WikipediA

Thrifty phenotype

The **thrifty phenotype** hypothesis says that reduced <u>fetal growth</u> is strongly associated with a number of chronic conditions later in life, including <u>coronary heart disease</u>, <u>stroke</u>, <u>diabetes</u>, and <u>hypertension</u>. This increased susceptibility is said to result from adaptations made by the fetus in an environment limited in its supply of nutrients. The thrifty phenotype is a component of the fetal origins hypothesis.

Contents
Evolutionary rationale
Benefit for offspring
Benefit for mother
Adverse effects
Molecular mechanisms
See also
References
External links

Evolutionary rationale

Benefit for offspring

Proponents of this idea say that in poor nutritional conditions, a pregnant woman can modify the development of her unborn child such that it will be prepared for survival in an environment in which resources are likely to be short, resulting in a thrifty phenotype (Hales & Barker, $1992^{[1][2]}$). It is sometimes called **Barker's hypothesis**, after Professor David J. P. Barker, researching at the University of Southampton who published the theory in 1990.^[3]

The thrifty phenotype hypothesis suggests that early-life <u>metabolic</u> adaptations help in survival of the organism by selecting an appropriate trajectory of growth in response to environmental cues. Recently, some scientists have proposed that the thrifty phenotype prepares the organism for its likely adult environment in long term.

Benefit for mother

However, environmental changes during early development may result in the selected trajectory becoming inappropriate, resulting in adverse effects on health. This paradox generates doubts about whether the thrifty phenotype is adaptive for human offspring. Thus, the thrifty phenotype should be considered as the capacity of all offspring to respond to environmental cues during early <u>ontogenetic</u> development. It has been suggested that the thrifty phenotype is the consequence of three unlike adaptive processes: maternal effects, niche construction and developmental plasticity, which all are influenced by the brain. While developmental plasticity demonstrates an adaptation by the offspring, <u>niche</u> construction and parental

effects are result of parental selections rather than offspring fitness. Therefore, the thrifty phenotype can be described as a manipulation of offspring phenotype for the benefit of maternal fitness. The information that enters <u>offspring</u> phenotype during early development mirror the mother's own developmental experience and the quality of the environment during her own maturation rather than predicting the possible future environment of the offspring^[4]

Adverse effects

Many human diseases in adulthood are related to growth patterns during early life, determining early-life nutrition as the underlying mechanism. Individuals with a thrifty phenotype will have "a smaller body size, a lowered metabolic rate and a reduced level of behavioural activity... adaptations to an environment that is chronically short of food" (Bateson & Martin, $1999^{[5]}$). Those with a thrifty phenotype who actually develop in an affluent environment may be more prone to metabolic disorders, such as obesity and <u>type II</u> diabetes, whereas those who have received a positive maternal forecast will be adapted to good conditions and therefore better able to cope with rich diets. This idea (Barker, $1992^{[6]}$) is now widely (if not universally) accepted and is a source of concern for societies undergoing a transition from sparse to better nutrition (Robinson, $2001^{[7]}$).

Risk factors of thrifty phenotype include <u>advanced maternal age</u> and placental insufficiency.^[8]

Molecular mechanisms

The ability to conserve, acquire and expend energy is believed to be an innate, ancient trait that is imbedded in the genome in a way that is quite protected against <u>mutations</u>.^[9] These changes are also believed to possibly be inherited across generations.^[9] Leptin has been identified as a possible gene for the acquisition of these thrifty traits.^[9]

On a larger anatomic scale, the molecular mechanisms are broadly caused by a suboptimal environment in the reproductive tract or maternal physiological adaptations to pregnancy.^[8]

See also

- Evolutionary developmental psychology
- Evolutionary physiology
- Phenotypic plasticity
- Trivers—Willard hypothesis
- Thrifty gene hypothesis
- Prenatal nutrition and birth weight

References

- 1. Hales CN, Barker DJ (July 1992). "Type 2 (non-insulin-dependent) diabetes mellitus: the thrifty phenotype hypothesis" (https://doi.org/10.1007%2FBF00400248). *Diabetologia*. **35** (7): 595–601. doi:10.1007/BF00400248 (https://doi.org/10.1007%2FBF00400248). PMID 1644236 (https://pubmed.ncbi.nlm.nih.gov/1644236).
- Hales, CN; Barker, DJ (2001). "The thrifty phenotype hypothesis" (https://doi.org/10.1093%2 <u>Fbmb%2F60.1.5</u>). British Medical Bulletin. 60: 5–20. doi:10.1093/bmb/60.1.5 (https://doi.org/ 10.1093%2Fbmb%2F60.1.5). PMID 11809615 (https://pubmed.ncbi.nlm.nih.gov/11809615).

- 3. Barker, D.J.P. (1997). "Maternal Nutrition, Fetal Nutrition, and Disease in Later Life". *Nutrition*, '**13**', pg. 807
- 4. Wells JC (February 2007). "The thrifty phenotype as an adaptive maternal effect". *Biol Rev Camb Philos Soc.* 82 (1): 143–72. doi:10.1111/j.1469-185X.2006.00007.x (https://doi.org/10. 1111%2Fj.1469-185X.2006.00007.x). PMID 17313527 (https://pubmed.ncbi.nlm.nih.gov/173 13527).
- 5. Martin, Paul; Bateson, Patrick (1999). *Design for a life: How behaviour develops*. London: Jonathan Cape. pp. 110–1. ISBN 0-224-05064-8.
- 6. Barker, D. J. P., ed. (1992). *Fetal and infant origins of adult disease*. London: British Medical Journal. ISBN 0-7279-0743-3.
- 7. Robinson R (February 2001). "The fetal origins of adult disease : No longer just a hypothesis and may be critically important in south Asia" (http://bmj.bmjjournals.com/cgi/cont ent/full/322/7283/375). BMJ. 322 (7283): 375–6. doi:10.1136/bmj.322.7283.375 (https://doi.or g/10.1136%2Fbmj.322.7283.375). PMC 1119617 (https://www.ncbi.nlm.nih.gov/pmc/articles/ PMC1119617). PMID 11179140 (https://pubmed.ncbi.nlm.nih.gov/11179140). Editorial
- Aiken, C. E.; Ozanne, S. E. (2013). "Transgenerational developmental programming" (https:// doi.org/10.1093%2Fhumupd%2Fdmt043). Human Reproduction Update. 20 (1): 63–75. doi:10.1093/humupd/dmt043 (https://doi.org/10.1093%2Fhumupd%2Fdmt043). PMID 24082037 (https://pubmed.ncbi.nlm.nih.gov/24082037).
- 9. Stöger R (February 2008). "The thrifty epigenotype: an acquired and heritable predisposition for obesity and diabetes?". *BioEssays*. **30** (2): 156–66. <u>doi:10.1002/bies.20700 (https://doi.or</u>g/10.1002%2Fbies.20700). PMID 18197594 (https://pubmed.ncbi.nlm.nih.gov/18197594).

External links

 British Medical Journal Topic Collections: Barker Hypothesis (http://bmj.bmjjournals.com/cgi/ collection/barker_hypothesis)

Retrieved from "https://en.wikipedia.org/w/index.php?title=Thrifty_phenotype&oldid=1066529514"

This page was last edited on 18 January 2022, at 20:20 (UTC).

Text is available under the Creative Commons Attribution-ShareAlike License 3.0; additional terms may apply. By using this site, you agree to the Terms of Use and Privacy Policy. Wikipedia® is a registered trademark of the Wikimedia Foundation, Inc., a non-profit organization.