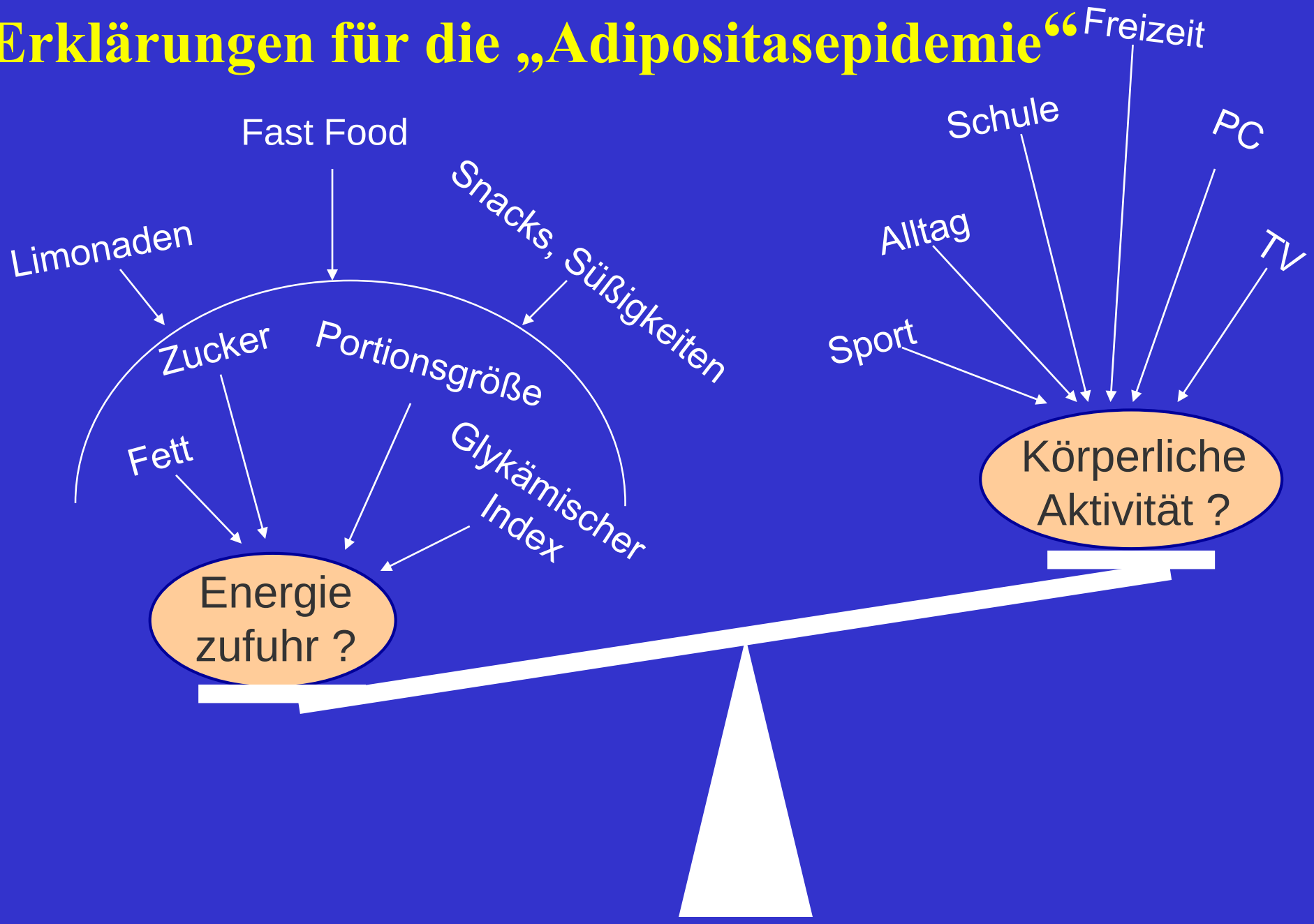


Figure 1. Diagnosis and management flowchart. *Measure insulin and proinsulin in patients with clinical features of PCSK1 deficiency. [Adapted from August GP *et al.* (86) with permission, © Endocrine Society.] [Republished with permission of Springer Science and Bus Media BV from Farooqi S and O'Rahilly S (87); permission conveyed through Copyright Clearance Center, Inc.]

Erklärungen für die „Adipositasepidemie“



Zusammenhang Fernsehen und Adipositas bei Kindern

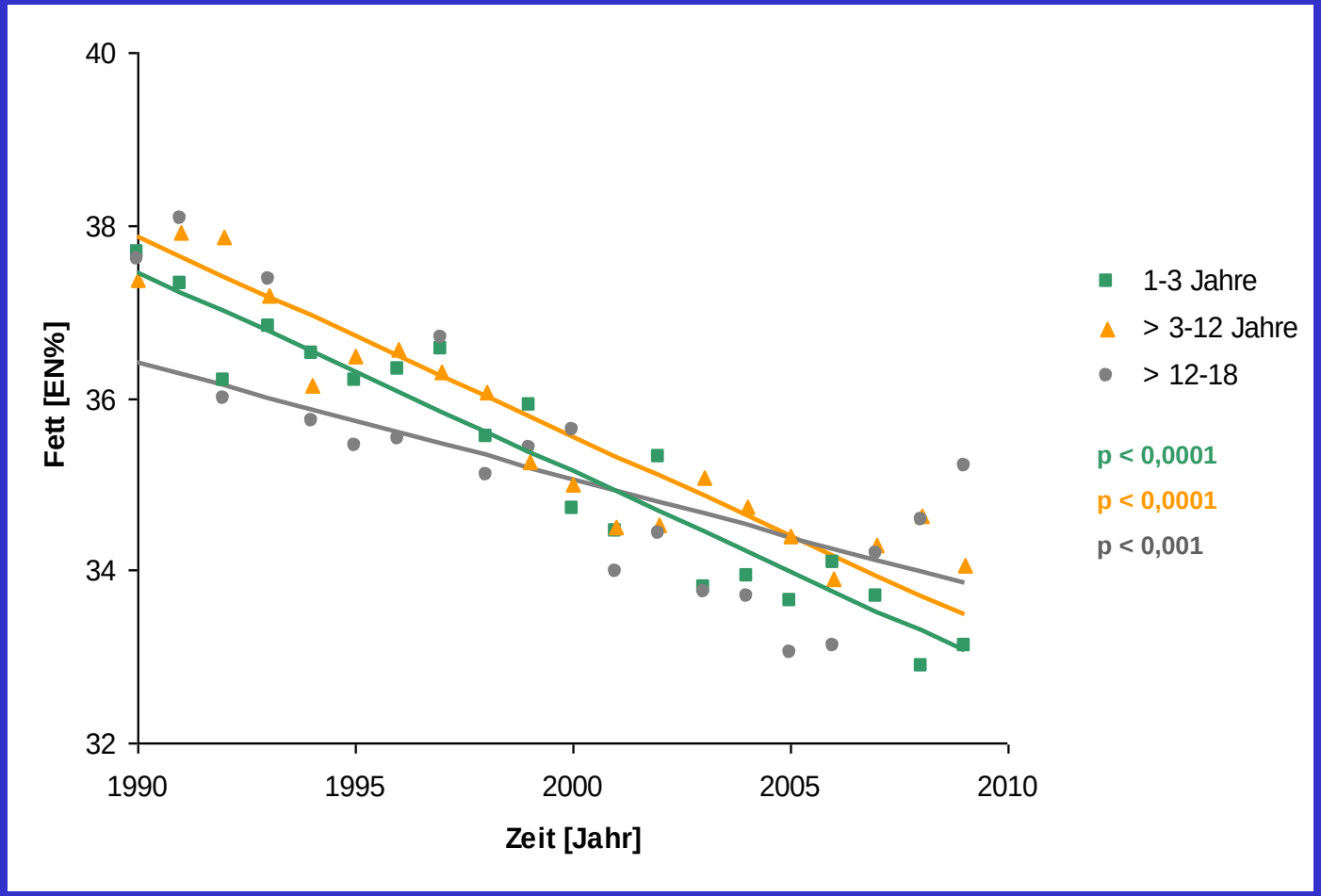
- Längsschnittstudie 2037 Kinder:
  - Kinder < 7 Jahre: jede Stunde Fernsehen/Tag -> BMI um 11% höher

    Zimmermann et al Am J Publ Health 2010

- Kinder im Vorschulalter
  - Kinder wählen Produkte nach Werbespot bevorzugt aus
  - Im Vorschulalter noch kein kritischer Verstand: Werbung schutzlos ausgeliefert

    Dixon Soc Sci Med 2007

20-Jahres-Trends Fettverzehr bei Kindern in Deutschland



Self-Reported Energy Intake by Age in Overweight and Healthy-Weight Children in NHANES, 2001–2008

WHAT'S KNOWN ON THIS SUBJECT: The relationship between energy intake and obesity in children has yielded inconsistent results. Efforts to improve dietary intake as a means of improving weight status have largely yielded disappointing results.

WHAT THIS STUDY ADDS: Self-reported energy intake for younger, but not older, overweight/obese children is higher than healthy-weight peers. In early childhood, higher (or excessive) energy intake may lead to onset of obesity, but other mechanisms may be important to maintain obesity through adolescence.

AUTHORS: Asheley Cockrell Skinner, PhD, Michael J. Steiner, MD, and Eliana M. Perrin, MD, MPH

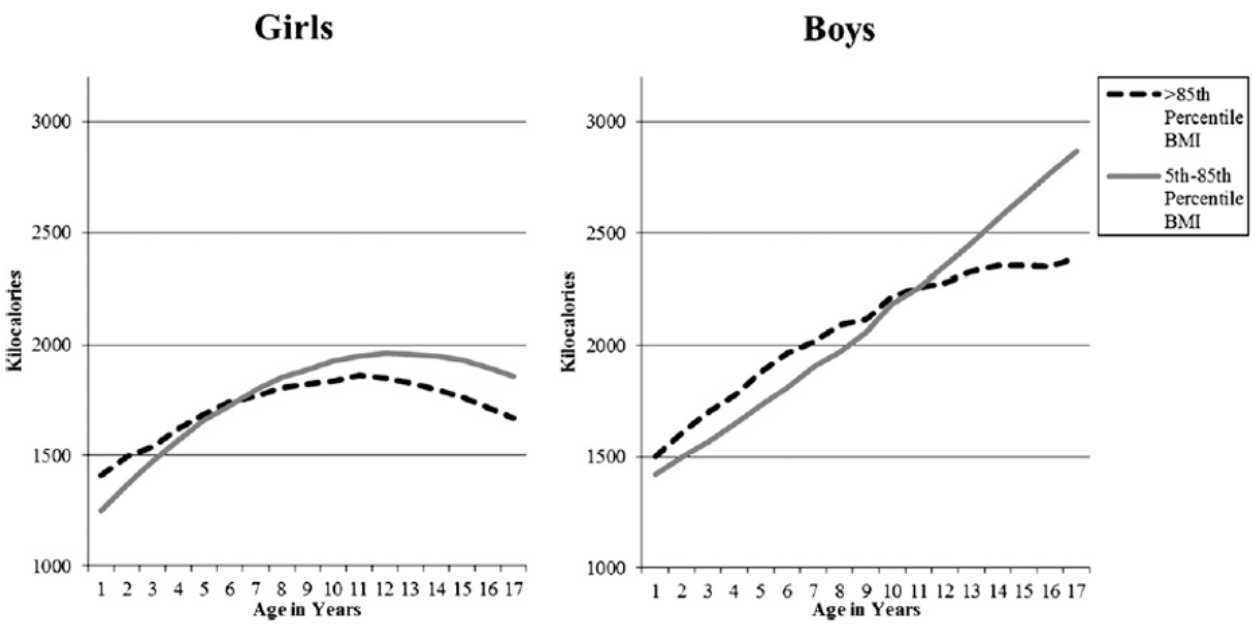
Division of General Pediatrics and Adolescent Medicine, Department of Pediatrics in the School of Medicine, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina

KEY WORDS

Obesity, energy intake, population-based survey, nutrition

ABBREVIATIONS

AMPM—automated multiple pass method
FPL—federal poverty level
NHANES—National Health and Nutrition Examination Survey



uted to the conception and design, acquisition
sis and interpretation of data; drafted the
it critically for important intellectual content;
roval of the version to be published. Dr
d to the conception and design, and
ata; drafted the article and revised it critically
lectual content; and gave final approval of the
shed. Dr Perrin contributed to the conception
e interpretation of data; drafted the article
cally for important intellectual content; and
l of the version to be published.

g/cgi/doi/10.1542/peds.2012-0605

012-0605

cation Jun 4, 2012

dence to Asheley Cockrell Skinner, PhD, CB

Cave

- Adipöse Kinder verzehren im Vergleich zu Normalgewichtigen Kinder weniger oder gleich viel !

Mögliche Erklärungen für diese Studienergebnisse:
zu wenig Bewegung für verzehrte Menge?
Underreporting?

Gutin Int J Obes 2007; Phillips Obes Res 2004

Einfluss Ernährung auf Körpergewicht

- Prospektive Studien
  - Verzehr **fast food** mit Adipositas assoziiert
    - N=3031 junge Erwachsene (18-30 J)
    - Follow up 15 Jahren

      [Pereira et al Lancet 2005;365:36-42]
  - Verzehr **gesüßte Getränke** führt zu Übergewicht
    - N=548 Schulkinder (Alter 11,7 J)
    - Follow up 19 Monate
    - Pro gesüßter Drink/ Tag steigt BMI um 0.18 kg/m²

      [Ludwig et al Lancet 2001;357:505-508]

	Anzahl	richtige Antworten im Durchschnitt	Streubreite richtige Antworten (Standardabweichung)
Nicht adipöse Kinder	95	12,2 (55%)	5-20 (3,1)
Adipöse Kinder	95	12,8 (58%)	2-19 (2,8)

- Ernährungswissen adipöser Kinder entspricht nicht adipösen Kindern (p=0,143)
  - Mangelndes Ernährungswissen nicht Ursache der Adipositas im Kindesalter

Reinehr et al Nutrition Research 2003

Lower protein in infant formula is associated with lower weight up to age 2 y: a randomized clinical trial[1–4]

Berthold Koletzko, Rüdiger von Kries, Ricardo Closa, Joaquín Escribano, Silvia Scaglioni, Marcello Giovannini, Jeannette Beyer, Hans Demmelmair, Dariusz Gruszfeld, Anna Dobrzanska, Anne Sengier, Jean-Paul Langhendries, Marie-Francoise Rolland Cachera, and Veit Grote for the European Childhood Obesity Trial Study Group

The American Journal of Clinical Nutrition

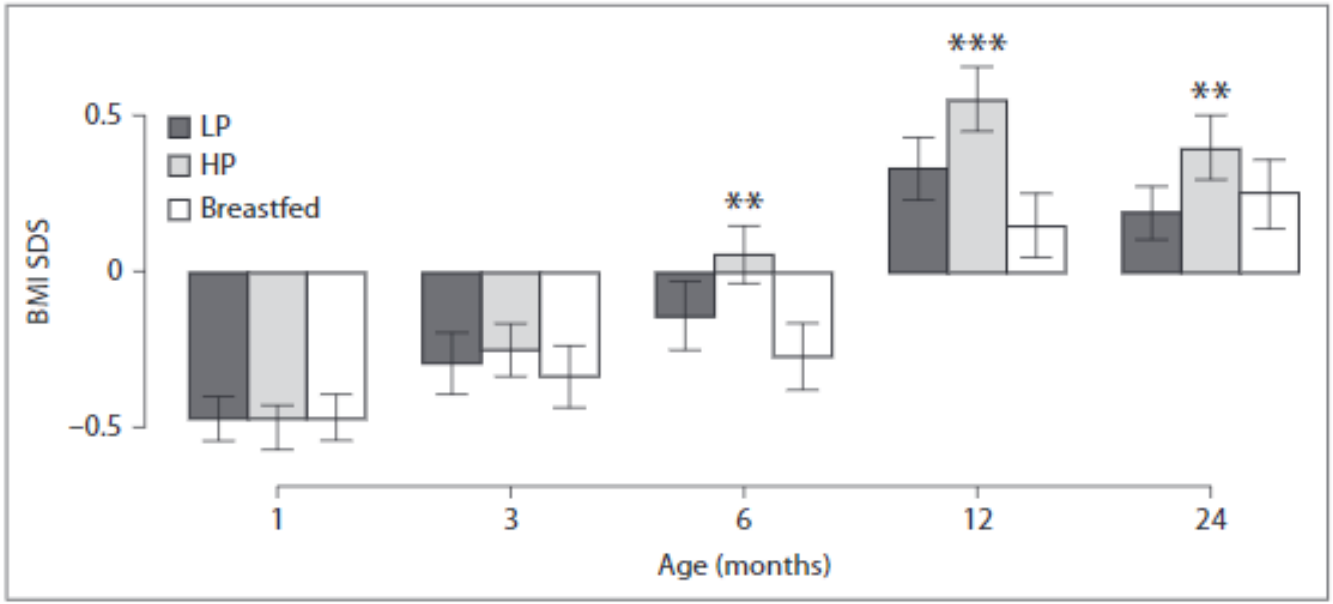


Fig. 4. BMI SDS from birth to age 2 years in subjects participating in the European Childhood Obesity Project fed breast milk, or randomized to receive for the first year of life formulas with LP or HP contents. Formula-fed infants in the HP group showed higher BMI values than breastfed infants in infancy and at 2 years of age. The group randomized to LP had significantly lower BMI levels than the HP group, and LP normalized BMI levels at age 2 years as compared to breastfed subjects. Drawn from data of Koletzko et al. [24].

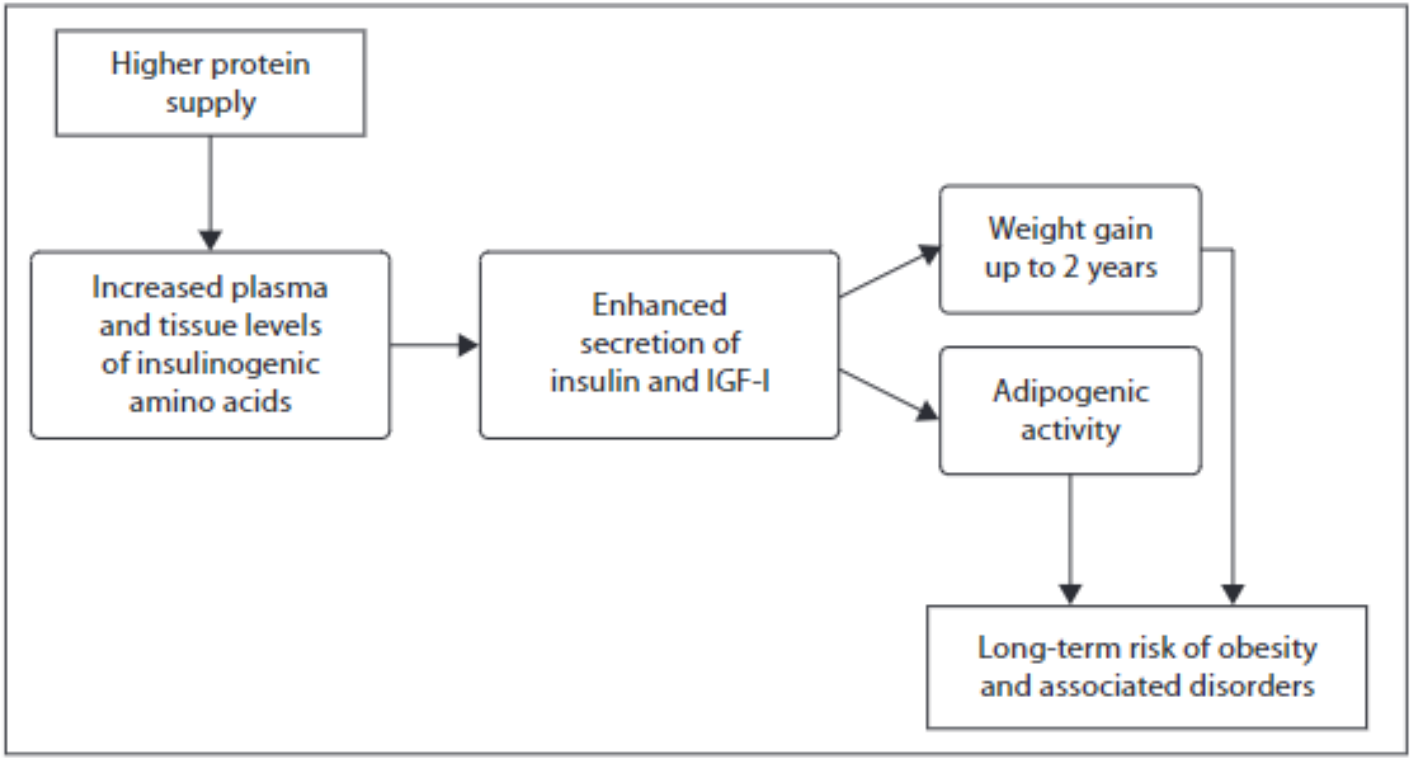


Fig. 3. The Early Protein Hypothesis suggests that a dietary protein supply to infants in excess of their metabolic requirements will lead to increased plasma and tissue concentrations of insulin-releasing amino acids and an enhanced secretion of insulin and IGF-I, which in turn will enhance early weight gain, adipogenic activity and long-term obesity risk. Redrawn after Koletzko et al. [23].

REVIEW ARTICLE

Obesity, Diabetes, and Gut Microbiota

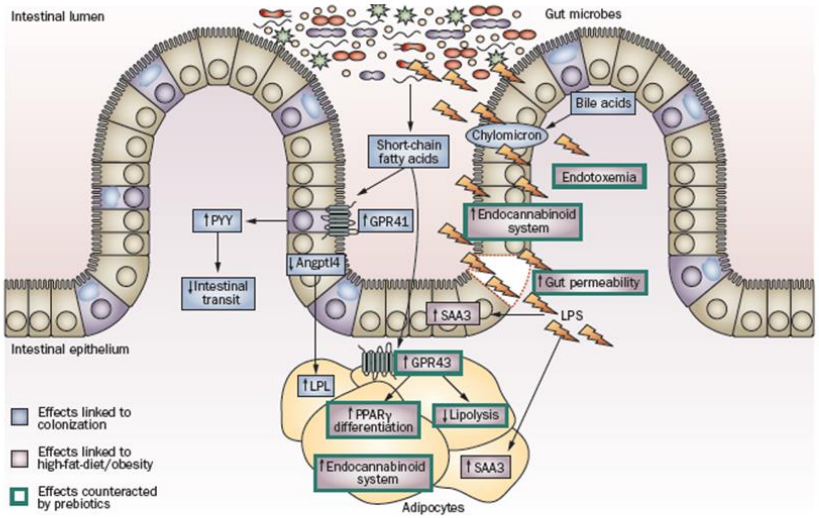
The hygiene hypothesis expanded?

Darmflora und Adipositas

- Backhed et al 2004 . Natl Acad Sci U S A 2004;101:15718–15723
  - Keimfreie Mäuse haben 40% weniger Körperfett, selbst wenn sie 29% mehr Energie zuführen im Vergleich zu Kontrollmäusen
  - 2 Wochen Kolonisation mit Keimen führt zu 57% Anstieg Körperfett, 2,3 fachen Anstieg Triglyceride und Insulinresistenz
- Turnbaugh PJ, et al . Nature 2009;457: 480–484
  - Adipositas mit reduzierter Bakterienvielfalt Darm assoziiert (weniger Bacteroidetes, mehr Actinobacteria) verglichen zu schlanken Zwillingen

Darmflora und Adipositas -Mechanismen-

- Endotoxine induzieren chron. geringgradige Entzündung (bekannt bei Adipositas, assoziiert zur Komorbidität)
- Mikroben beeinflussen freie Fettsäuren (assoziiert zur Insulinresistenz)
- Darmbakterien beeinflussen GI- Hormone
  - Darmbakterien sezernieren u.a. Hydrolasen, welche u.a. auch Polypeptide wie PYY oder GLP-1 abbauen



- Wir schlafen zu wenig oder zur falschen Zeit…

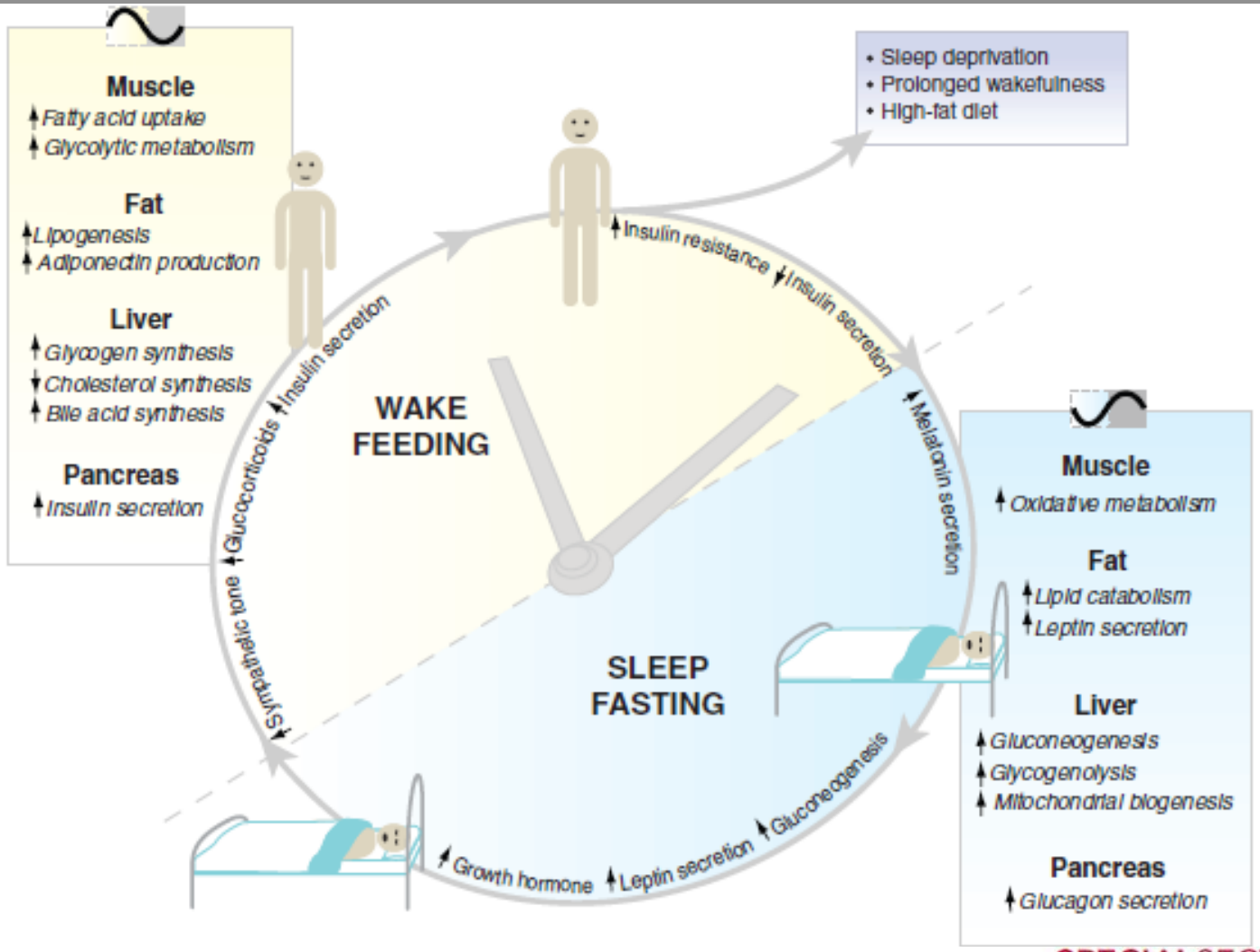
PNAS

Light at night increases body mass by shifting the time of food intake

Laura K. Fonken[a,1], Joanna L. Workman[b], James C. Walton[a], Zachary M. Weil[a], John S. Morris[b], Abraham Haim[c], and Randy J. Nelson[a,b]

Departments of [a]Neuroscience and [b]Psychology, Ohio State University, Columbus, OH 43210; and [c]Israeli Center for Interdisciplinary Research in Chronobiology, University of Haifa, Haifa 31905, Israel

Edited* by David L. Denlinger, Ohio State University, Columbus, OH, and approved September 3, 2010 (received for review June 24, 2010)



Downloaded from www.sciencemag.org on March 27, 2012

SPECIALSECTION

Science 2010

REVIEW

Circadian Integration of Metabolism and Energetics

Joseph Bass[1,2,3,4]* and Joseph S. Takahashi[5,6]

of posttranslational signaling as a mechanism controlling of circadian cycles (*4*), and recent work has shown that the SCN neuronal coupling network itself has intrinsic oscillatory function that can emerge in the absence of cell-autonomous oscillators (*5*). RNA interference screening of mammalian cells also indicates coupling of the peripheral clock to phosphatidylinositol 3-kinase signaling (*6*). Further research is warranted to elucidate the impact of posttranslational signal-

Review

Downloaded from adc.bmj.com on October 31, 2012 - Published by group.bmj.com

ADC Online First, published on October 29, 2012 as 10.1136/archdischild-2012-302263

Original article

OPEN ACCESS

Systematic review and meta-analyses of risk factors for childhood overweight identifiable during infancy

Stephen Franklin Weng,[1] Sarah A Redsell,[2] Judy A Swift,[3] Min Yang,[4] Cristine P Glazebrook[5]

▶ Additional appendices are published online only. To view these files please visit the journal online (http://dx.doi.org/10.1136/archdischild-2012-302263).

[1]Division of Epidemiology and Public Health, School of Community Health Sciences, University of Nottingham, Nottingham, UK
[2]School of Nursing, Midwifery and Physiotherapy, University of Nottingham, Queen's Medical Centre, Nottingham, UK
[3]Division of Nutritional

ABSTRACT

Objective To determine risk factors for childhood overweight that can be identified during the first year of life to facilitate early identification and targeted intervention.

Design Systematic review and meta-analysis.

Search strategy Electronic database search of MEDLINE, EMBASE, PubMed and CAB Abstracts.

Eligibility criteria Prospective observational studies following up children from birth for at least 2 years.

Results Thirty prospective studies were identified. Significant and strong independent associations with childhood overweight were identified for maternal pre-

What is already known on this topic

- There is evidence that overweight or obesity during childhood increases the risk of adult obesity.
- Previous reviews have identified rapid weight gain, high birth weight and maternal smoking in pregnancy as important risk factors for childhood obesity.

Risikofaktoren für Adipositas mit 6 Jahren

- Rasche Gewichtszunahme erste Lebensjahr
  - 3,9x faches Risiko
- Hohes Geburtsgewicht
  - 2,3x fach erhöhtes Risiko ab >4000g
- Rauchen Schwangerschaft
  - 1,5 faches Risiko
- Mutter übergewichtig vor Schwangerschaft
  - 1,4 fach erhöhtes Risiko

- Stillen protektiv (**evtl.)**
  - 15% Reduktion
- Späte Einführung feste Kost (evtl.)
  - Früh (< 4 Lm): 1,1 faches Risiko

Die psychosoziale Perspektive

- Warum essen wir?
  - Hunger
  - weil es angeboten wird
  - Langeweile
  - Stress
  - Frust

} Rolle der Familie

- denn Essen löst positive Gefühle aus