Stress

Montag 12:15 – 13:45 Uhr

HZO 80

Prof. Oliver T. Wolf

FAKULTÄT FÜR PSYCHOLOGIE
Arbeitseinheit Kognitionspsychologie
www.cog.psy.ruhr-uni-bochum.de

Terminübersicht

20.10.25	Übersicht und Einführung
27.10.25	Stress und das SNS: Walter Cannon (Dr. Katja Langer)
03.11.25	Stress und die HHNA: Hans Selye
10.11.25	Stress und die HHNA: Munck und Sapolsky Die kognitive Wende: Lazarus
17.11.25	Stress und Gesundheit: McEwen und die allostatische Belastung Stress im Arbeitsleben: Siegrist und die Effort Reward Imbalance
24.11.25	Burnout (Dipl. Psych. Natalie Freund)
01.12.25	Soziale Evaluation als bedeutsamer Stressor: Dickerson & Kemeny (Dr. Katja Langer)
08.12.25	Soziale Unterstützung als Stresspuffer/Oxytozin
15.12.25	Stress und Gehirn: akute und chronische Effekte
12.01.26	Pränataler Stress und seine Folgen
(Aufzeichnung)	Frühkindlicher Stress und seine Folgen (Prof. Robert Kumsta)
19.01.26	Posttraumatische Belastungsstörung

Beispielfrage K-Prim

Folgende Resultate kann die primäre Einschätzung gemäß Lazarus erbringen

Antwort	wahr	falsch
motivirrelevant		
bedrohlich		
herausfordernd		
günstig-positiv		

Literatur

- Schlotz, W., & Phillips, D.I.W. (2009). Fetal origins of mental health: Evidence and mechanisms. *Brain, Behavior and Immunity*, *23,* 905-916.
- Glover, V. (2011). Annual Research Review: Prenatal stress and the origins of psychopathology: an evolutionary perspective. *Journal of Child Psychology and Psychiatry, 52*, 356–367.
- Entringer, S., Buss, C., & Wadhwa, P. D. (2015). Prenatal stress, development, health and disease risk: A psychobiological perspective – 2015 Curt Richter Award Paper. *Psychoneuroendocrinology, 62,* 366-375.

Pränatale Einflüsse

Gliederung

- Barker-Hypothese
- Evolutionäre Perspektive
- Psychobiologische Perspektive

Pränataler Stress: Effekte (Coe et al., 2003)

- HHNA-Hyperaktivität
- reduzierte Neurogenese
- vermindertes Hippocampusvolumen

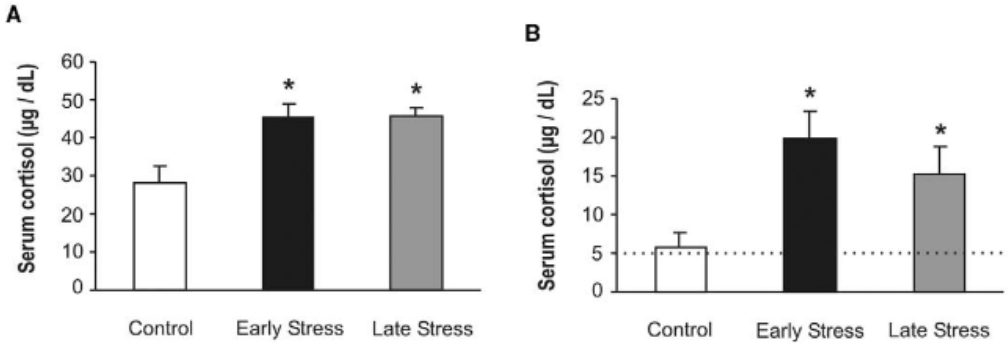


Figure 2. Basal cortisol levels **(A)** and values after an overnight dexamethasone suppression test (DST) **(B)**. Animals generated from early and late stress pregnancies had significantly elevated cortisol levels, both at baseline and after the DST. Horizontal dashed line indicates the maximum level of plasma cortisol (5 µg/dL) considered normal (e.g., failure to stay suppressed). Results are presented as mean ± SEM (µg cortisol/dL). For statistical comparisons, one-way analysis of variance was used, followed by Tukey's multiple comparison test. * $p < .05$, significantly different from the control monkeys.

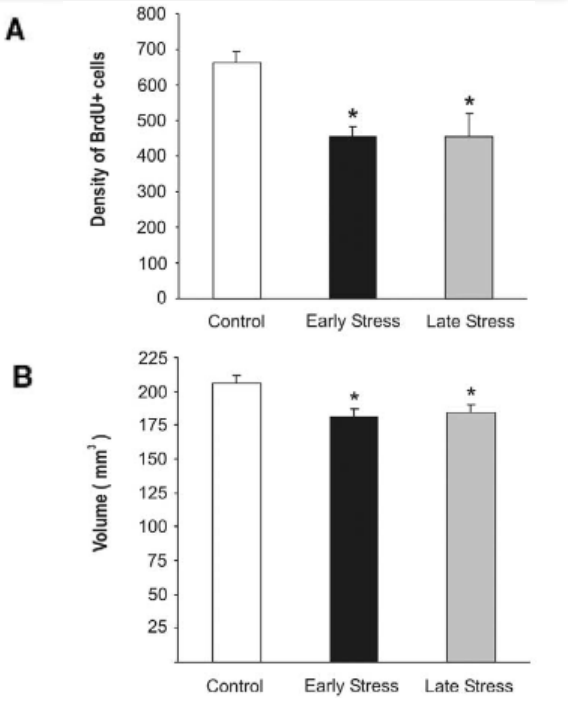


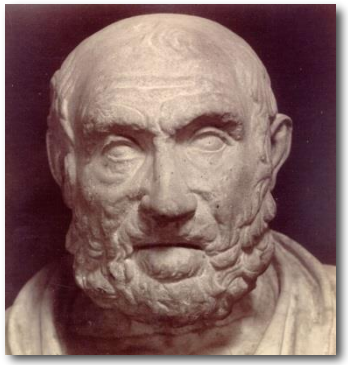
Figure 3. **(A)** Postnatal hippocampal neurogenesis was evaluated by quantifying 5-bromo-2′-deoxyuridine (BrdUrd)-positive cells in samples collected from the entire anterior–posterior extent of the hippocampal formation. Cell proliferation in the dentate gyrus was significantly less in monkeys from both early and late stress pregnancies. Results are the mean density ± SEM (number of BrdUrd-positive cells/1 mm^3 of the granule cell layer). **(B)** Hippocampal volume measurements. Postmortem hippocampal volumetry revealed that prenatal stress resulted in a significantly reduced hippocampal volume in both early (−12%) and late stress monkeys (−10%). Results are the mean volume (mm^3) ± SEM. For group comparisons, one-way analysis of variance was used, followed by Tukey's post hoc analysis. * $p < .05$, significantly different from control monkeys.

Pränatale Einflüsse: Barker-Hypothese

- **Barker-Hypothese**
- Evolutionäre Perspektive
- Psychobiologische Perspektive

Barker-Hypothese: „fetal origins of adult disease“

- Bedeutung des mütterlichen Wohlbefindens während der Schwangerschaft



Hippokrates
(~460 – 375 v. Chr.)

Text der Mahabharata
(Indien, ~ 1050 v. Chr.)

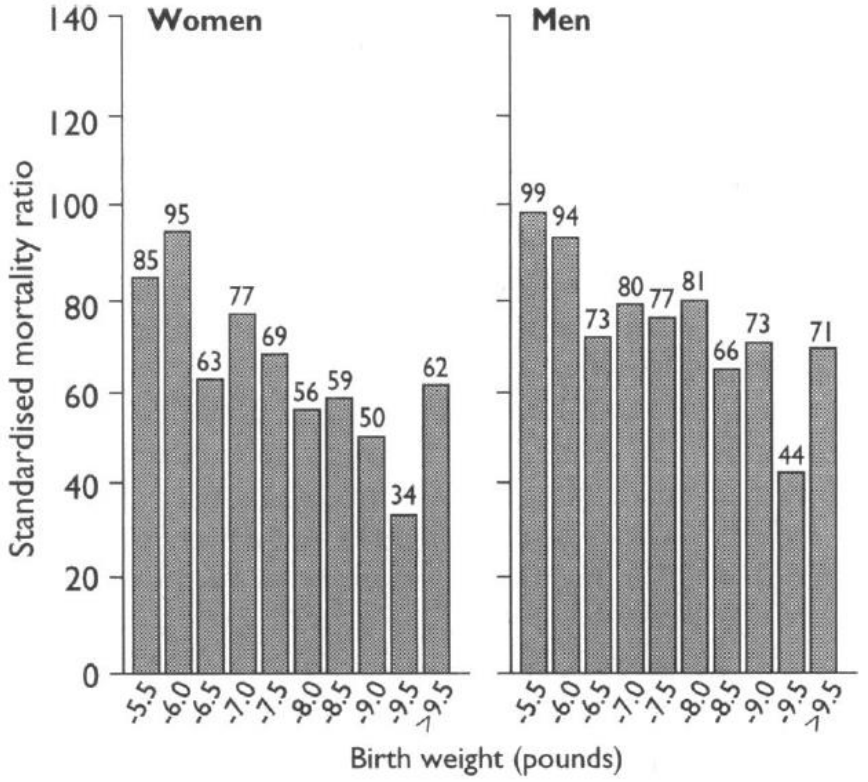
- 1950er bis 1970er Jahre: starke Betonung der vermeintlich anhaltenden Effekte früher aversiver Erfahrungen
  → keine empirischen Belege
  → Konzept aus der Mode… bis 1990er Jahre

Barker-Hypothese: epidemiologische Studien (Osmond et al., 1993)

Zusammenhang von Mortalitätsrate (koronare Herzerkrankung) und Geburtsgewicht:

- N = 15.726 (m & w)
- *1911-1930 in Hertfordshire
- niedriges Geburtsgewicht: < 2500g (5.5 lb)

"…these effects extend continuously across the normal range of distribution of birth weight"

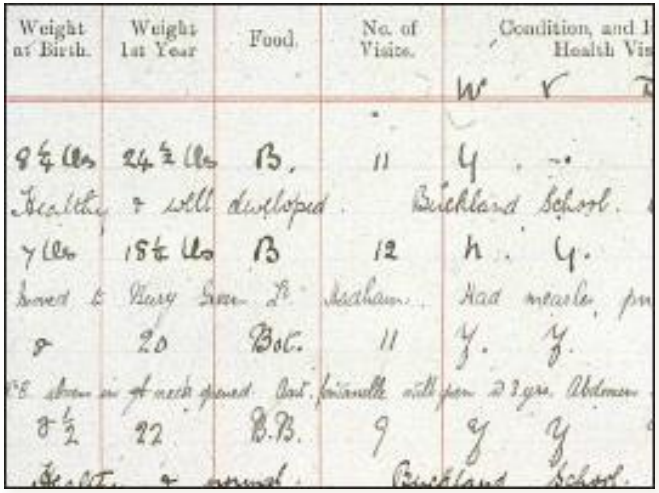


Barker-Hypothese: Ursprung

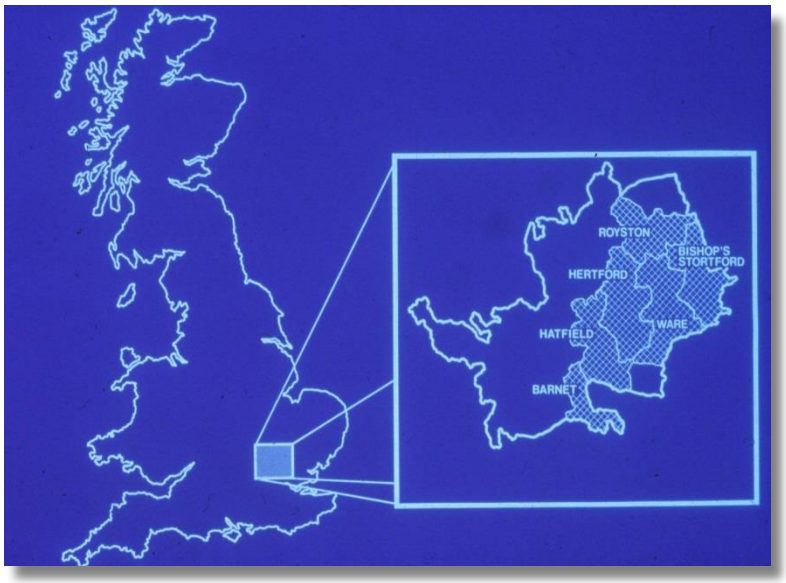
Ethel M. Burnside

„Chief Health Visitor and Lady Inspector of Midwives“

David Barker



Weight at Birth.	Weight 1st Year	Food.	No. of Visits.	Condition, and Health Vis
				W V
9¼ lbs	24½ lbs	B.	11	G . -
Healthy & well developed.				Buckland School.
7 lbs	18¼ lbs	B	12	h . G.
Sweet & Very Sma...				Had measles, pn
8	20	Bot.	11	Y. Y.
...e shown in ...				
8½	22	B.B.	9	Y Y
... & normal.				Buckland School.



Hertfordshire-Kohorte

Barker-Hypothese: Definition

„Fetal Origins of Adult Disease“
oder **„Barker-Hypothese“:**

‘changes in the fetal environment may result in developmental adaptations that permanently change structure, physiology, and metabolism, thereby predisposing individuals to chronic diseases such as coronary heart disease and type 2 diabetes’

Barker-Hypothese: BBC-Dokumentation – Video

Barker-Hypothese: Befunde

- „Developmental Origins of Health and Disease“ (DOHaD)

→ in mehreren epidemiologischen Studien weltweit bestätigt (N > 100.000 insgesamt)

→ Assoziation von **niedrigem Geburtsgewicht** & **kleiner Körpergröße** mit signifikant **erhöhtem Krankheitsrisiko** (und Krankheitsmarkern) im Erwachsenenalter für u.a.

→Hypertonie

→verändertem Glucose-Metabolismus, Typ-2-Diabetes

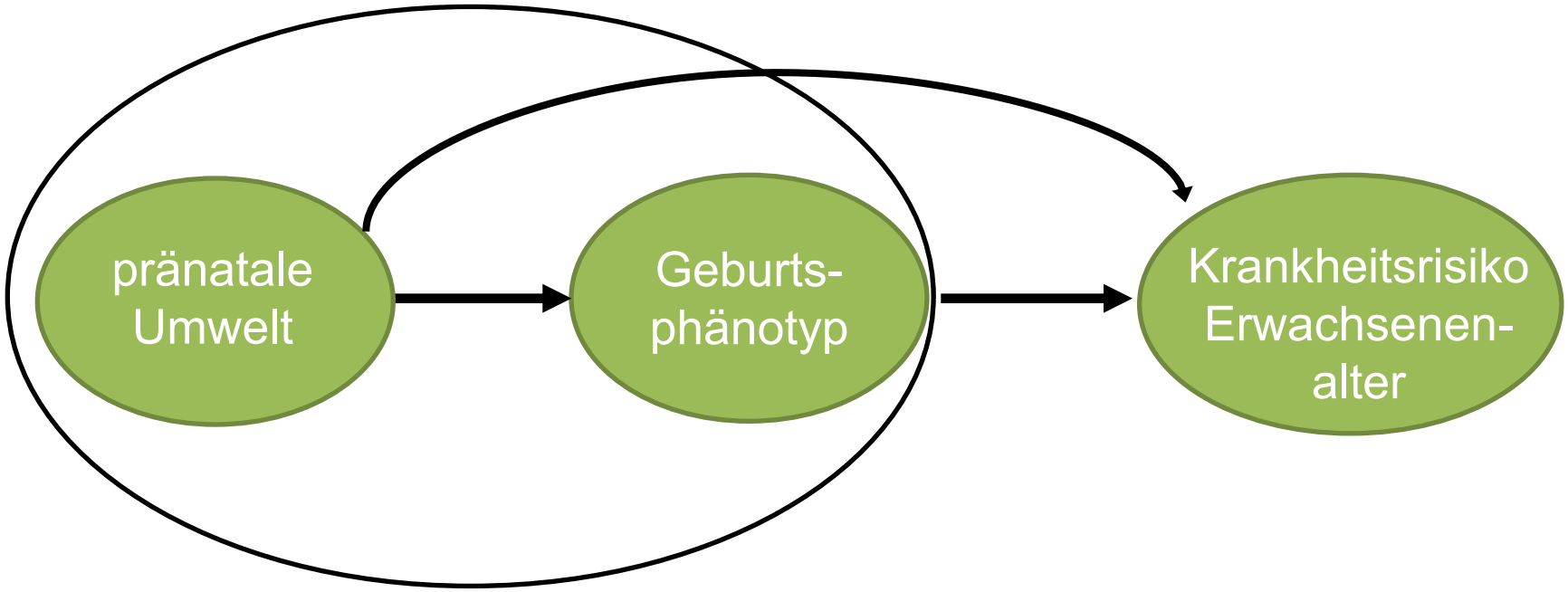
→Fettleibigkeit & erhöhte Blutfettwerte

→ koronare Herzkrankheit

Barker-Hypothese: Rolle des Geburtsphänotyps

- **Geburtsphänotyp**: Geburtsgewicht, Größe bei Geburt, Dauer der Schwangerschaft, Kopfumfang
- fetales Wachstum als Gesamtmarker für fetale Umwelt
  → reflektiert in relativ leicht zu erhebenden Größenmaßen bei Geburt wie Geburtsgewicht

Barker-Hypothese: Rolle des Geburtsphänotyps



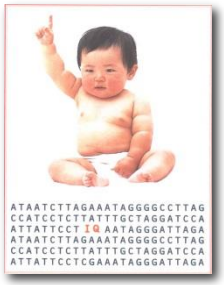
"The observed relationship between birth phenotype and disease risk does not imply a causal role of being born small but primarily reflects the sensitivity of fetal growth to adverse intrauterine influences"

Barker-Hypothese: Geburtsgewicht & Psychologie / Psychopathologie

- zunehmende Belege für Zusammenhang zwischen **fetalem Wachstum** und **behavioralen Outcomes** sowie **psychischer Gesundheit** im späteren Leben

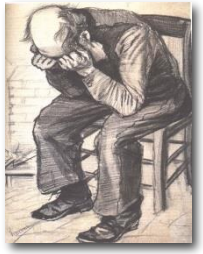
Geburtsgewicht…

… kognitive Funktionen



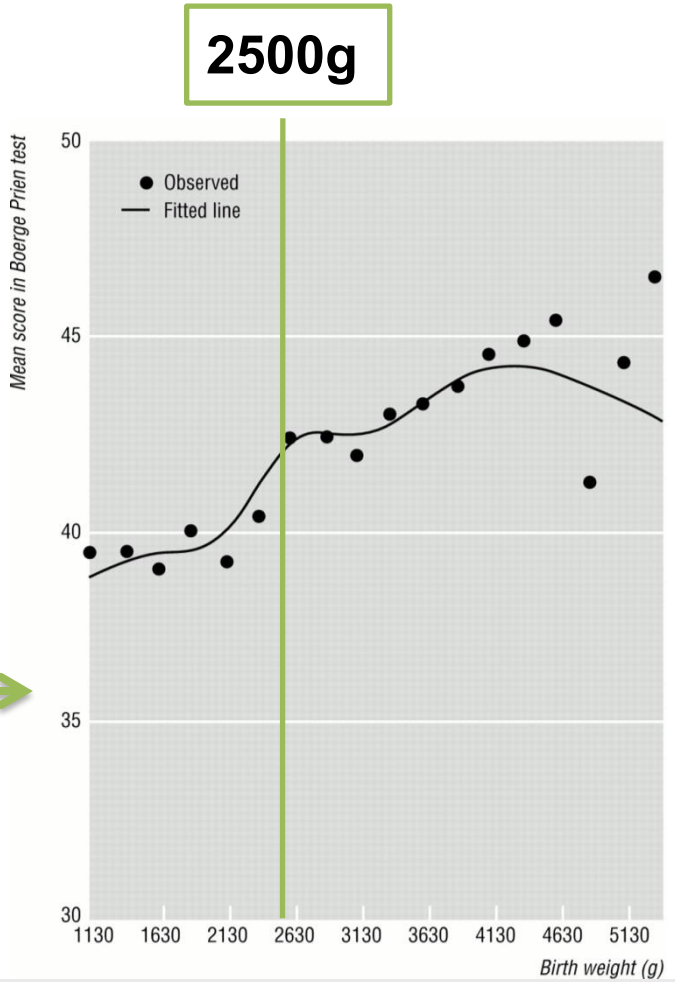
… Verhaltensauffälligkeiten (z.B. ADHS)

… Depression



Barker-Hypothese: Geburtsgewicht & kognitive Funktionen (Sorensen et al., 1997)

- niedrigerer IQ bei Kindern mit niedrigem Geburtsgewicht (< 15. Perzentil) vs. „normaler Range“ (3.8 – 15.2 Punkte, 10 Studien)
- **Zusammenhang über die gesamte Range von Geburtsgewichten hinweg?**
  → ja, aber eher kleiner Effekt
- Bsp.: Studie mit 4300 dänischen Wehrpflichtigen: Zunahme von Boerge Prien IQ-Test Scores mit erhöhtem Geburtsgewicht



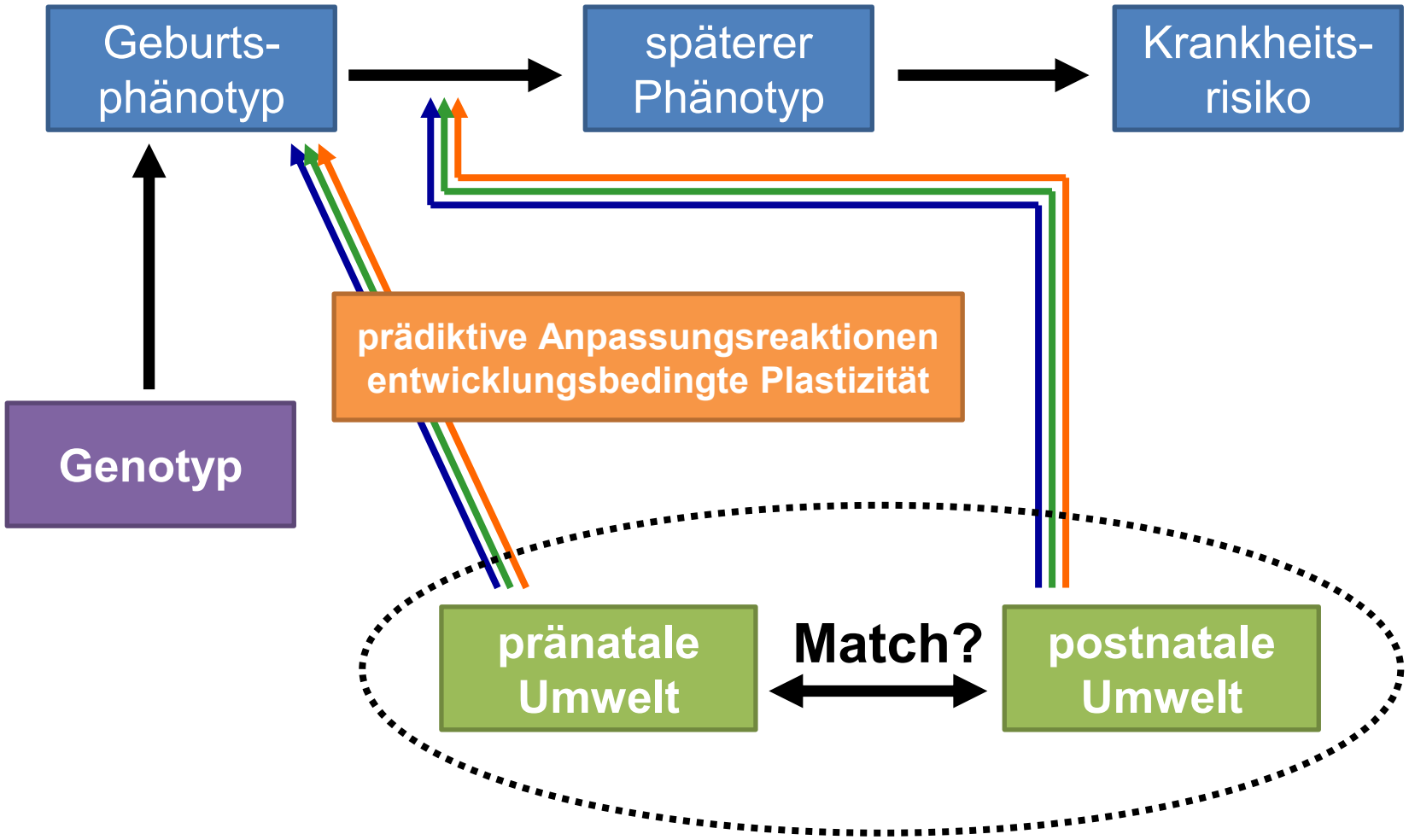
Barker-Hypothese: Geburtsgewicht & kognitive Funktionen (Sorensen et al., 1997)

- **andere Studien**: ähnliche Ergebnisse
  → Effekt unabhängig von sozialer Schicht & innerhalb gleichgeschlechtlicher Blutsverwandter beobachtet
- **aber**: Effekt von Geburtsgewicht deutlich geringer als von sozialer Schicht
- Bsp.: 1958 British Cohort Study (N = 10.845, 7-16 Jahre)
  → erklärte Varianz durch
  - Geburtsgewicht: 0.5–1.0%
  - soziale Schicht: 2.9–12.5% (Jefferis et al., 2002)

Pränatale Einflüsse: evolutionäre Perspektive

- Barker-Hypothese
- **Evolutionäre Perspektive**
- Psychobiologische Perspektive

Evolutionäre Perspektive: Modell (Gluckman & Hanson, 2004)



Evolutionäre Perspektive: pränataler Stress & Psychopathologie

THE JOURNAL OF CHILD PSYCHOLOGY AND PSYCHIATRY
Journal of Child Psychology and Psychiatry 52:4 (2011), pp 356–367 doi:10.1111/j.1469-7610.2011.02371.x

Annual Research Review: Prenatal stress and the origins of psychopathology: an evolutionary perspective

Vivette Glover
Institute of Reproductive and Developmental Biology, Imperial College London, UK

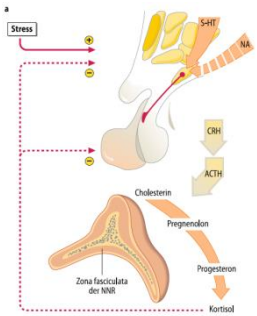
Table 1 Variety of effects of prenatal stress observed in children, and their possible evolutionary value

Effect	Reference	Evolutionary value
Increased anxiety	O'Connor, Heron, & Glover, 2002; Van Den Bergh & Marcoen, 2004; Bergman et al., 2007; de Bruijn et al., 2009	Greater vigilance and alertness to danger
Increased ADHD *More readily distracted attention* *Increased impulsivity*	O'Connor, Heron, Golding et al., 2002; Obel, Henriksen et al., 2003; Huizink et al., 2002; Rodriguez & Bohlin, 2005; Van Den Bergh & Marcoen, 2004	Greater sensitivity to dangerous signals More readiness to explore and find new environments
Increased conduct disorder *Breaking rules* *Increased aggression*	O'Connor, Heron, & Glover, 2002; Van Den Bergh & Marcoen, 2004; de Bruijn et al., 2009; Rice et al., 2010; E. D. Barker & Maughan, 2009	More willingness to explore and find new environments More likely to fight off predators or other threats
Lower cognitive performance	Bergman et al., 2007; Laplante et al., 2008; Mennes et al., 2006; Huizink et al., 2003)	May be associated with more readily distracted attention Different ways of thinking which can be adaptive
More mixed handedness	Obel, Hedegaard et al., 2003; Glover et al., 2004; Rodriguez & Waldenstrom, 2008	Side effect of altered neurodevelopment linked with increased ADHD etc.
Altered function of the HPA axis	O'Connor et al., 2005; Yehuda et al., 2005; Gutteling et al., 2005; Huizink et al., 2008; Van den Bergh et al., 2008; Entringer, Kumsta et al., 2009	Altered basal diurnal output, and response to new stressor
Fewer male offspring	Peterka et al., 2004; Obel et al., 2007	Mother invests more in female offspring

Evolutionäre Perspektive: pränataler Stress & Psychopathologie

(Glover, 2011)

- ADHS
- Angststörungen / Ängstlichkeit
- Verhaltensstörungen
- kognitive Funktionen
- gemischte Händigkeit
- veränderte Funktion der HPA-Achse
- Reproduktionsverhalten & Geschlechterverhältnis

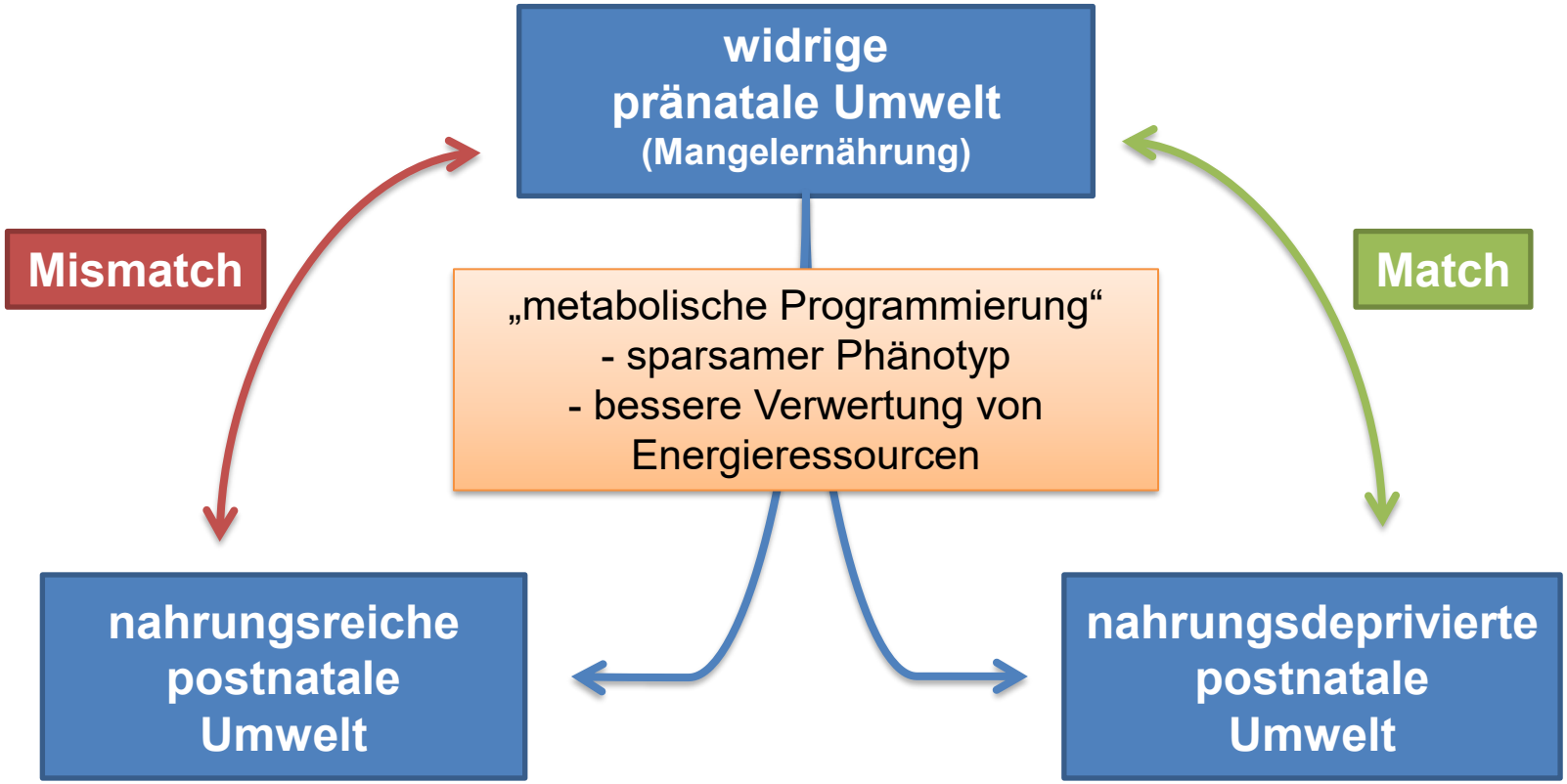


Key points

- Prenatal stress is associated with increased risk for a range of neurodevelopmental, behavioural and cognitive changes in the child. Not all children are affected in the same way and most are not affected at all.
- This may have been of adaptive value for our ancestors but lead to psychopathology in our modern society.
- Increased anxiety and distractible attention may have helped to increase vigilance and alertness to danger in a stressful environment, aggression to fighting predators, and impulsivity and breaking rules to seeking out new safer environments.
- This evolutionary perspective on the long-term effects of prenatal stress may stimulate new thinking for research and intervention.

Evolutionäre Perspektive: entwicklungsbedingte Plastizität

(Gluckman & Hanson, 2004)



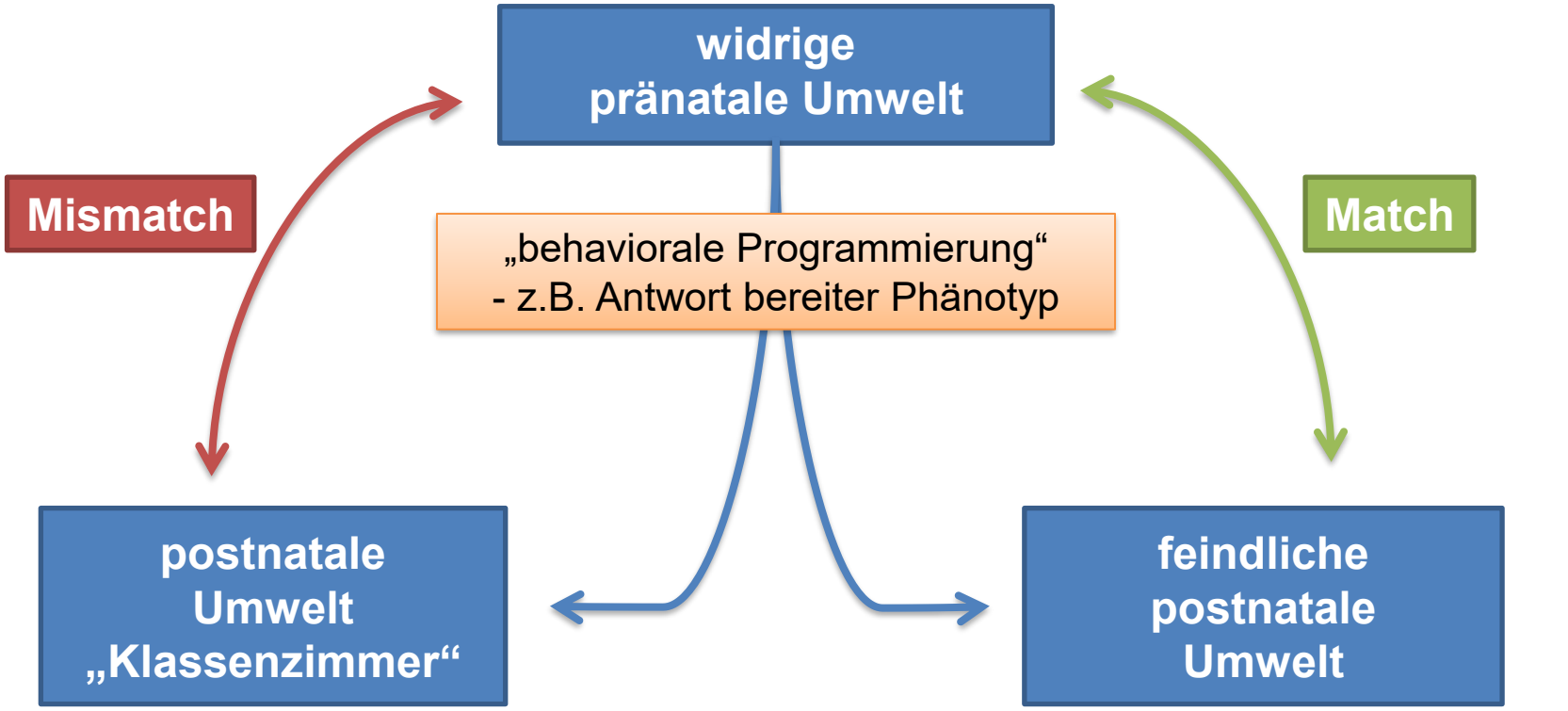
→ erhöhtes Risiko für Typ-2-Diabetes und metabolisches Syndrom
→ erhöhtes Risiko für kardiovaskuläre Erkrankungen

→ bessere Überlebenschancen

Evolutionäre Perspektive: entwicklungsbedingte Plastizität

(Gluckman & Hanson, 2004)

Spekulation: Relevanz des Modells für **Psycho**pathologie?



→ ADHS
→ Verhaltensstörung

→ bessere Überlebenschancen
→ z.B. Migration aus Afrika
(40.000 – 50.000 v. Chr.)

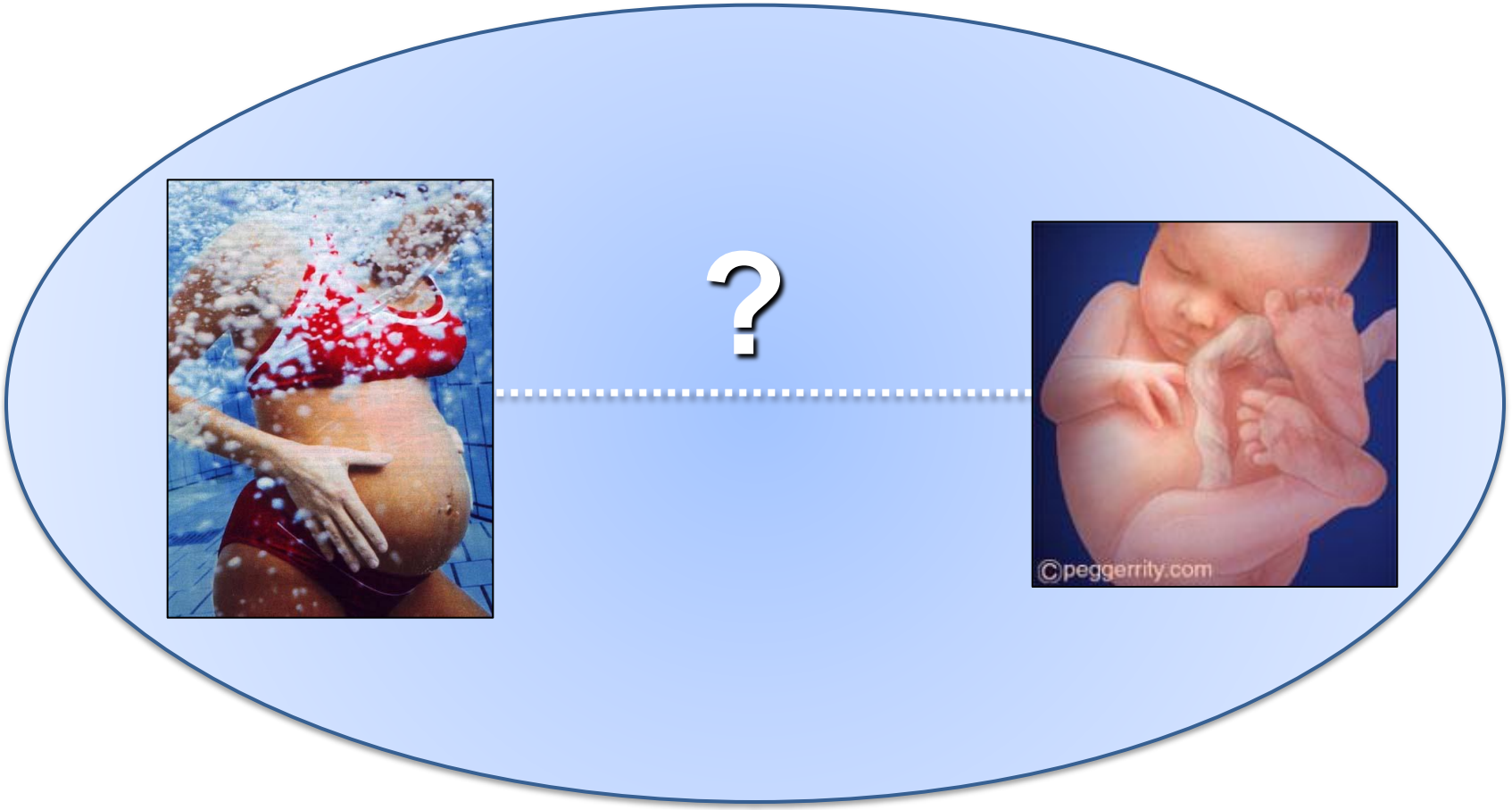
Pränatale Einflüsse: psychobiologische Perspektive

RUB

- Barker-Hypothese
- Evolutionäre Perspektive
- **Psychobiologische Perspektive**

Psychobiologische Perspektive: Rolle der Plazenta

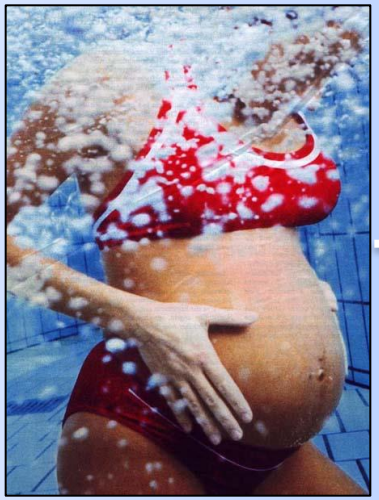
Mechanismus der Interaktion zwischen Mutter & Fötus

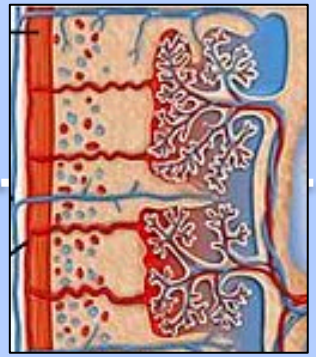


Psychobiologische Perspektive: Rolle der Plazenta

Aufnahme von Nährstoffen
Gasaustausch
Beseitigung von Abfallstoffen
→ keine Vermischung von mütterlichem & fetalem Blutfluss

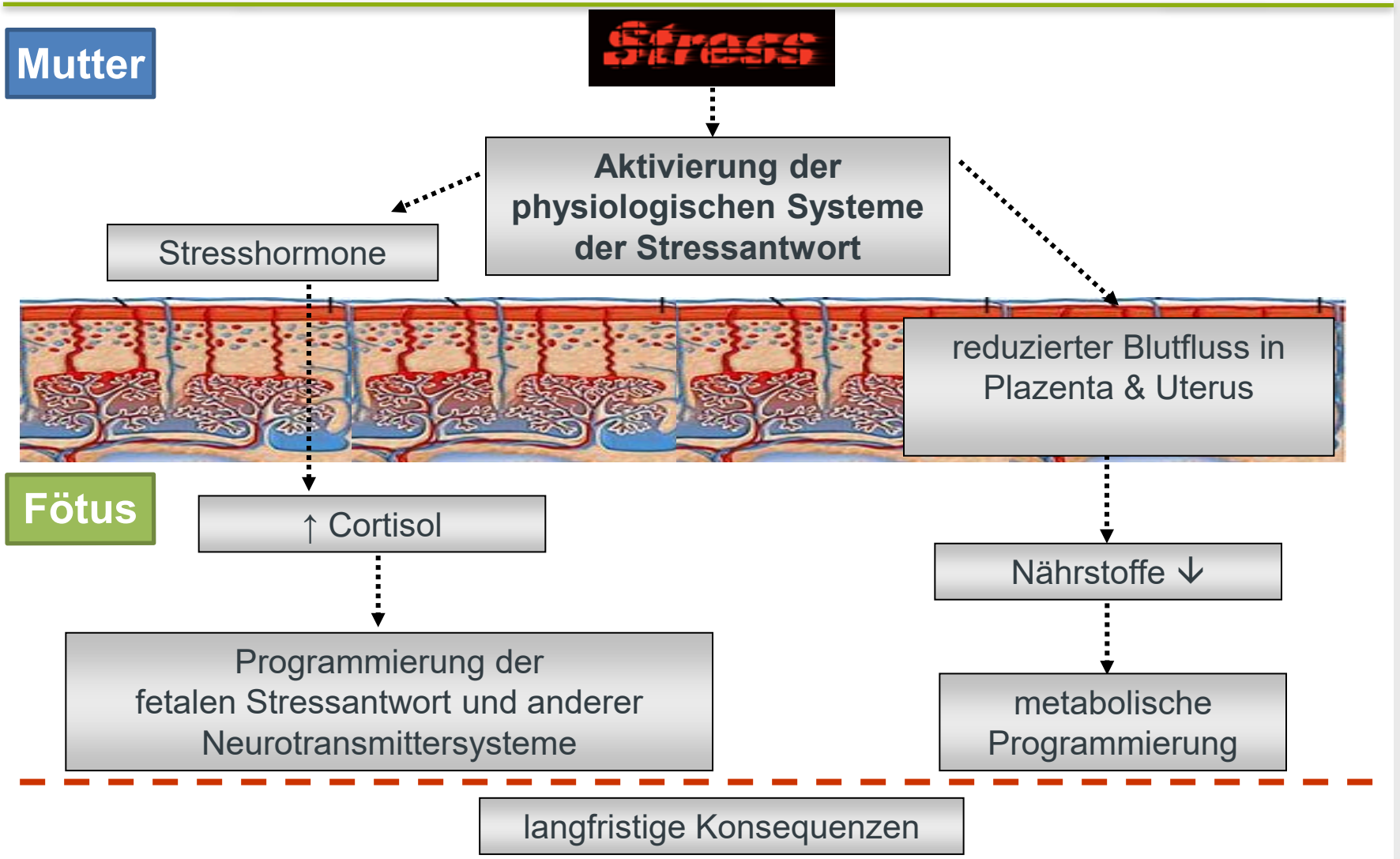
Plazenta





Plazentaschranke

Psychobiologische Perspektive: Rolle der Plazenta



Psychobiologische Perspektive: fetale Widrigkeiten & psychische Gesundheit

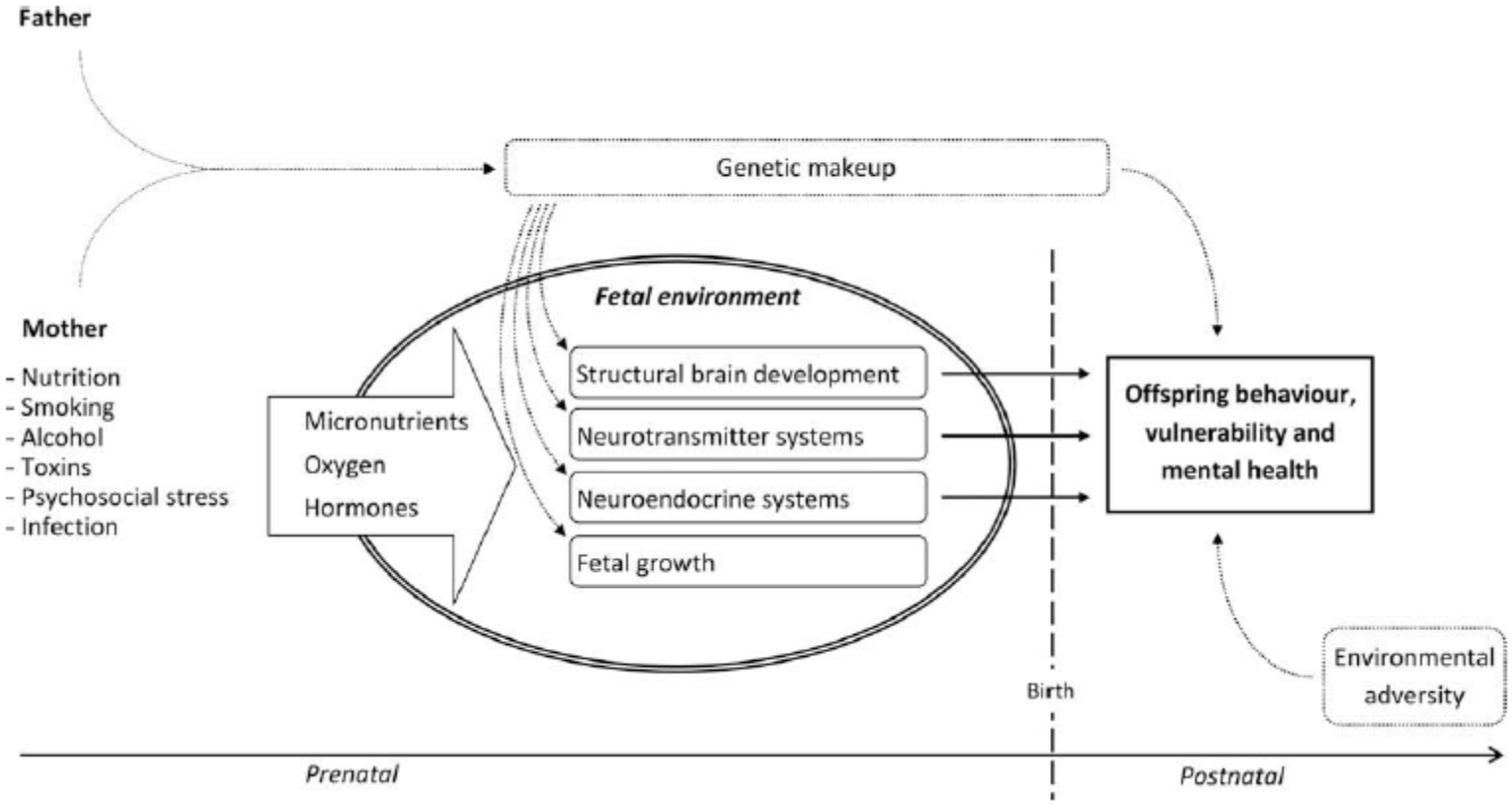


Fig. 1. A schematic summary of the principal factors, effects, pathways and confounders underlying the associations of fetal adversity with mental health. The principal pathway from specific maternal factors during pregnancy to increased risk of maladjusted behaviour, vulnerability, and mental health problems via fetal adversity is represented by solid boxes and arrows. Dashed boxes and arrows represent additional influences that act at different stages of the pathway.

Schlotz & Phillips (2009)

Psychobiologische Perspektive: Glucocorticoide & Plazenta (Reynolds, 2013)

- CRH-Produktion der Plazenta bei Primaten (nicht bei anderen Säugetieren)
- Stimulierung der CRH-Produktion in Plazenta durch Cortisol (im Gegensatz zu inhibitorischen Wirkungen auf hypothalamischer Ebene)
- positive Feedback-Schleife
- steigende Cortisol- & CRH-Werte im Verlauf der Schwangerschaft

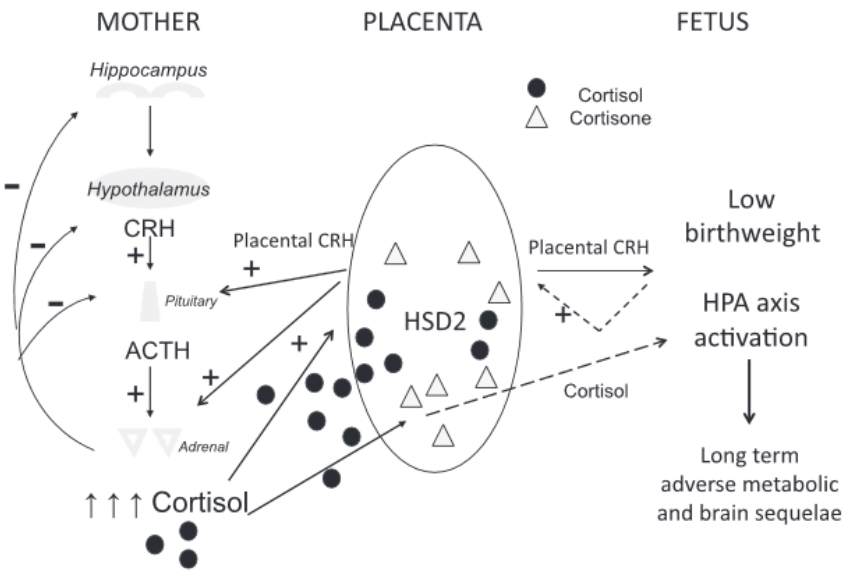


Fig. 1 Glucocorticoid signalling between mother, placenta and fetus. Figure shows interaction between maternal, placental and fetal compartments during pregnancy leading to overexposure of the developing fetus to glucocorticoids. Activation of the maternal HPA axis during pregnancy leads to increased circulating levels of cortisol (filled circles). Placental CRH also directly stimulates the maternal pituitary and adrenal to further increase cortisol levels, while maternal cortisol also stimulates placental CRH production. Maternal cortisol passes through the placenta where it is broken down by the enzyme HSD2 into inactive cortisone (grey triangles). The fetus can also signal to the placenta to increase production of placental CRH when fetal metabolic demands increase. Overexposure of the developing fetus to excess cortisol leads to fetal HPA axis activation which is associated with low birthweight and long term adverse programmed outcomes including metabolic and brain sequelae. CRH – corticotropin releasing hormone, ACTH – adrenocorticotropin hormone, HSD2 – 11β hydroxysteroid dehydrogenase type 2.

Psychobiologische Perspektive: Glucocorticoide & Plazenta (Wadhwa, 2005)

Interaktion der Plazenta mit HPA-Achse:

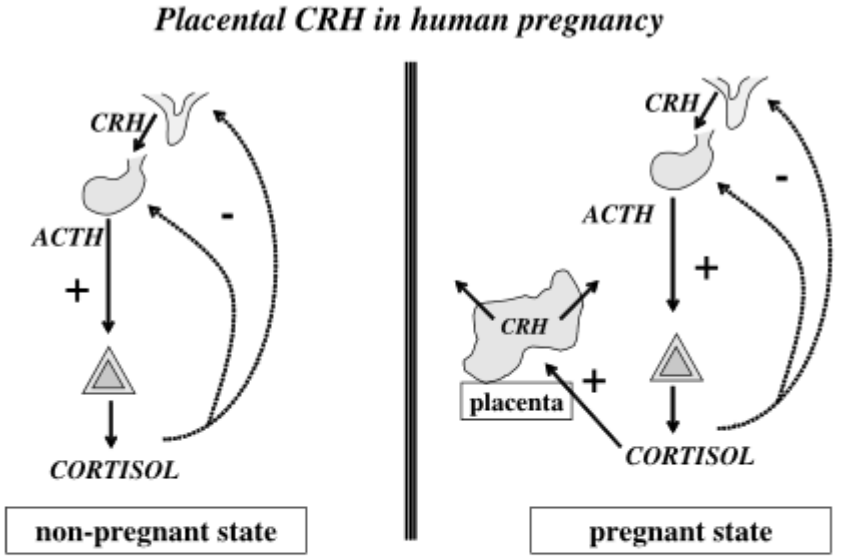


Figure 4 Placental CRH in human pregnancy. In pregnancy, the placenta is a major extra-hypothalamic site for CRH production and action. In contrast to the negative control exerted on the brain and pituitary, cortisol stimulates the production of CRH in the placenta, establishing a positive feedback loop that terminates upon delivery.

Psychobiologische Perspektive: pränataler Stress, Entwicklung & Krankheitsrisiko

Psychoneuroendocrinology 62 (2015) 366–375

Contents lists available at ScienceDirect

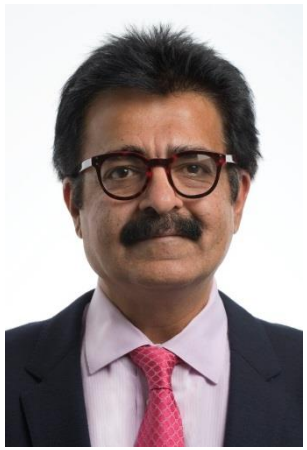
Psychoneuroendocrinology

journal homepage: www.elsevier.com/locate/psyneuen

Review

Prenatal stress, development, health and disease risk: A psychobiological perspective—2015 Curt Richter Award Paper

Sonja Entringer[a,b,*,1], Claudia Buss[a,b,*,1], Pathik D. Wadhwa[b,c,d,e]



Psychobiologische Perspektive: Geburtsgewicht & Cortisol

Psychoneuroendocrinology (2005) 30, 591–598

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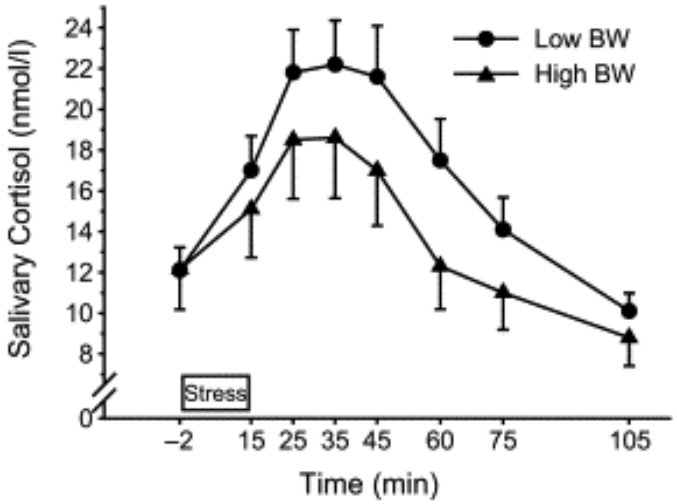
Birth weight is associated with salivary cortisol responses to psychosocial stress in adult life

Stefan Wüst*, Sonja Entringer, Ilona S. Federenko[1], Wolff Schlotz, Dirk H. Hellhammer

Department of Psychobiology, University of Trier, Johanniterufer 15, 54290 Trier, Germany



→ niedriges Geburtsgewicht mit höherer Cortisol-Stressreaktion assoziiert

Psychobiologische Perspektive: Geburtsgewicht & Cortisol

REVIEW

Glucocorticoid excess and the developmental origins of disease: Two decades of testing the hypothesis — 2012 Curt Richter Award Winner

Rebecca M. Reynolds *

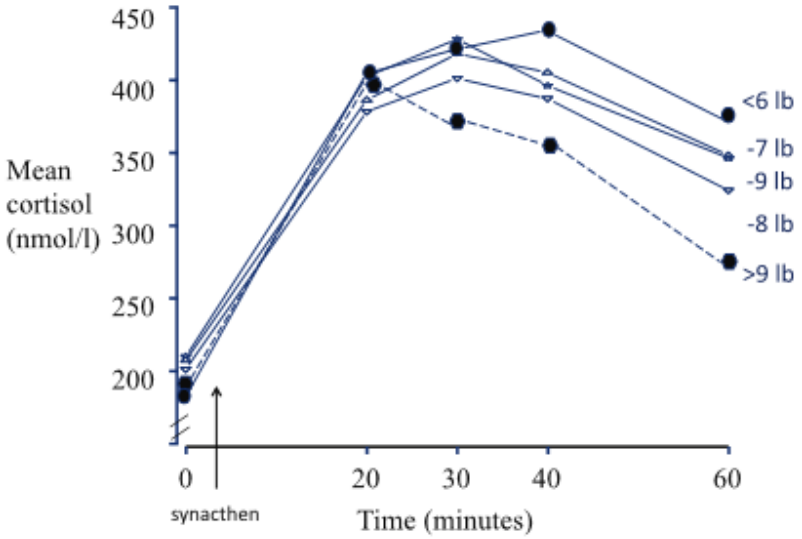


Fig. 2 Low birthweight is associated with activation of the hypothalamic–pituitary–adrenal axis but no differences in central negative feedback sensitivity. Figure shows cortisol levels following a low dose (0.25 mg) dexamethasone suppression test and a very low dose (1 μg) short synacthen test in 205 Hertfordshire men. Baseline (time 0 min) samples are those on the morning after the low dose dexamethasone suppression test. Synthetic ACTH was then administered with further blood sampling at 20, 30, 40 and 60 min. Cortisol levels are in nmol/l; to convert to μg/dl divide by 27.59. The baseline samples did not differ according to birthweight suggesting no changes in central negative feedback sensitivity of the hypothalamic–pituitary–adrenal (HPA) axis. Men of lowest birthweight (6.5 lb (2.92 kg) or less) had the highest peak cortisol response to synacthen and the greatest overall profile of response, consistent with activation of the HPA axis. $p = 0.03$ from longitudinal analysis, interaction of cortisol response by birth weight with time. Filled circles with solid lines represent birth weight of 6.5 lb (2.92 kg) or less and filled hexagons with dashed lines represent birth weight of >9.5 lb (4.31 kg).

→ niedriges Geburtsgewicht mit höherer Cortisol-Aufwachreaktion assoziiert

Psychobiologische Perspektive: Geburtsgewicht & Hippocampus

Brief Communications

Maternal Care Modulates the Relationship between Prenatal Risk and Hippocampal Volume in Women But Not in Men

Claudia Buss,[1,2] Catherine Lord,[2] Mehereen Wadiwalla,[2] Dirk H. Hellhammer,[1] Sonia J. Lupien,[2] Michael J. Meaney,[2] and Jens C. Pruessner[2]

[1]University of Trier, Department for Theoretical and Clinical Psychobiology, 54290 Trier, Germany, and [2]Douglas Hospital Research Center, Montreal, Quebec, Canada H4H 1R3

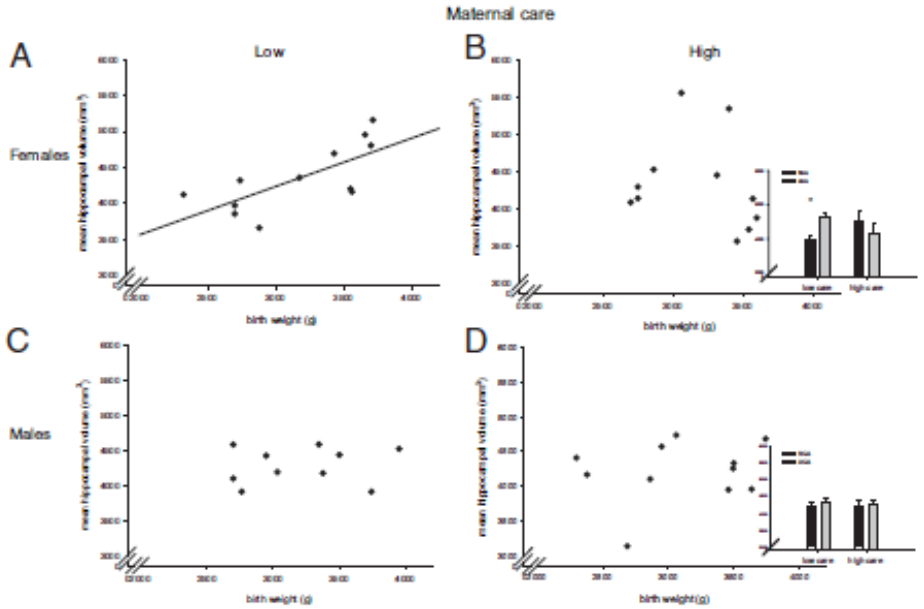


Figure 1. Association between birth weight and mean hippocampal volume. ***A***, ***C***, In females reporting low maternal care, there is a linear relationship between birth weight and hippocampal volume (***A***; $n = 12$; $r = 0.76$; $p = 0.01$), whereas no such correlation was present in the males reporting low maternal care (***C***; $n = 10$; $r = 0.26$; $p = 0.54$). ***B***, ***D***, In subjects reporting high maternal care, birth weight and hippocampal volume were not correlated in females (***B***; $n = 11$; $r = -0.36$; $p = 0.35$) or in males (***D***; $n = 11$; $r = 0.15$; $p = 0.69$). Error bars indicate SEM. $^*p < 0.05$.

→ niedriges Geburtsgewicht mit kleinerem Hippocampusvolumen assoziiert
(aber: nur bei Mädchen mit „low maternal care“)

Psychobiologische Perspektive: mütterliches Cortisol & Amygdala

Maternal cortisol over the course of pregnancy and subsequent child amygdala and hippocampus volumes and affective problems

Claudia Buss[a,1], Elysia Poggi Davis[a,b], Babak Shahbaba[c], Jens C. Pruessner[d], Kevin Head[b], and Curt A. Sandman[b]

Departments of [a]Pediatrics, [b]Psychiatry and Human Behavior, and [c]Statistics, University of California, Irvine, CA 92697; and [d]Centre for Studies in Aging, Departments of Psychology, Psychiatry, Neurology and Neurosurgery, McGill University, Montreal QC, Canada H4H 1R3

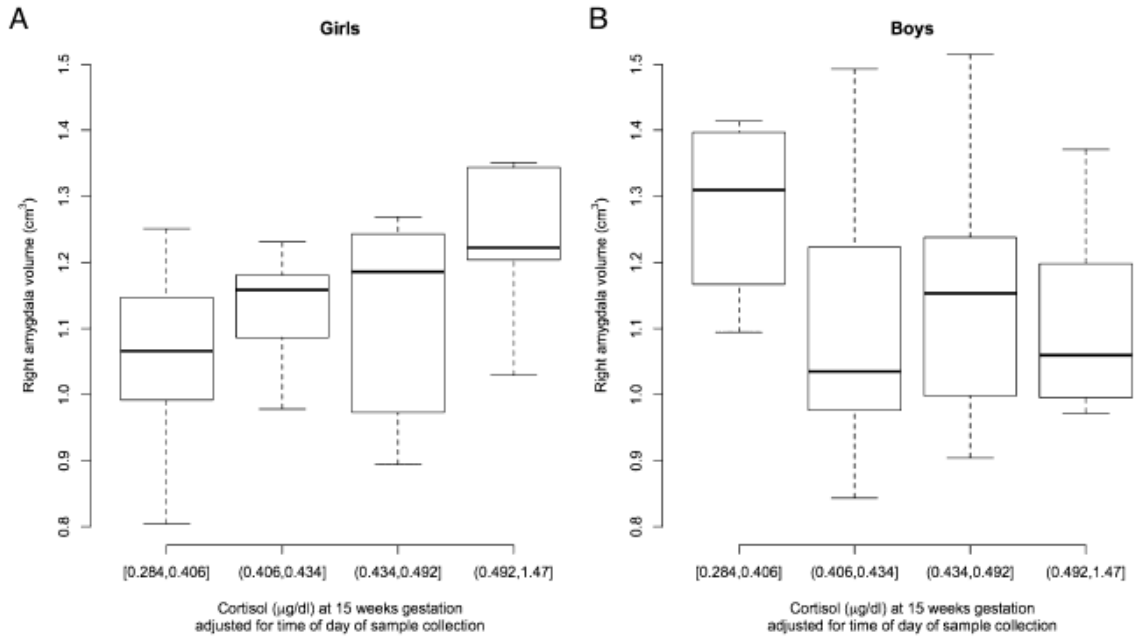


Fig. 1. Box plots show maternal cortisol concentrations at 15 wk gestation and children's right amygdala volumes at 7 y of age. For illustrative purposes, right amygdala volumes are depicted based on quartiles of mothers' cortisol concentrations (lowest and highest cortisol concentration for each of the four cortisol categories are provided). (*A*) In girls ($n = 35$), higher maternal cortisol concentrations at 15 wk gestation were associated with larger right amygdala volumes. (*B*) In boys ($n = 30$), there was no association between maternal cortisol concentrations at 15 wk gestation and right amygdala volumes.

→ höhere mütterliche Cortisolspiegel mit größerem Amygdala-Volumen assoziiert (bei Mädchen)

Psychobiologische Perspektive: mütterliches Cortisol & Amygdala

Maternal cortisol over the course of pregnancy and subsequent child amygdala and hippocampus volumes and affective problems

Claudia Buss[a,1], Elysia Poggi Davis[a,b], Babak Shahbaba[c], Jens C. Pruessner[d], Kevin Head[b], and Curt A. Sandman[b]

Departments of [a]Pediatrics, [b]Psychiatry and Human Behavior, and [c]Statistics, University of California, Irvine, CA 92697; and [d]Centre for Studies in Aging, Departments of Psychology, Psychiatry, Neurology and Neurosurgery, McGill University, Montreal QC, Canada H4H 1R3

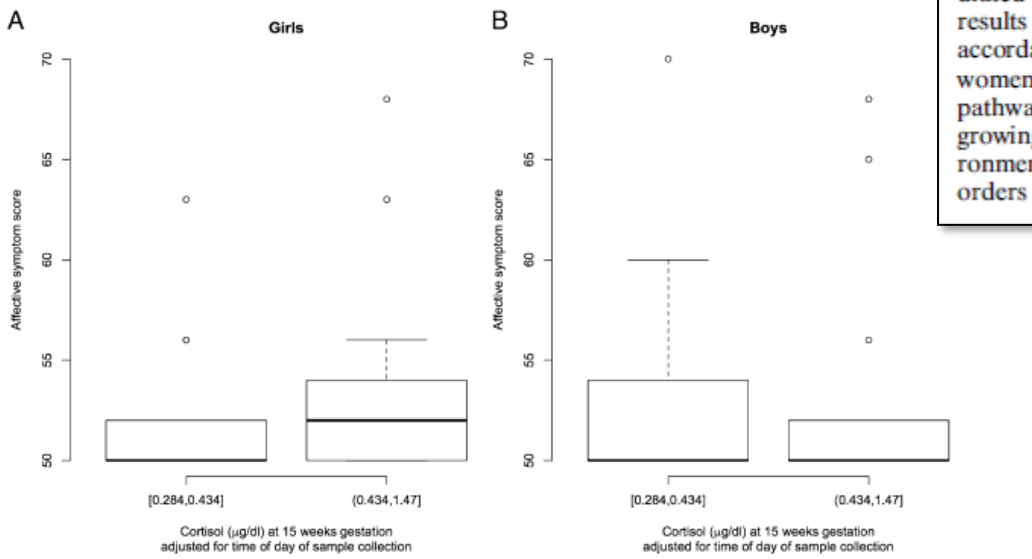


Fig. 2. Box plots show maternal cortisol concentrations at 15 wk gestation and children's scores on the affective problem scale of the CBCL at 7 y of age. For illustrative purposes, right amygdala volumes are depicted based on median of mothers' cortisol concentrations (lowest and highest cortisol concentration for each of the two cortisol categories are provided). (*A*) In girls ($n = 35$), higher maternal cortisol concentrations at 15 wk gestation were associated with more affective problems. (*B*) In boys ($n = 30$), there was no association between maternal cortisol concentrations at 15 wk gestation and affective problems.

In conclusion, findings from the present study provide support for the premise that susceptibility for affective disorders may, in part, be programmed in utero, and that this effect may be mediated through changes in anatomy of the amygdala. These results suggest potential clinical significance because they are in accordance with the higher prevalence of affective disorders in women than in men (81) and they point to a plausible etiological pathway. Thus, the results of the present study add to the growing awareness of the importance of the intrauterine environment and suggest the origins of neurodevelopmental disorders may have their foundations very early in life.

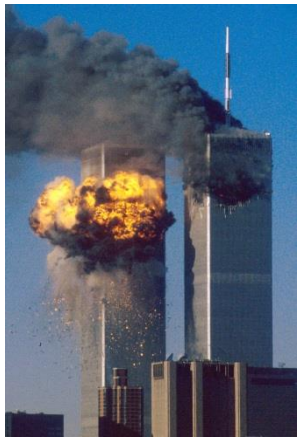
→ höhere mütterliche Cortisolspiegel mit gesteigerten affektiven Problemen assoziiert (bei Mädchen)

Psychobiologische Perspektive: „Natürliche Experimente“

Katastrophen wie…

- Erdbeben
- Blizzard
- 9/11

Einfluss auf Schwangerschaft & weitere Entwicklung des Kindes



Psychobiologische Perspektive: mütterlicher Stress & Krankheitsrisiko

Psychoneuroendocrinology 62 (2015) 366–375

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Review

Prenatal stress, development, health and disease risk: A psychobiological perspective—2015 Curt Richter Award Paper

Sonja Entringer[a,b,*,1], Claudia Buss[a,b,*,1], Pathik D. Wadhwa[b,c,d,e]

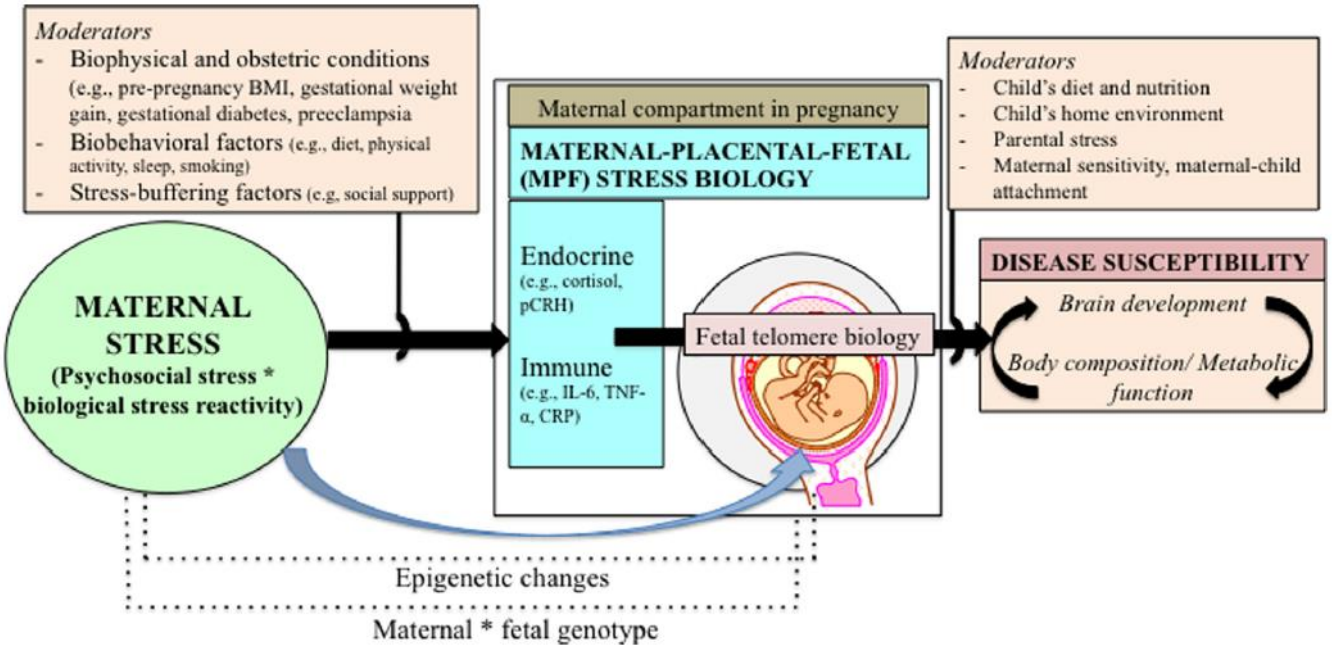


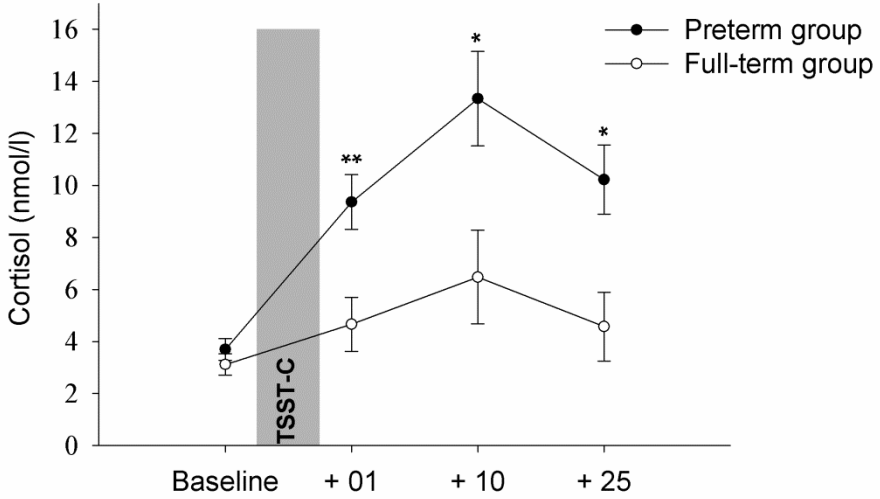
Fig. 1. Conceptual framework to study the effects of maternal stress during pregnancy on disease susceptibility.

Psychobiologische Perspektive: Vererbbarkeit von Traumata – Video

Psychobiologische Perspektive: Frühgeburt & Stressreaktivität

(Quesada et al., 2014)

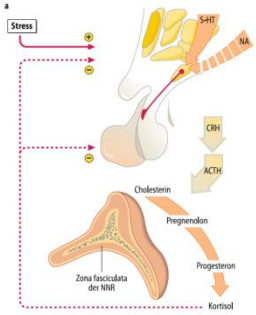
→ erhöhte Stressreaktivität bei Frühgeborenen



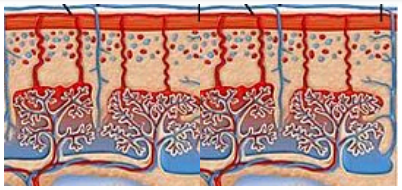
Zusammenfassung

- **Barker-Hypothese**:
  - fetale Umwelt → Risiko für Krankheiten
  - Geburtsphänotyp als Marker

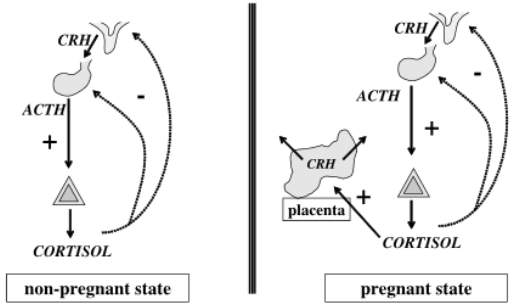
- **Evolutionäre Perspektive**:
  - Plastizität → Vorbereitung auf Umwelt
  - Match / Mismatch von prä-/postnataler Umwelt



Zusammenfassung



- **Psychobiologische Perspektive**:
  - Diffusion von **mütterlichem Cortisol** durch **Plazenta**:
    - positive Feedback-Schleife → ↑ CRH & ↑ Cortisol
    - ↑ Amygdala-Volumen & affektive Probleme bei Mädchen
  - Assoziation von **niedrigem Geburtsgewicht** mit…
    - ↑ Cortisol bei späterem psychosozialem Stress
    - ↑ Cortisol-Aufwachreaktion
    - teilweise kleineres Hippocampusvolumen bei Mädchen



Terminübersicht

20.10.25	Übersicht und Einführung
27.10.25	Stress und das SNS: Walter Cannon (Dr. Katja Langer)
03.11.25	Stress und die HHNA: Hans Selye
10.11.25	Stress und die HHNA: Munck und Sapolsky Die kognitive Wende: Lazarus
17.11.25	Stress und Gesundheit: McEwen und die allostatische Belastung Stress im Arbeitsleben: Siegrist und die Effort Reward Imbalance
24.11.25	Burnout (Dipl. Psych. Natalie Freund)
01.12.25	Soziale Evaluation als bedeutsamer Stressor: Dickerson & Kemeny (Dr. Katja Langer)
08.12.25	Soziale Unterstützung als Stresspuffer/Oxytozin
15.12.25	Stress und Gehirn: akute und chronische Effekte
12.01.26	Pränataler Stress und seine Folgen
(Aufzeichnung)	Frühkindlicher Stress und seine Folgen (Prof. Robert Kumsta)
19.01.26	Posttraumatische Belastungsstörung