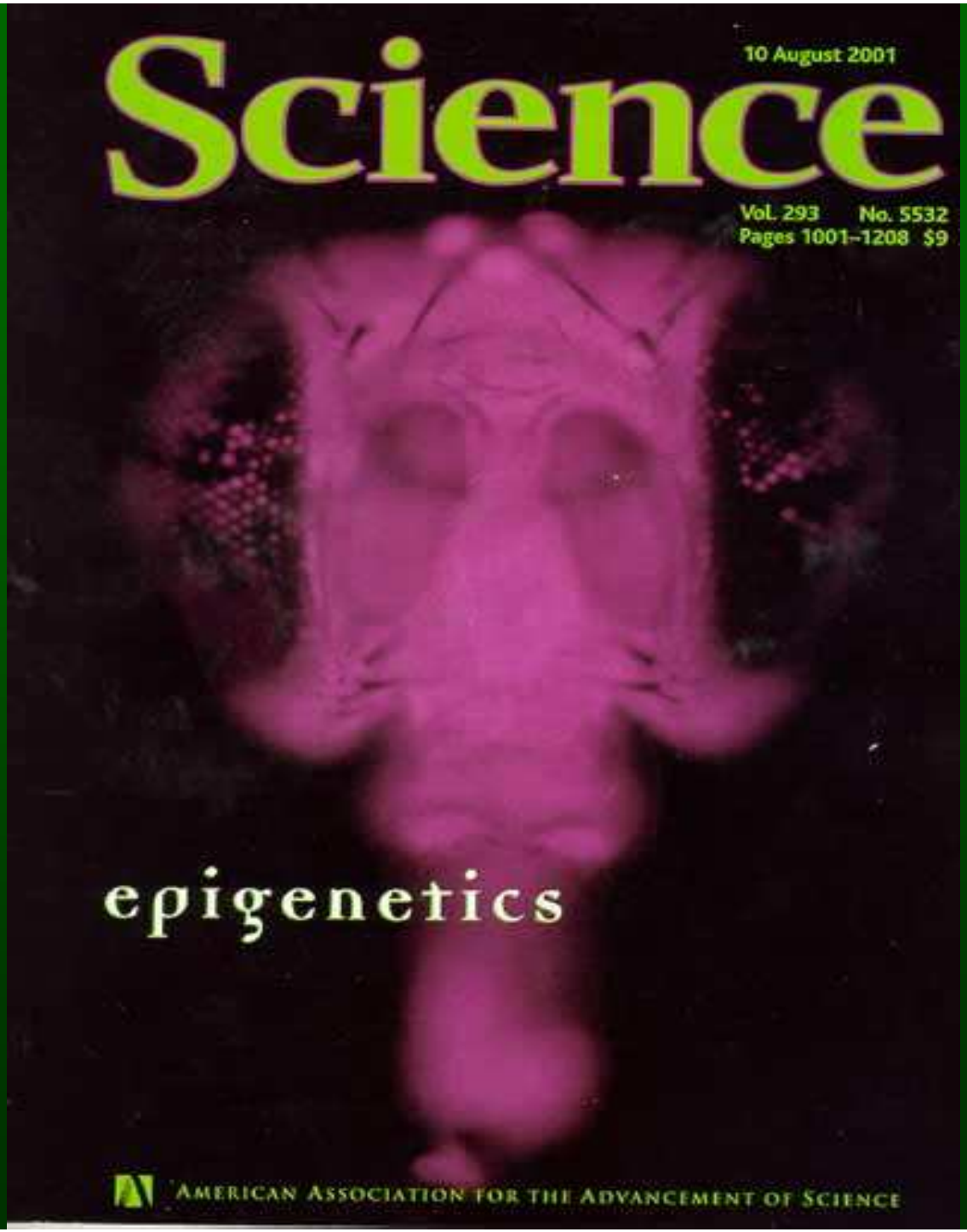
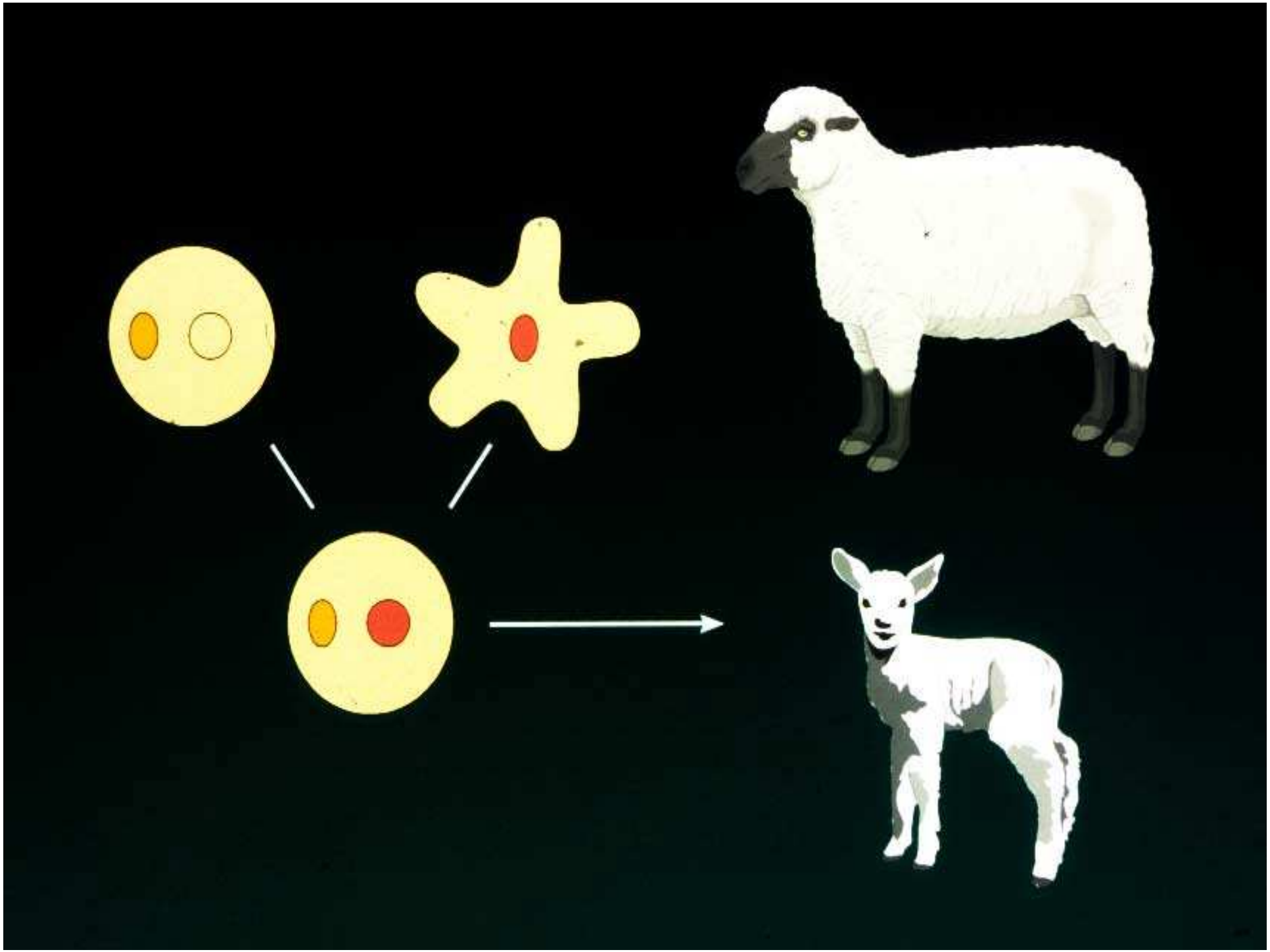

Science
10 August 2001
Vol. 293 No. 5532
Pages 1001–1208 $9
epigenetics
American Association for the Advancement of Science


AMERICAN ASSOCIATION FOR THE ADVANCEMENT OF SCIENCE
SCIENCE
19 DECEMBER 1997
VOL. 278 • PAGES 2021–2192
$7.00
Breakthrough of the Year
CLONING



NEWS & VIEWS

ENTOMOLOGY

Royal aspirations

GENE E. ROBINSON

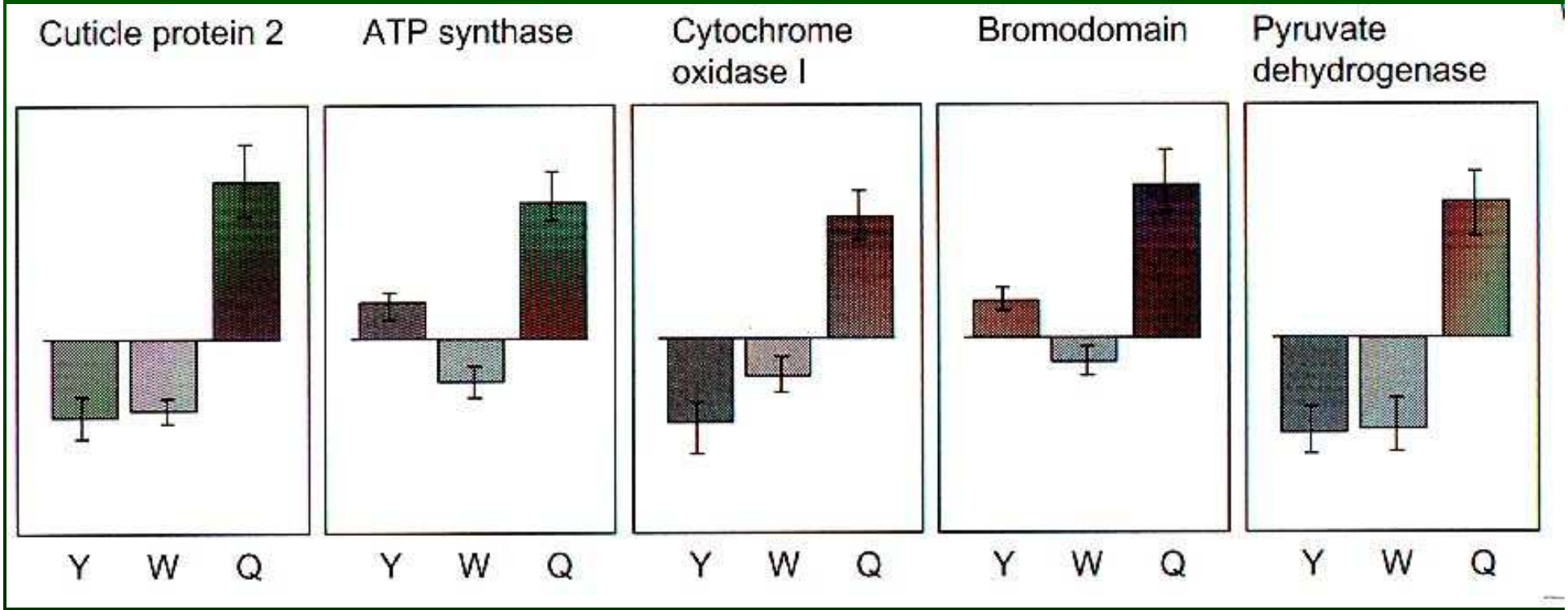
ARTICLE

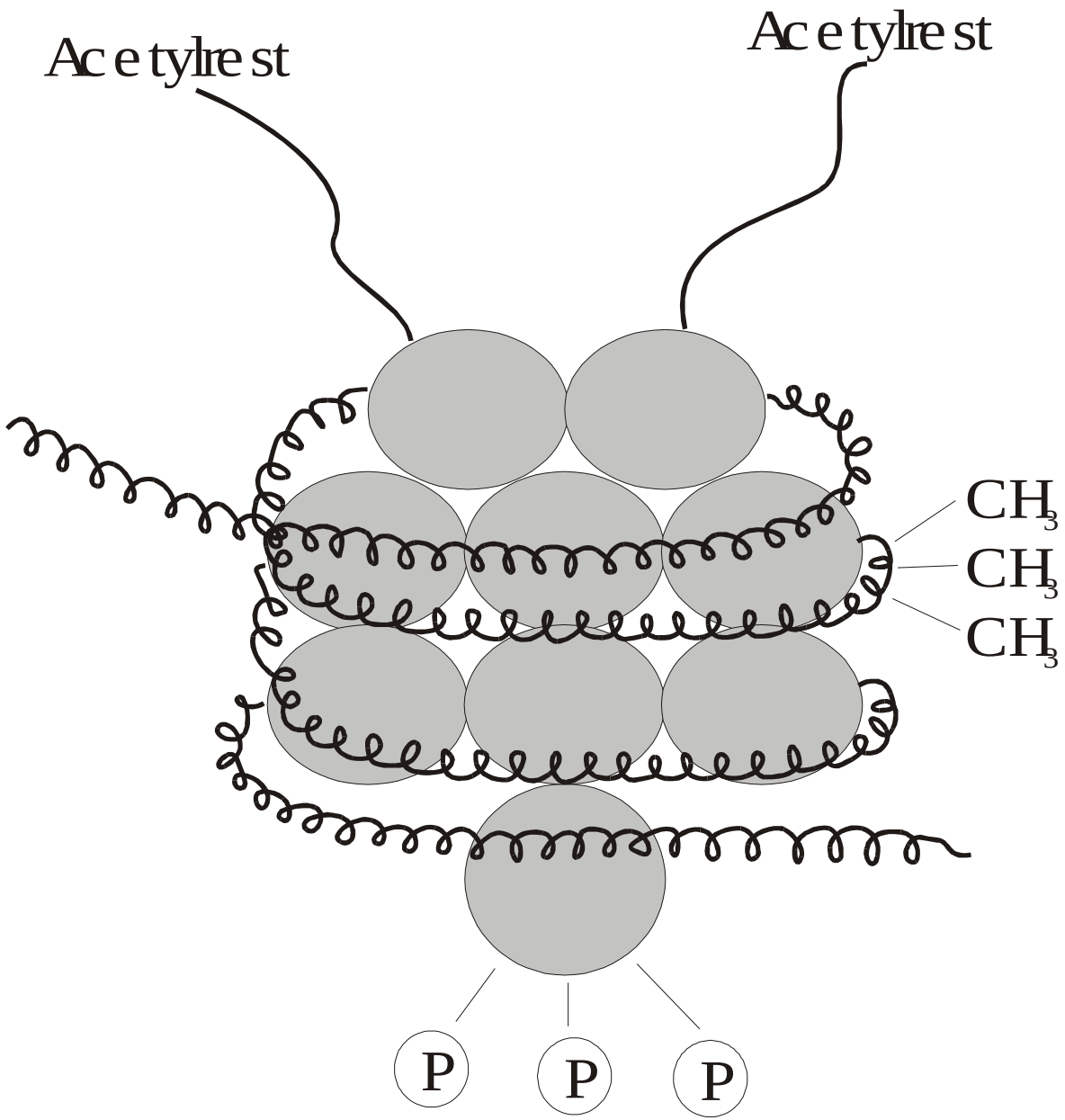
doi:10.1038/nature10093

Royalactin induces queen differentiation in honeybees

Masaki Kamakura[1]

Königin Drohne Arbeiterin




Acetylrest
Acetylrest
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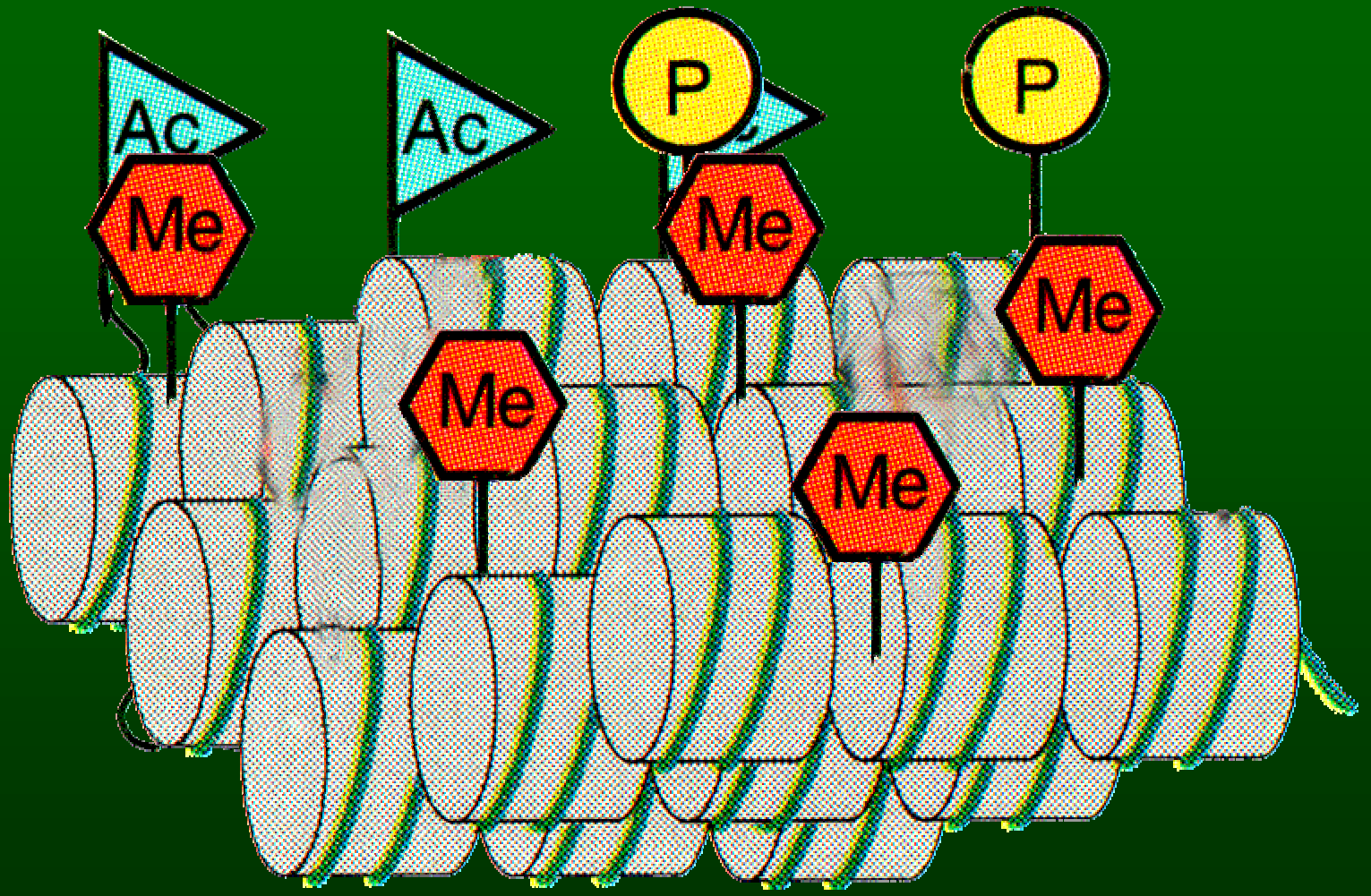
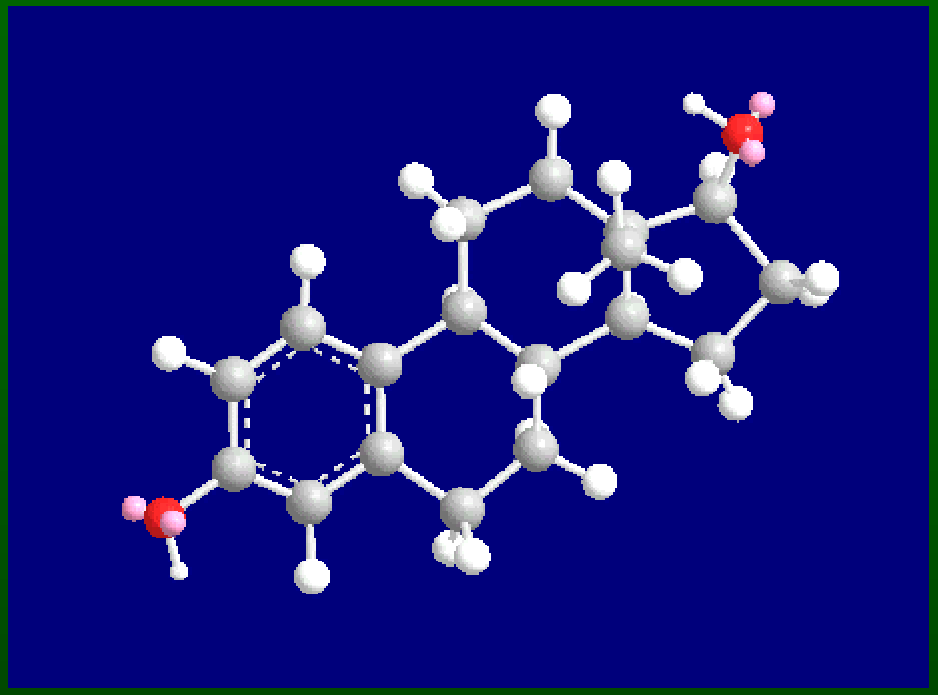
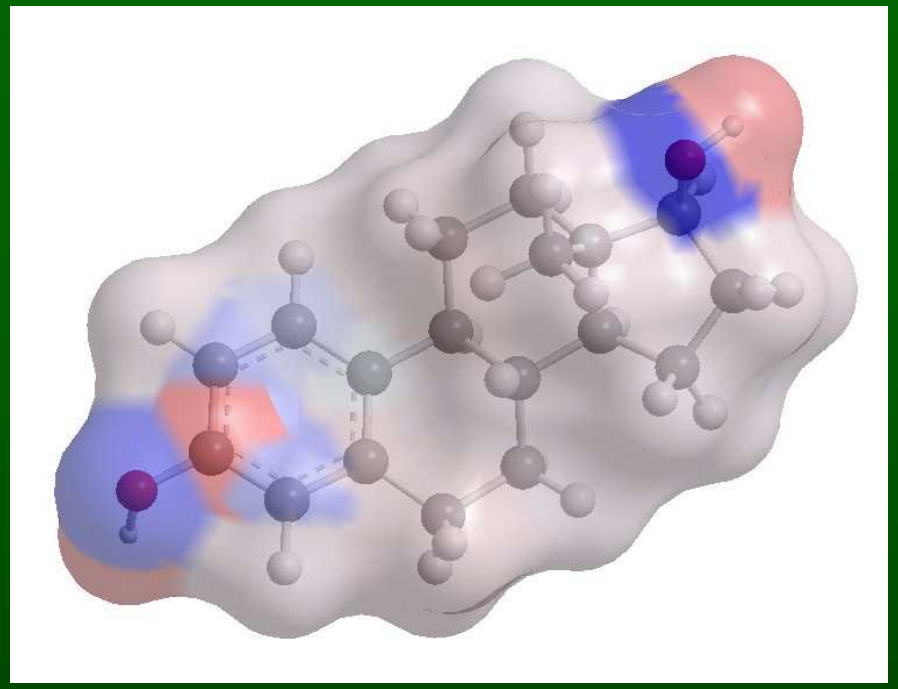
HETEROCHROMATIN



SCIENCE VOL 293 10 AUG 2001 p1065

17-β estradiol





Molecular dynamics simulation of 17-β estradiol at 300 Kelvin (ca. 250 fs)

The 3D-structure of the molecule is depicted on the right side (with partial charges projected onto the `surface´). The atoms of the molecule generate a `force field´ which causes motion of the atoms (e.g. vibration, rotation etc.) that can be depicted by a molecular dynamics simulation. The energy of the motion depends on the thermodynamic temperature. Molecular dynamics illustrates that molecules are not `stiff´ (the same holds true for the protein receptor)! Calculations performed with Cambridgesoft ChemDraw 3D Ultra. The time scale has been extended around 10^{13}-fold. In reality, one half-cycle corresponds to around 250 femtoseconds.

Published in final edited form as:
In Vivo. 2010 ; 24(2): 173–178.

17β-Estradiol Acting as an Electron Mediator: Experiments *In Vitro*

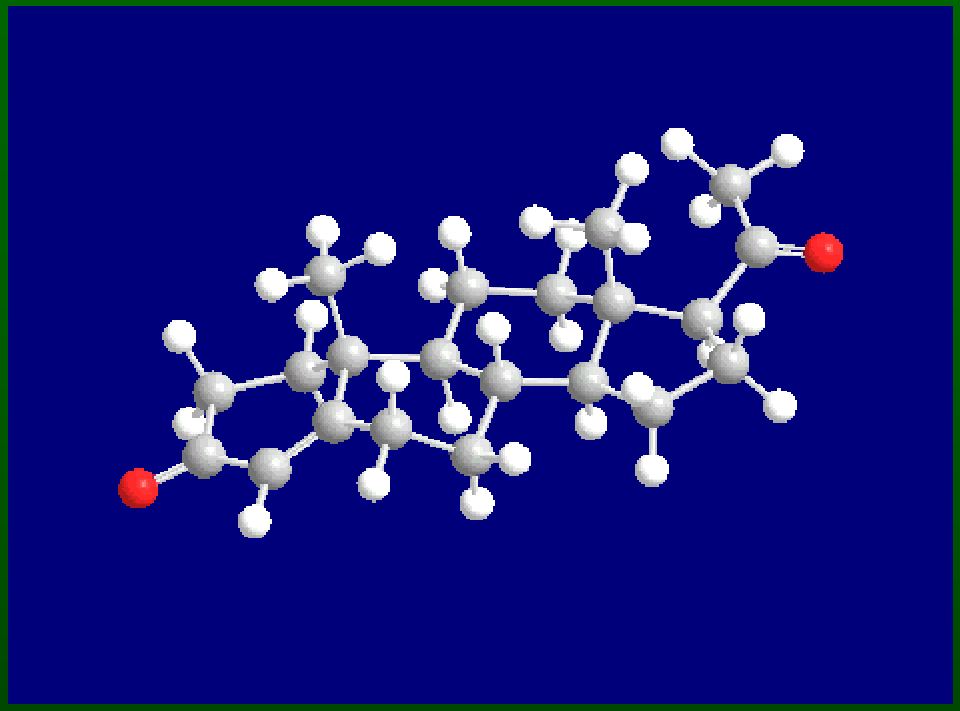
Nikola Getoff[1], **Heike Schittl**[1], **Marion Gerschpacher**[2], **Johannes Hartmann**[2], **Johannes C. Huber**[2], and **Ruth-Maria Quint**[1]

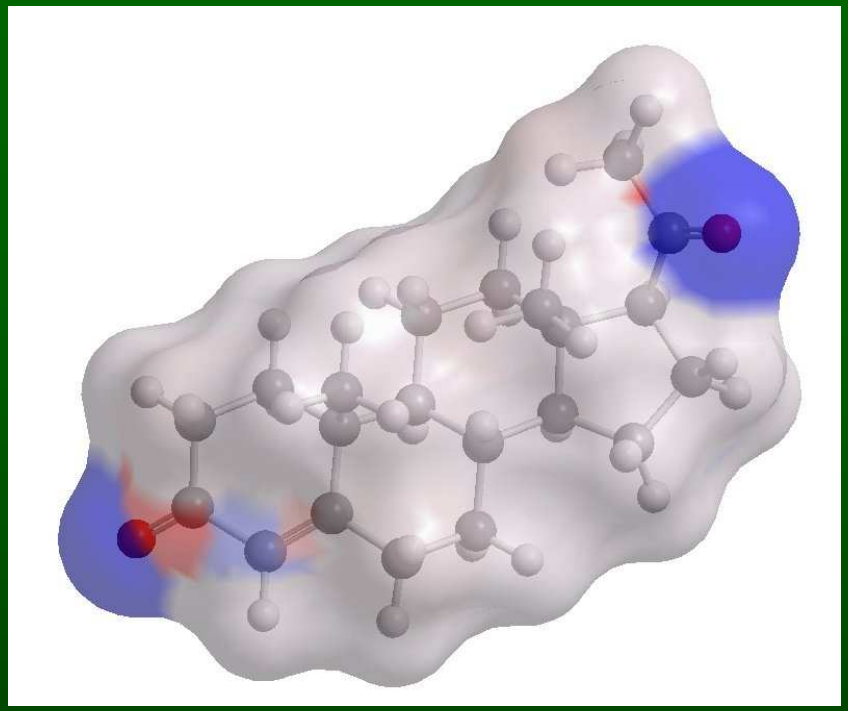
[1] Section of Radiation Biology, Faculty of Life Sciences, University of Vienna, A-1090 Vienna

[2] Department of Gynecological Endocrinology and Reproduction, Medical University of Vienna, A-1090 Vienna

Abstract

progesterone

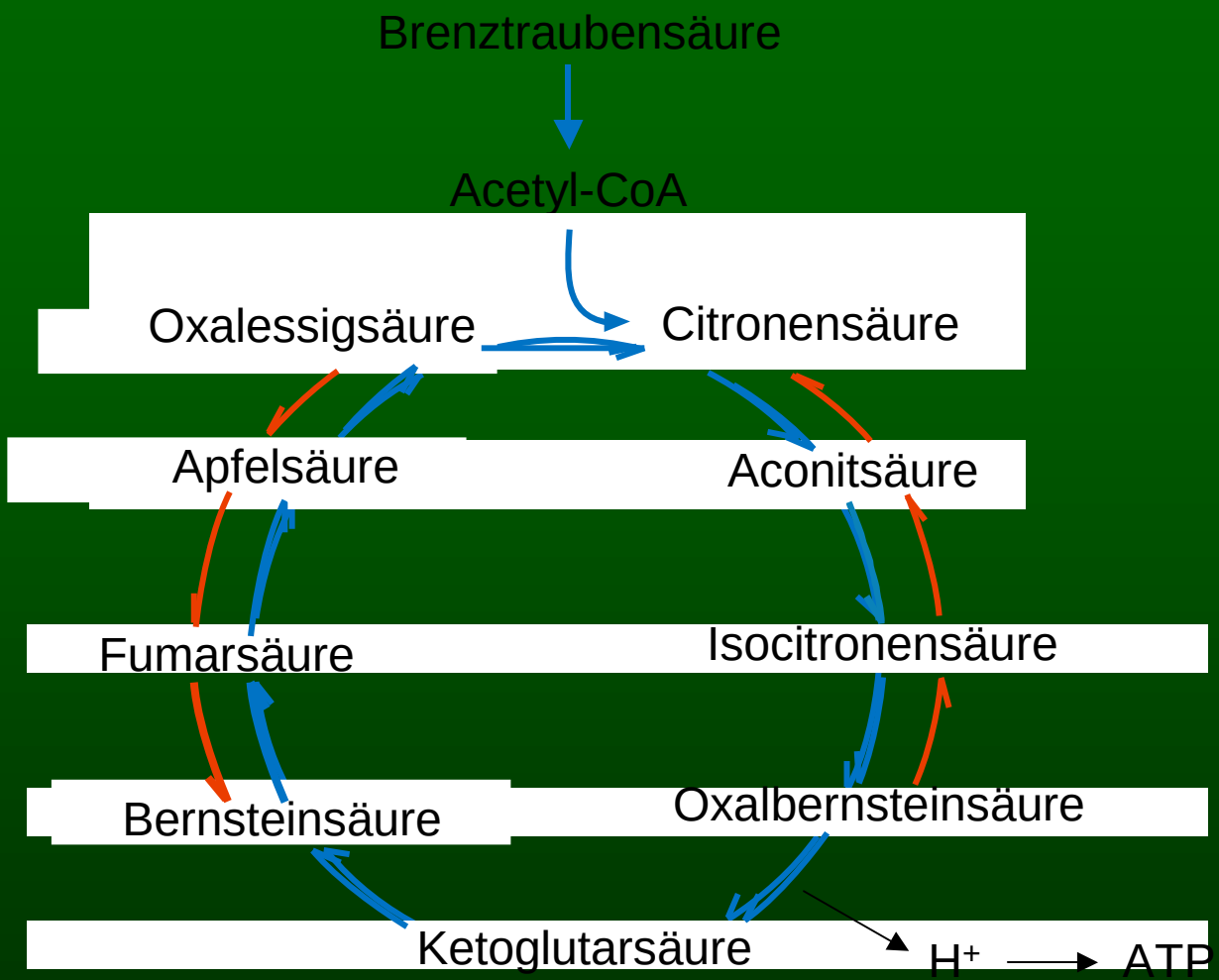


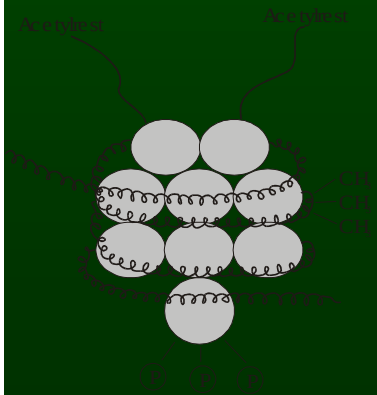


Molecular dynamics simulation of progesterone at 300 Kelvin (ca. 600 fs)

The 3D-structure of the molecule is depicted on the right side (with partial charges projected onto the `surface´). The atoms of the molecule generate a `force field´ which causes motion of the atoms (e.g. vibration, rotation etc.) that can be depicted by a molecular dynamics simulation. The energy of the motion depends on the thermodynamic temperature. Molecular dynamics illustrates that molecules are not `stiff´ (the same holds true for the protein receptor)! Calculations performed with Cambridgesoft ChemDraw 3D Ultra. The time scale has been extended around 10^{13}-fold. In reality, one half-cycle corresponds to around 600 femtoseconds.

METABOLISMUS GREIFT IN DEN EPIGENETISCHEN CODE EIN





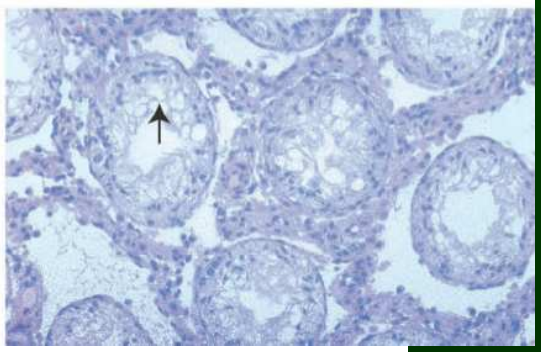
Trans-HHS Workshop: Diet, DNA Methylation Processes and Health

Diet, Methyl Donors and DNA Methylation: Interactions between Dietary Folate, Methionine and Choline[1,2]

Mihai D. Niculescu and Steven H. Zeisel[3]

Department of Nutrition, School of Public Health, School of Medicine, University of North Carolina, Chapel Hill, NC 27599-7400

J. Nutr. 132: 2333S–2335S, 2002



Unfertile ground. The fungicide vinclozolin, which is sp
like these, can cause fertility problems in male offspring

Epigenetic Transgenerational Actions of Endocrine Disruptors and Male Fertility

Matthew D. Anway, Andrea S. Cupp,* Mehmet Uzumcu,† Michael K. Skinner‡

Transgenerational effects of environmental toxins require either a chromosomal or epigenetic alteration in the germ line. Transient exposure of a gestating female rat during the period of gonadal sex determination to the endocrine disruptors vinclozolin (an antiandrogenic compound) or methoxychlor (an estrogenic compound) induced an adult phenotype in the F_1 generation of decreased spermatogenic capacity (cell number and viability) and increased incidence of male infertility. These effects were transferred through the male germ line to nearly all males of all subsequent generations examined (that is, F_1 to F_4). The effects on reproduction correlate with altered DNA methylation patterns in the germ line. The ability of an environmental factor (for example, endocrine disruptor) to reprogram the germ line and to promote a transgenerational disease state has significant implications for evolutionary biology and disease etiology.

DEVELOPMENTAL BIOLOGY

Endocrine Disrupters Trigger Fertility Problems in Multiple Generations

A fungicide and a pesticide, both already known to be toxic to animals, have revealed a potentially even darker side: On page 1466, researchers report that the two chemicals cause fertility defects in male rats that are passed down to nearly every male in subsequent generations. No other known toxin has been shown to do that, according to the study's authors and other scientists. The startling results seem to support the controversial idea that such hormonelike chemicals, known as endocrine disrupters, could be causing population-wide reproductive problems, such as lowered sperm counts in men. But many scientists caution against drawing conclusions until other labs have confirmed the unexpected findings.

malities in lab animals. Over the past 15 years, many scientists have come to think that these endocrine disrupters are potentially causing harmful effects, such as cancer and reproductive abnormalities, in humans, too.

It was already known that when pregnant rats are treated with relatively high doses of vinclozolin every day, their male offspring are sterile, Gray notes. But Skinner and his team found that when they injected vinclozolin into the abdomens of pregnant rats during a specific window of pregnancy—8 to 15 days into gestation—they got a different result. Although the offspring's testes appeared normal and the rodents could reproduce, their sperm count dropped 20% compared to control mice, their sperm motil-

nature International weekly journal of science

Metabolic disorders: Fathers' nutritional legacy

Michael K. Skinner

Nature **467** , 922–923 (21 October 2010) | doi:10.1038/467922a
Published online 20 October 2010

A female can develop a diabetes-like disease due to a high fat content in her father's diet before she was conceived. Epigenetic modifications of the father's sperm DNA might underlie this peculiar observation. See Letter p.963

LETTER

doi:10.1038/nature09491

Chronic high-fat diet in fathers programs β-cell dysfunction in female rat offspring

Sheau-Fang Ng[1], Ruby C. Y. Lin[2], D. Ross Laybutt[3], Romain Barres[4], Julie A. Owens[5] & Margaret J. Morris[1]

ificant role in the chemical
or drink the water. Aluminum
f its abundance and toxicity.

The Epigenetic Sins of the Father

Chemotherapy, irradiation, and environmental toxins can cause DNA damage that, unless repaired, can be transmitted to the

CONTINUED ON PAGE 1375

ORIGINAL ARTICLE

Advancing Paternal Age and Autism

Abraham Reichenberg, PhD; Raz Gross, MD, MPH; Mark Weiser, MD; Michealine Bresnahan, PhD; Jeremy Silverman, PhD; Susan Harlap, MBBS; Jonathan Rabinowitz, PhD; Cory Shulman, PhD; Dolores Malaspina, MD; Gad Lubin, MD; Haim Y. Knobler, MD; Michael Davidson, MD; Ezra Susser, MD, DrPH

Arch Gen Psychiatry. 2006 Sep;63(9):1026-32.

Results: There was a significant monotonic association between advancing paternal age and risk of ASD. Offspring of men 40 years or older were 5.75 times (95% confidence interval, 2.65-12.46; $P<.001$) more likely to have ASD compared with offspring of men younger than 30 years, after controlling for year of birth, socioeconomic status, and maternal age. Advancing maternal age showed no association with ASD after adjusting for paternal age. Sensitivity analyses indicated that these findings were not the result of bias due to missing data on maternal age.

Conclusions: Advanced paternal age was associated with increased risk of ASD. Possible biological mechanisms include de novo mutations associated with advancing age or alterations in genetic imprinting.

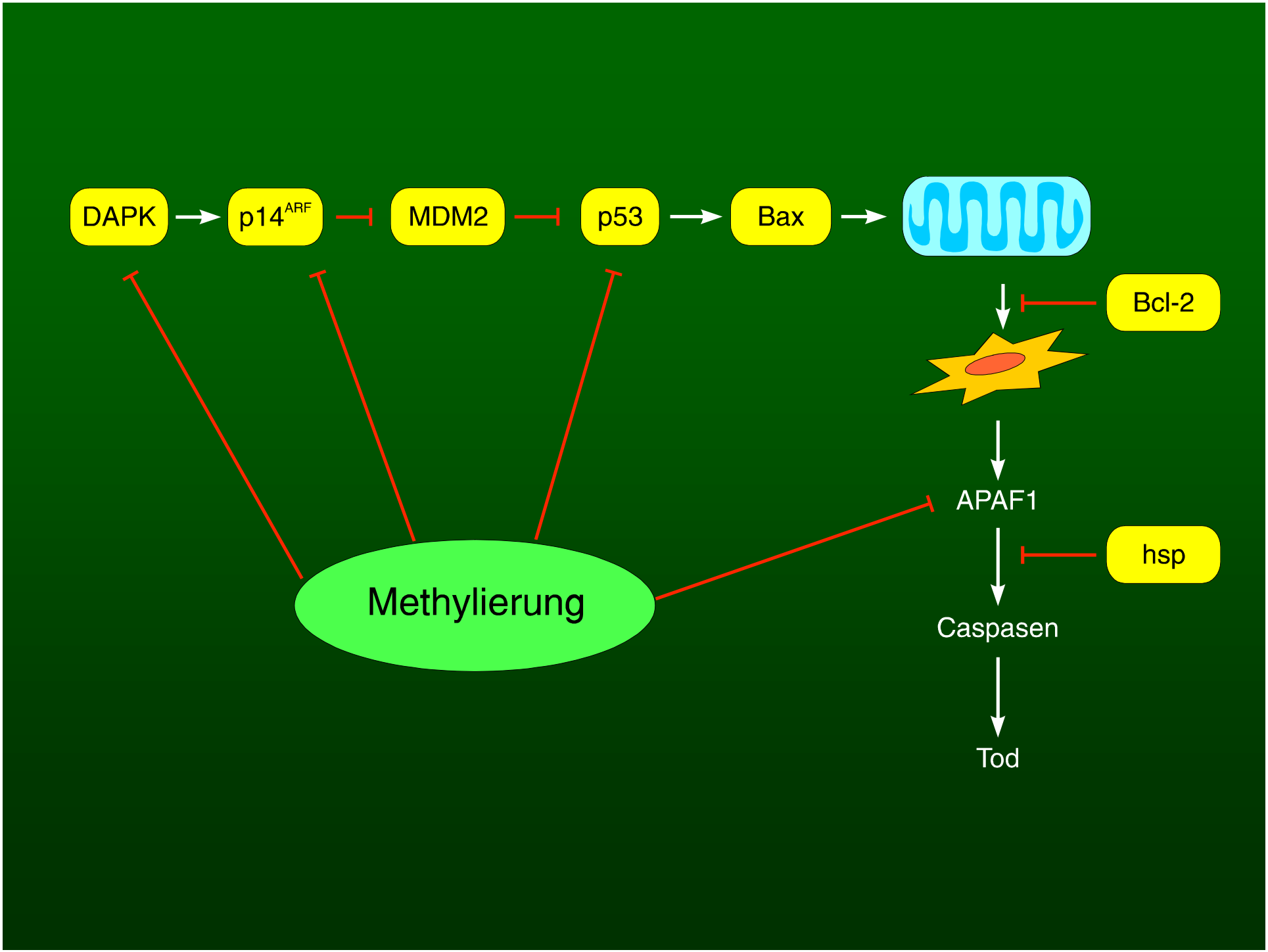
Reichenberg A et al. Arch Gen Psychiatry. 2006 Sep;63(9):1026-32.

REVIEWS

THE FUNDAMENTAL ROLE OF EPIGENETIC EVENTS IN CANCER

Peter A. Jones and Stephen B. Baylin‡*

Patterns of DNA methylation and chromatin structure are profoundly altered in neoplasia and include genome-wide losses of, and regional gains in, DNA methylation. The recent explosion in our knowledge of how chromatin organization modulates gene transcription has further highlighted the importance of epigenetic mechanisms in the initiation and progression of human cancer. These epigenetic changes — in particular, aberrant promoter hypermethylation that is associated with inappropriate gene silencing — affect virtually every step in tumour progression. In this review, we discuss these epigenetic events and the molecular alterations that might cause them and/or underlie altered gene expression in cancer.


DAPK
p14ARF
MDM2
p53
Bax
Bcl-2
APAF1
hsp
Caspasen
Tod
Methylierung

The NEW ENGLAND JOURNAL of MEDICINE

REVIEW ARTICLE

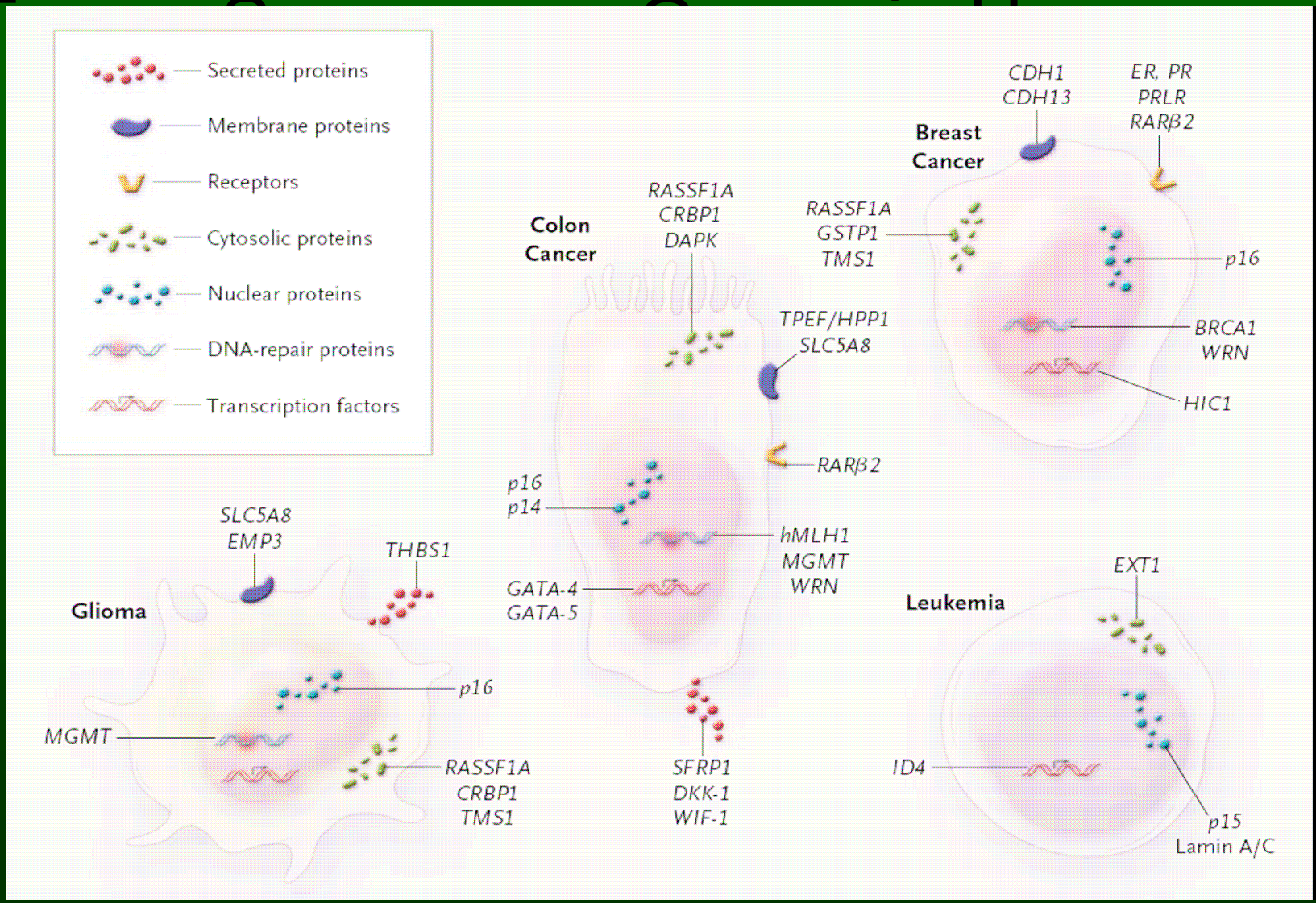
MOLECULAR ORIGINS OF CANCER

Epigenetics in Cancer

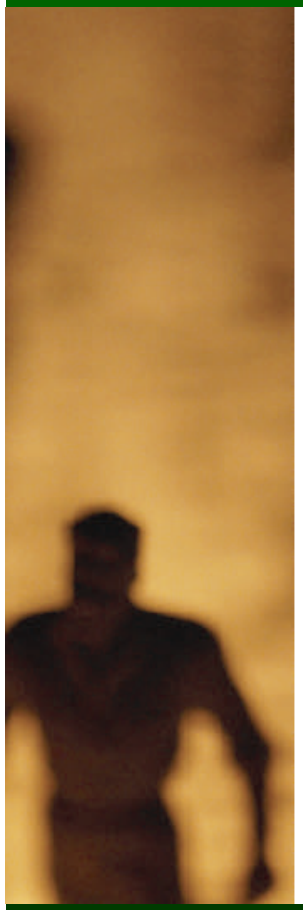
Manel Esteller, M.D., Ph.D.

N Engl J Med. 2008 Mar 13;358(11):1148-59.

Profile of Hypermethylation of the CpG Island in the Promoter Region of T



Esteller M, N Engl J Med. 2008 Mar 13;358(11):1148-59.



Epigenetics in human disease and prospects for epigenetic therapy

Gerda Egger, Gangning Liang, Ana Aparicio & Peter A. Jones

Departments of Biochemistry and Molecular Biology and Urology, USC/Norris Comprehensive Cancer Center, Keck School of Medicine of the University of Southern California, 1441 Eastlake Avenue, Room 8302L, Los Angeles, California 90089-9181, USA (e-mail: jones_p@ccnt.hsc.usc.edu)

Epigenetic mechanisms, which involve DNA and histone modifications, result in the heritable silencing of genes without a change in their coding sequence. The study of human disease has focused on genetic mechanisms, but disruption of the balance of epigenetic networks can cause several major pathologies, including cancer, syndromes involving chromosomal instabilities, and mental retardation. The development of new diagnostic tools might reveal other diseases that are caused by epigenetic alterations. Great potential lies in the development of 'epigenetic therapies' — several inhibitors of enzymes controlling epigenetic modifications, specifically DNA methyltransferases and histone deacetylases, have shown promising anti-tumorigenic effects for some malignancies.

Table 2 **Epigenetic drugs**

Target	Drug	Clinical trials
DNA methylation	5-Azacytidine	Phase I/II/III
	5-Aza-2′-deoxycytidine	Phase I/II/III
	FCDR	
	Zebularine	
	Procainamide	
	EGCG	Phase I
	Psammaplin A	
	Antisense oligomers	Phase I
Histone deacetylase	Many[55], including:	
	Phenylbutyric acid	Phase I/II
	SAHA	Phase I/II
	Depsipeptide	Phase I/II
	Valproic acid	Phase I/II

EGCG, epigallocatechin-3-gallate; FCDR, 5-fluoro-2′-deoxycytidine; SAHA, suberoylanilide hydroxamic acid.

Epigenetic therapy

The NEW ENGLAND JOURNAL of MEDICINE

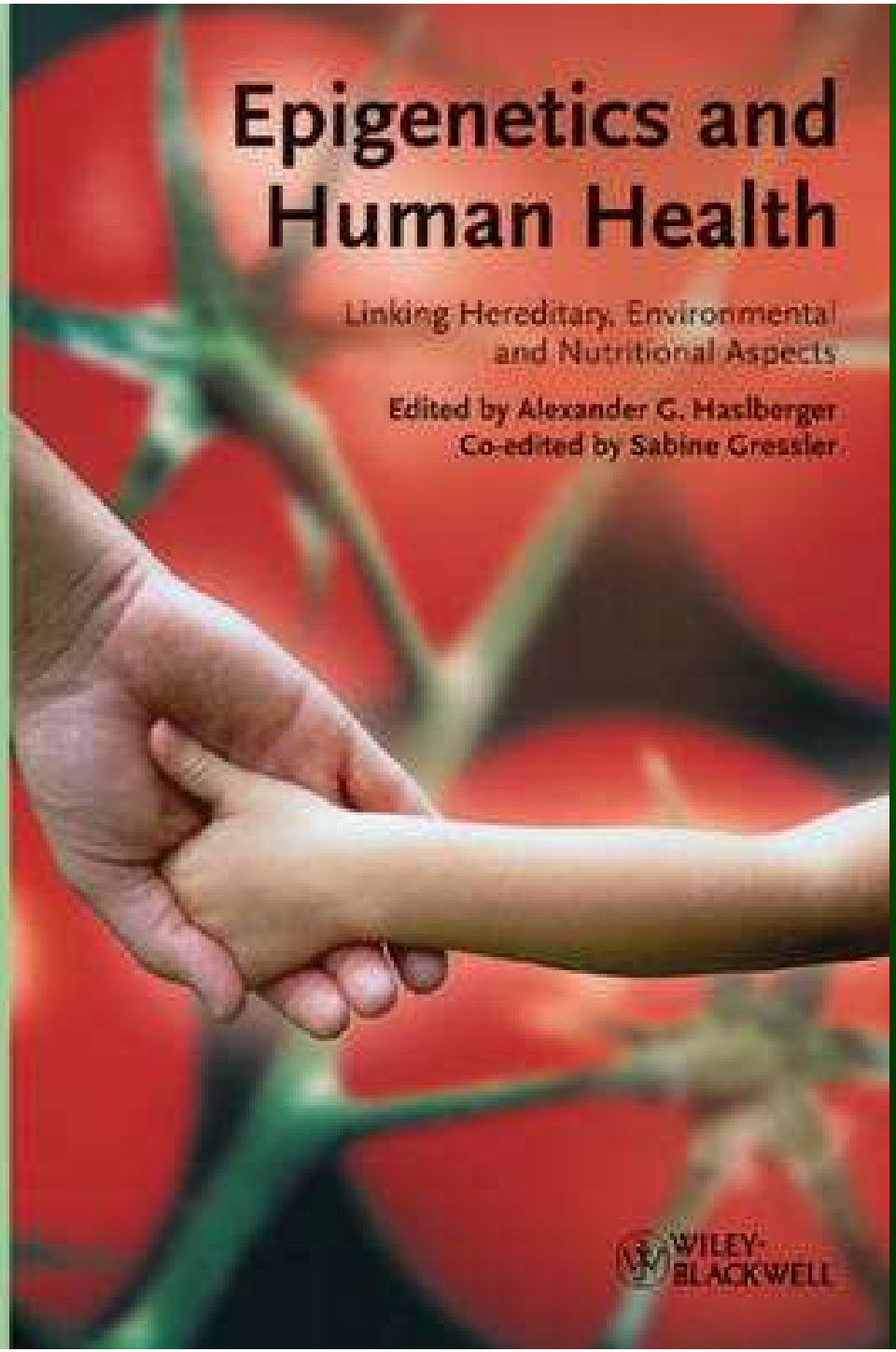
REVIEW ARTICLE

MECHANISMS OF DISEASE

Effect of In Utero and Early-Life Conditions on Adult Health and Disease

Peter D. Gluckman, M.D., D.Sc., Mark A. Hanson, D.Phil., Cyrus Cooper, M.D., and Kent L. Thornburg, Ph.D.

N Engl J Med. 2008 Jul 3;359(1):61-73.


Epigenetics and Human Health
Linking Hereditary, Environmental and Nutritional Aspects
Edited by Alexander G. Haslberger
Co-edited by Sabine Gressler
WILEY-BLACKWELL

14
Epigenetics Aspects in Gyneacology and Reproductive Medicine

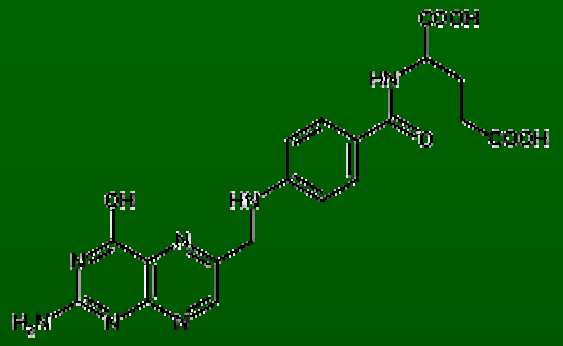
Alexander Just and Johannes Huber

Abstract

Epigenetics regulates human genotypical activity throughout the entire lifecycle. Preexisting schemes in our genome's methylization and acetylization are modified through interaction with our environment and nutrition. Because the genome is not static, it is highly adaptable, allowing adjustment to the environment. Further, pregnancy, birth and early childhood determine our lives themselves. Scientific knowledge of epigenetics will influence different aspects in medicine care in the future. Our intention is to describe some special facts in this field. Epigenetics frees us of the theory of a destiny that is predefined genetically. We have much more responsibility for our own and our descendants' daily lives.

Epigenetics and Human Health
Edited by Alexander G. Haslberger, Co-edited by Sabine Gressler

ISBN: 978-3-527-32427-9



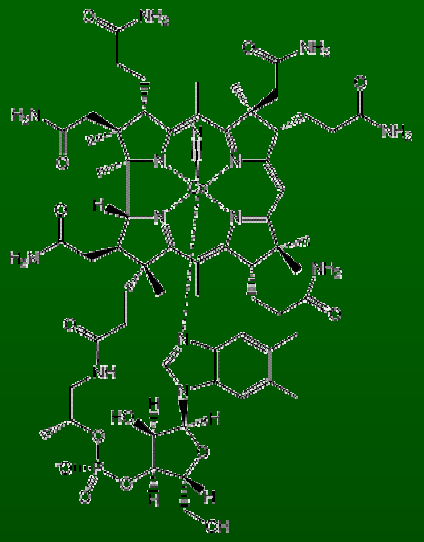
Folsäure

Perikonzeptionelles Defizit von

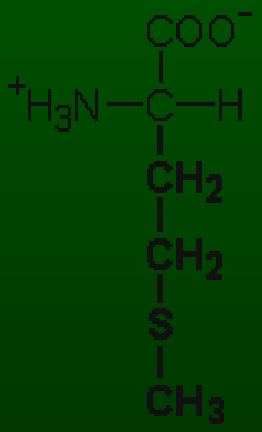
Vitamin B12
Folat

Methionin

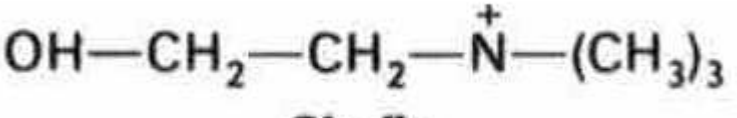
Cholin



Vitamin B12



Methionin



Cholin

bewirkt multiple Risikoerhöhung
für das Kind

Yajnik CS et al. Diabetologia 2008;51:29-38.

Empfohlene Tageseinnahme (Adequate Intake = AI)

- Männer: 550 mg Choline / d
- Frauen: 425 mg Choline / d
- Schwangerschaft: 850 mg Choline / d

3. Institute of Medicine, National Academy of Sciences USA. Choline. Dietary reference intakes for folate, thiamin, riboflavin, niacin, vitamin B12, panthothenic acid, biotin, and choline. Washington, DC: National Academy Press, 1998:390–422.

Nichtgravide Frauen

10,7 µM freies Cholin

2,7 µM gebundenes Cholin

Schwangere Frauen (SSW 36)

16,5 µM freies Cholin

3,5 µM gebundenes Cholin

11. Ozarda I, Uncu G, Ulus I. Free and phospholipid-bound choline concentrations in serum during pregnancy, after deliver and in newborns. Arch Physiol Biochem 2002;110:393–399. [PubMed: 12530624]

doi: 10.1111/j.1365-2222.2009.03234.x

Clinical & Experimental Allergy, **39**, 875–882

ORIGINAL ARTICLE Epidemiology of Allergic Disease

Maternal vitamin D intake during pregnancy is inversely associated with asthma and allergic rhinitis in 5-year-old children

M. Erkkola*, M. Kaila†, B. I. Nwaru‡, C. Kronberg-Kippilä§, S. Ahonen‡, J. Nevalainen‡, R. Veijola¶, J. Pekkanen‖,**, J. Ilonen††,‡‡, O. Simell§§, M. Knip†,¶¶ and S. M. Virtanen†,‡,§

Maternal vitamin D intake during pregnancy and early childhood wheezing[1–4]

Graham Devereux, Augusto A Litonjua, Stephen W Turner, Leone CA Craig, Geraldine McNeill, Sheelagh Martindale, Peter J Helms, Anthony Seaton, and Scott T Weiss

Conclusion: Increasing maternal vitamin D intakes during pregnancy may decrease the risk of wheeze symptoms in earlychildhood.

Am J Clin Nutr 2007;85:853–9.

Am J Clin Nutr 2007;85:853–9.

Pediatr Allergy Immunol 2010: 21: 29–37
DOI: 10.1111/j.1399-3038.2009.00949.x

PEDIATRIC ALLERGY AND IMMUNOLOGY

Maternal diet during pregnancy and allergic sensitization in the offspring by 5 yrs of age: a prospective cohort study

Nwaru BI, Ahonen S, Kaila M, Erkkola M, Haapala A-M, Kronberg-Kippilä C, Veijola R, Ilonen J, Simell O, Knip M, Virtanen SM.
Maternal diet during pregnancy and allergic sensitization in the offspring by 5 yrs of age: a prospective cohort study.
Pediatr Allergy Immunol 2010: 21: 29–37.

Maternal consumption of citrus fruits during pregnancy may increasethe risk to allergic sensitization in the offspring, whereas vitamin D intake may have a beneficial effect.

Acta Neurol Scand 2010: 121: 20–23 DOI: 10.1111/j.1600-0404.2009.01181.x

ACTA NEUROLOGICA
SCANDINAVICA

Season of birth and multiple sclerosis in Sweden

Salzer J, Svenningsson A, Sundström P. Season of birth and multiple sclerosis in Sweden.
Acta Neurol Scand: 2010: 121: 20–23.

Antnataler Stress

im 2. Trimenon
ist assoziiert mit erhöhtem Risiko für

Schizophrenie

Lit:
Hultmann CM et al. Br. Med J 1999;318:421-426.
Imamura Y et al. Acta Psychiatr. Scand. 1999;100: 344-349.
Van Os J et al. Br.J.Psychiatry 1998;172:324-326.

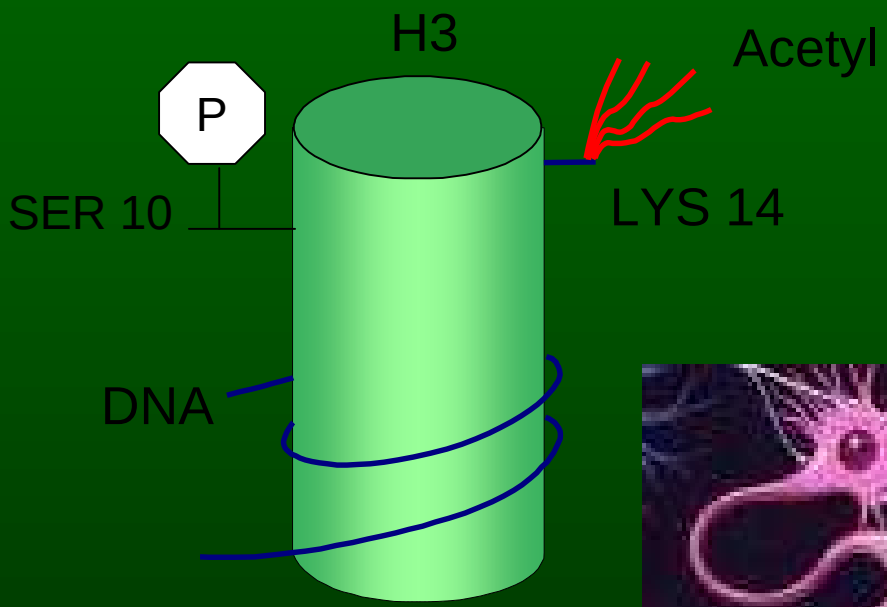
Selten

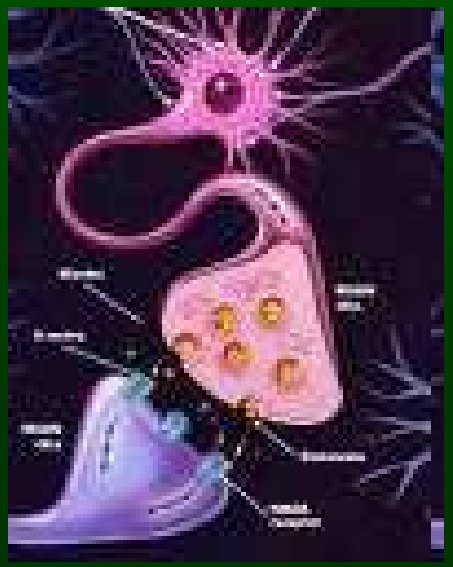
Depression – Drug Abuse

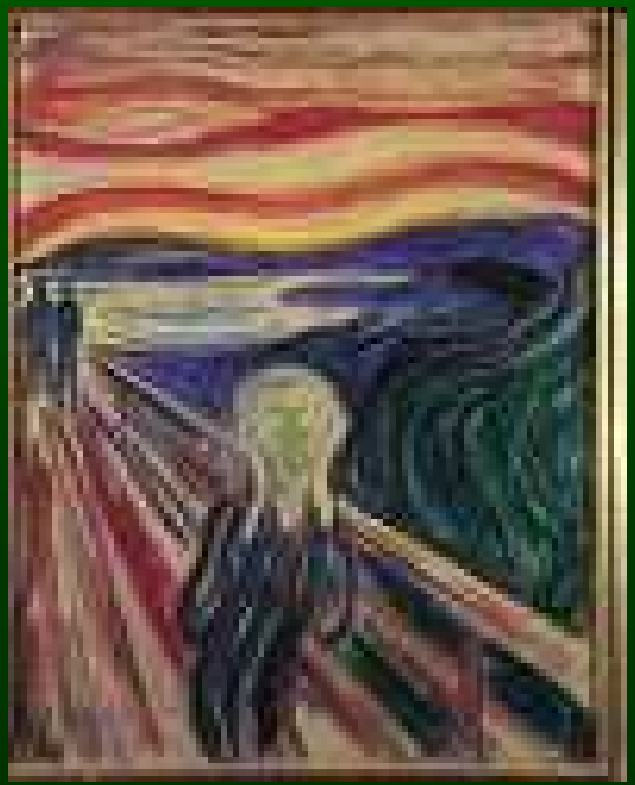
Lit:
Huttunen MO et al. Arch Gen. Psychiatry 1978;35:429-431.
Niskanen
Watson JB et al. Dev. Psychopathol. 1999;11:457-466.

Weinstock M, Brain Behav Immun. 2005 Jul;19(4):296-308. Epub 2004 Nov 19.

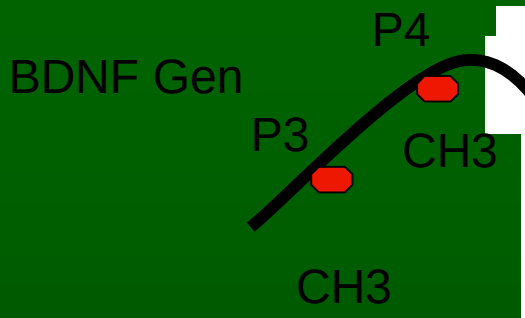
ANGST

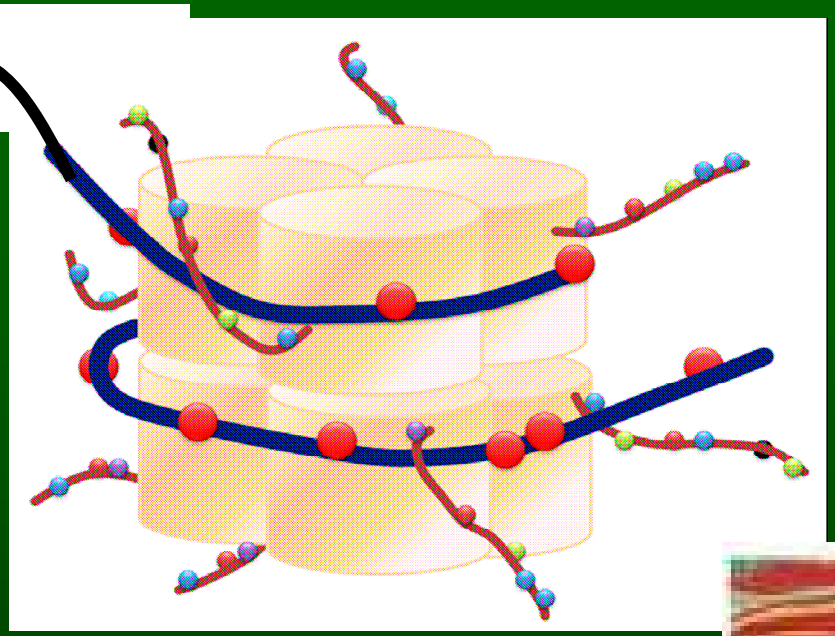




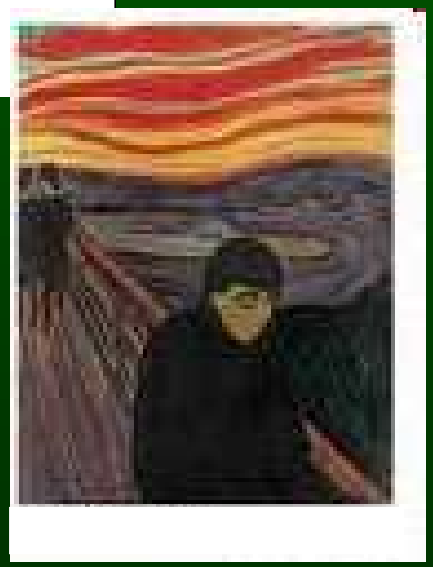


SOZIALER STRESS





IMIPRAMIN
FLUOXETIN



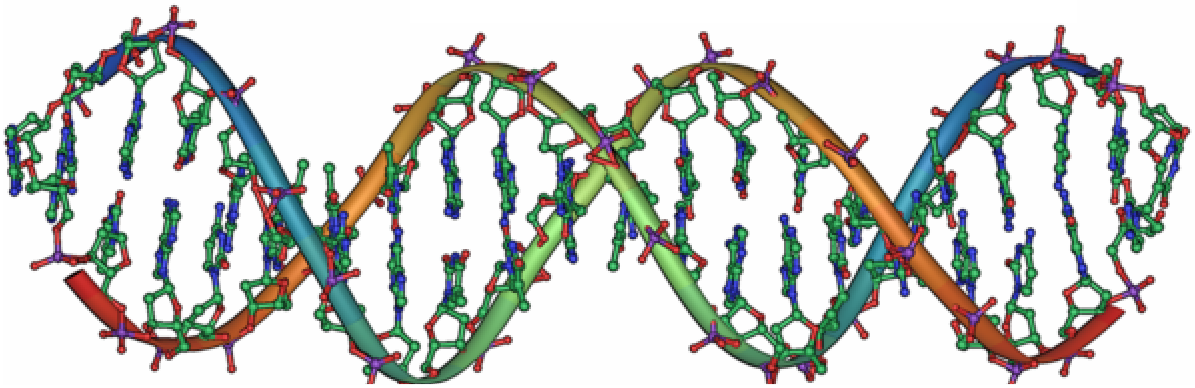
Tsankova NM et al. Nat. Neurosci. 9 (2006) 519–525.

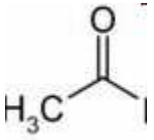
COCAIN (AMPHETAMIN)

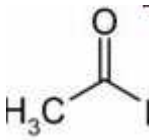
FOS B Promotor

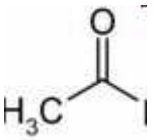
BDNF Promotor

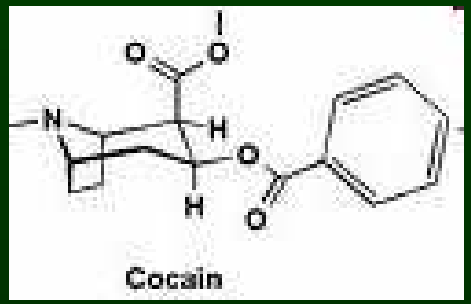
Cdk5 Promotor

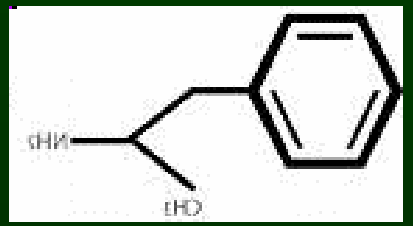












Intrauterine Corticoide

reduzieren

- Duodenales Homeoboxgen 1
- Pankreatischen Transkriptionsfaktor Pdx1
- Anzahl der β-Zellen

Ozanne SE et al. Diabetologia 2005;48:547-52.

Intrauterine Corticoide

erhöhen das postpartale Risiko für

- Hypertonie

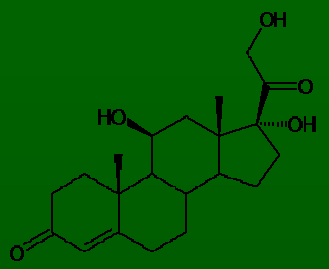
Charalambous M et al. Curr Opin Endocrinol Diabetes Obes 2007;14:3-12.

- Insulinresistenz

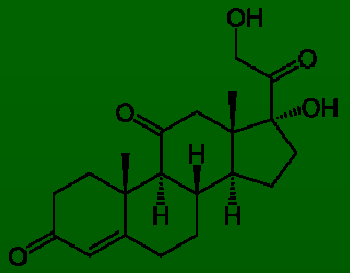
Nyirenda MJ et al. J. Clin Invest 1998; 101:2174-81.

- Stressreaktionen

Welberg LAM et al. Neuroscience 2001;104:71-9.



Cortisol



Cortison

Cortisol ⟶ Cortison Plazenta

↑

17β Hydroxysteroiddehydrogenase 2

⊤

Stress, Nikotin, Malnutrition

Brown RW et al. Endocrinology 1996;137:794–7.
Kajantie E et al. J Clin Endocrinol Metab 2003;88(1):493–500.

De Weerth C et al. Early Hum Dev 2003;74:139– 51.

Huizink AC et al. J Am Acad Child Adolesc Psychiatry 2002;41(9):1078 – 85.

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Brouwers EPM et al. Infant Behav Dev 2001;24(1):95 –106.

O’Connor TG et al. Br J Psychiatry 2002;180:502–8.

Wadhwa Pd et al. Prog Brain Res 2001;133:131– 42.

Reversal of Maternal Programming of Stress Responses in Adult Offspring through Methyl Supplementation: Altering Epigenetic Marking Later in Life

Ian C. G. Weaver,[1,2] Frances A. Champagne,[1] Shelley E. Brown,[3] Sergiy Dymov,[3] Shakti Sharma,[1] Michael J. Meaney,[1,2] and Moshe Szyf[2,3]

The Journal of Neuroscience, November 23, 2005 • 25(47):11045–11054 • 11045

Human Molecular Genetics, 2009, Vol. 18, No. 20 **3769–3778**
doi:10.1093/hmg/ddp319
Advance Access published on July 15, 2009

DNA methylation and gene expression differences in children conceived *in vitro* or *in vivo*

Sunita Katari[1†], Nahid Turan[1†], Marina Bibikova[2], Oluwatoyin Erinle[1], Raffi Chalian[3], Michael Foster[3], John P. Gaughan[4], Christos Coutifaris[3] and Carmen Sapienza[1,5,*]

Several of the genes whose expression differs between the two groups have been implicated in chronic metabolic disorders, such as obesity and type II diabetes. These findings suggest that there may be epigenetic differences in the gametes or early embryos derived from couples undergoing treatment for infertility. Alternatively, assisted reproduction technology may have an effect on global patterns of DNA methylation and gene expression. In either case, these differences or changes may affect long-term patterns of gene expression.

PROCEEDINGS OF THE ROYAL SOCIETY B

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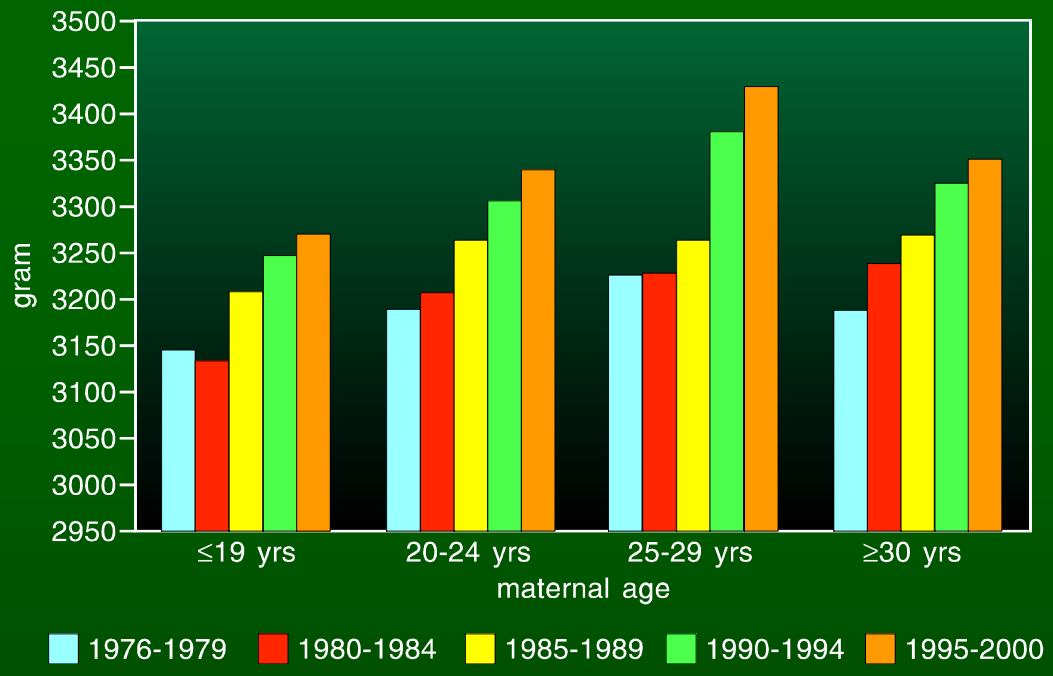
Proc. R. Soc. B
doi:10.1098/rspb.2010.2314
Published online

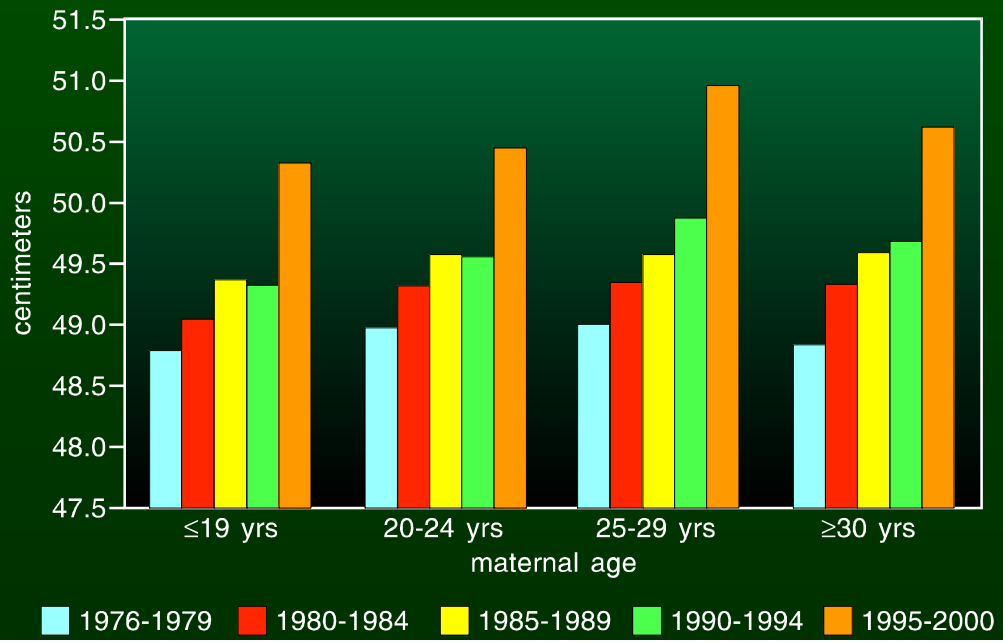
Effects of *in utero* odorant exposure on neuroanatomical development of the olfactory bulb and odour preferences

Josephine Todrank[1,2,*], Giora Heth[1,2] and Diego Restrepo[1]

Mozarts Nachtmusik lässt Frühchen gedeihen

Wenn Frühgeborene Musik von Mozart hören, verbrennt ihr Körper weniger Energie unter Ruhebedingungen, und sie entwickeln sich dadurch günstiger. Israelische Neonatologen vom Lis-Maternity-Krankenhaus in Tel Aviv haben das herausgefunden, indem sie den Energieverbrauch von Frühgeborenen überprüften. Die Babys waren nach der dreißigsten bis sechsunddreißigsten Schwangerschaftswoche geboren worden. Sie wurden noch im Brutkasten versorgt, waren jedoch gesundheitlich stabil. Bereits nachdem die eine Gruppe zehn Minuten lang eine Baby-Mozart-CD hörte, sank deren Nährstoff- und Sau-

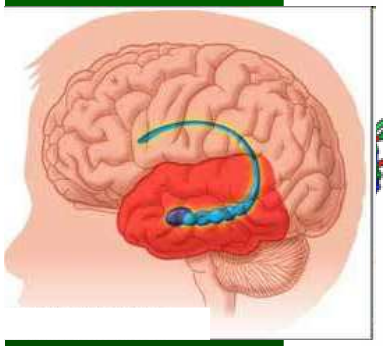

3500
3450
3400
3350
3300
3250
3200
3150
3100
3050
3000
2950
gram
≤19 yrs
20-24 yrs
25-29 yrs
≥30 yrs
maternal age
1976-1979
1980-1984
1985-1989
1990-1994
1995-2000

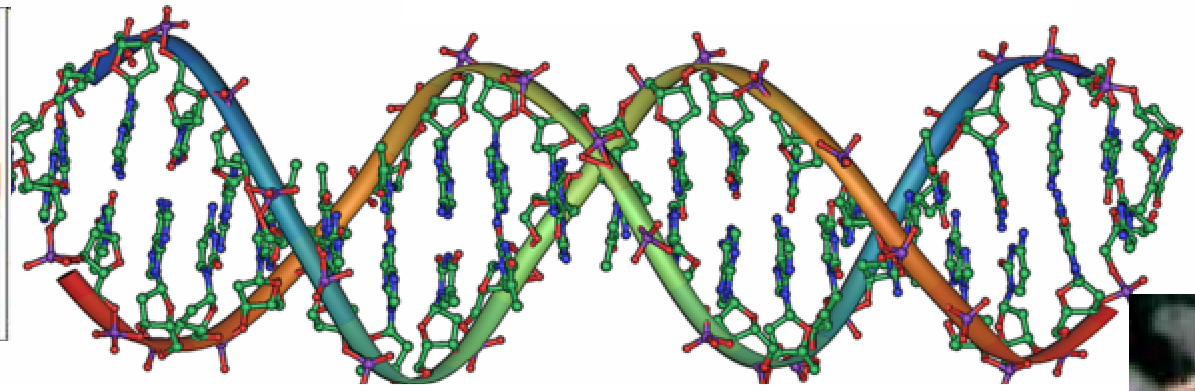

51.5
51.0
50.5
50.0
49.5
49.0
48.5
48.0
47.5
centimeters
≤19 yrs
20-24 yrs
25-29 yrs
≥30 yrs
maternal age
1976-1979
1980-1984
1985-1989
1990-1994
1995-2000

Fehlende taktile Reize postpart

GLUCOKORTIKOIDREZEPTOR

Ex 1 Ex2 Ex3 Ex4 Ex5 Ex6 Ex7



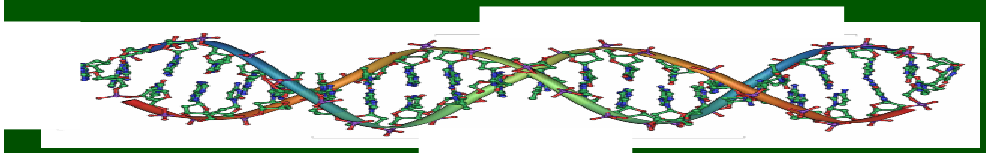


CH 3 CH 3 CH 3 CH 3 CH 3 CH 3 CH 3

NGF 1 A

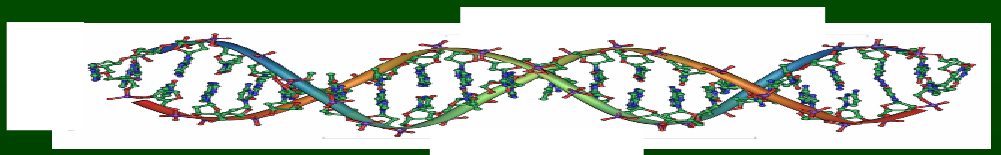
Fehlende maternal care

OXYITOCIN REZEPTOR GEN

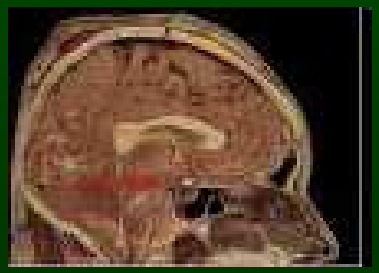


CH 3 **CH 3** **CH 3**

ÖSTROGEN REZEPTOR GEN



CH 3 **CH 3** **CH 3** **CH 3**



Area praeoptica

Fehlende taktile Reize postpartal

verändern Cortisolrezeptoren

im Hippocampus und damit die Amplitude

der Stressreaktionen

Weaver ICG et al. Nat Neurosci 2004;7:847- 54.

Fehlen taktile Reize in Postpartalphase

Hypermethylierung des

Oxytocin Rezeptor Gens

Östrogen Rezeptor α Gens

in Area Präoptica

Champagne FA. et al.Endocrinology 147 (2006) 2909–2915.

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ScienceDirect
Frontiers in Neuroendocrinology 29 (2008) 398–412
Frontiers in Neuroendocrinology
www.elsevier.com/locate/yfrne

Review

Epigenetics, brain evolution and behaviour

Eric B. Keverne [a,*], James P. Curley [b]

[a] *Sub-Department of Animal Behaviour, University of Cambridge, Madingley, Cambridge, CB23 8AA, UK*
[b] *Department of Psychology, Columbia University, New York, 10025, USA*

Available online 10 March 2008

Frühgeburtlichkeit (vor SSW 36)

Erhöht in Pubertät das Risiko für

Insulinresistenz

Hypertonie

Hofman PL et al. N Engl J Med 2004;351:2179-86. [Erratum, N Engl J Med 2004;351:2888.]

Hovi P et al. N Engl J Med 2007;356:2053-63.

Diabetologia (2005) 48: 547–552
DOI 10.1007/s00125-005-1669-7

ARTICLE

S. E. Ozanne · C. B. Jensen · K. J. Tingey ·
H. Storgaard · S. Madsbad · A. A. Vaag

Low birthweight is associated with specific changes in muscle insulin-signalling protein expression

Received: 30 September 2004 / Accepted: 25 October 2004 / Published online: 24 February 2005

Mütterliche Unterernährung

begünstigt

zentrale Obesity

Skelettmuskelreduktion beim Kind

Vickers MH et al. Am J Physiol Endocrinol Metab 2000;279:E83-E87.

Langley-Evans SC et al. Life Sci 1999;64:965-74.

Überschiessende postpartale Gewichtskompensation

intrauterine Wachstumsretardierung

prädisponiert zu
Koronarerkrankungen
Hypertonie
Diabetes 2

Bharghava SK et al. N Engl J Med 2004;350:865-75.

Barker DJP et al. N Engl J Med 2005; 353:1802-9.

Epigenetic regulation of the glucocorticoid receptor in human brain associates with childhood abuse

Patrick O McGowan[1,2], Aya Sasaki[1,2], Ana C D'Alessio[3], Sergiy Dymov[3], Benoit Labonté[1,4], Moshe Szyf[2,3], Gustavo Turecki[1,4] & Michael J Meaney[1,2,5]

VOLUME 12 | NUMBER 3 | MARCH 2009 NATURE NEUROSCIENCE

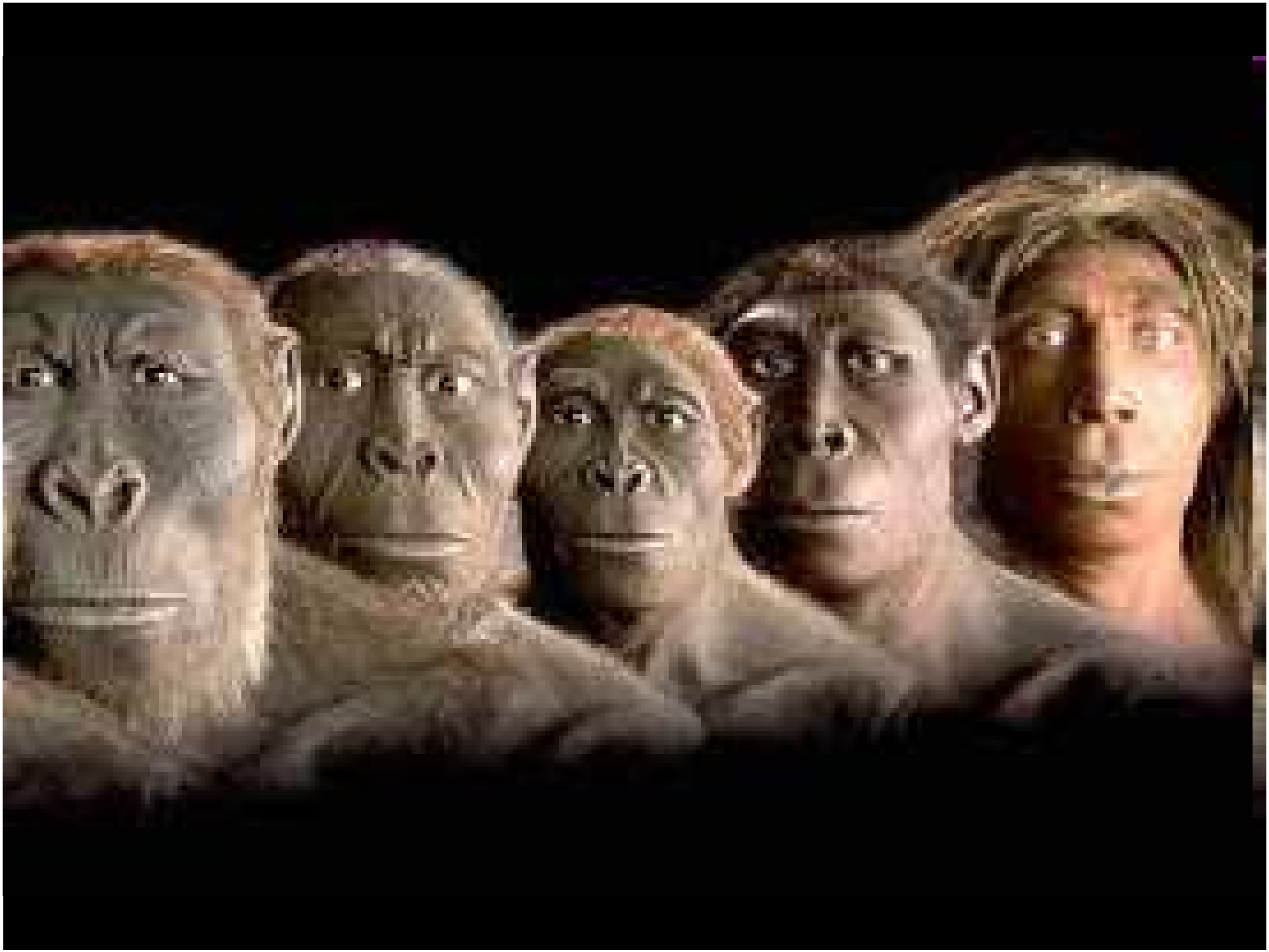
Low early-life social class leaves a biological residue manifested by decreased glucocorticoid and increased proinflammatory signaling

Gregory E. Miller[a,1], Edith Chen[a], Alexandra K. Fok[b,c], Hope Walker[a], Alvin Lim[a], Erin F. Nicholls[a], Steve Cole[d,e,f], and Michael S. Kobor[b,c]

14716–14721 | PNAS | August 25, 2009 | vol. 106 | no. 34

Fred Hoyle

Edwin Hubble




DER SPIEGEL
„GOTT IST AN ALLEM SCHULD!“
Der Kreuzzug der neuen Atheisten

Vol 462|24/31 December 2009 nature

NEWS & VIEWS

AGEING

Diet and longevity in the balance

Thomas Flatt

SCIENCE VOL 328 16 APRIL 2010

Photograph of a dietary restriction practitioner before starting dietary restriction with adequate nutrition and after 7 years of dietary restriction