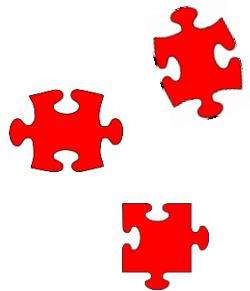




## Multimorbiditet i primærhelsetjensten

Linn Getz



Linn Getz, lege og professor i medisinske atferdsfag  
Inst. for samfunnsmedisin og sykepleie, NTNU  
Allmennmedisinsk forskningsenhet (AFE)  
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**ISM**  
Allmennmedisinsk  
forskningsenhet

St Olav og NTNU medisin-helse

## Faglig tilnærming?



## Hermann (34)

Psykiatrisk sykepleier,  
1 + 1 barn  
Separert 2014 og 2017

Diabetes Type 1  
Psoriasis  
Recidiverende halsbetennelser  
Refluks-øsofagitt (GERD)  
Astma  
IBD (Irritabel tarm)  
Depressive episoder  
Søvnproblemer  
**Korsryggsmerter;**  
«Forlatt» av fysioterapeuten

Fotoet er arrangert

## Faglig tilnærming?

**Evidens-basert?  
Bio-psyko-sosial?  
Pasient-sentrert?  
Problem orientert?**

**...Person-sentrert?  
«...Personalized»?**

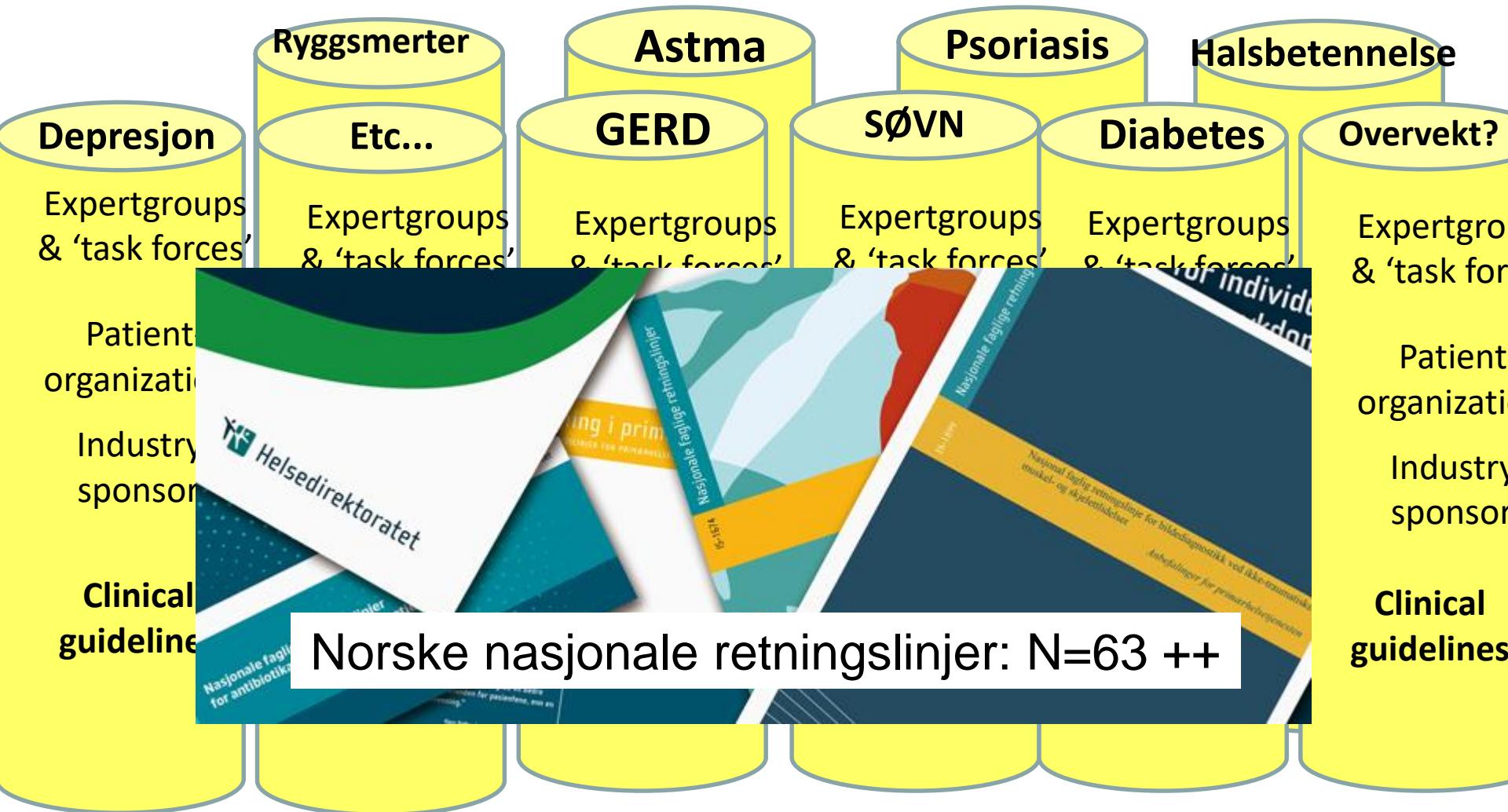
**Narrativ medisin?**

## Hermann (34)

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Søvnproblemer  
**Korsryggsmerter;**  
«Forlatt» av fysioterapeuten

# „SILO-MEDISIN“





«Vi kan ikke løse problemene  
med de samme tankene vi  
brukte da vi skapte dem»

- Albert Einstein



Kunnskap for en bedre verden

[Startseite](#)[Studier](#)[Student i Trondheim](#)[Forskning](#)

Studier » Utdanningsområder » Medisin, helse- og sosialfag » Medisinstudiet (CMED)

- [Studiets startside](#)
- [Om studieprogrammet](#)
- [Opptak](#)
- [Jobbmuligheter](#)
- [Studiets oppbygging](#)
- [Studieveileddning](#)
- [Studiemiljø](#)
- [Utenlandsoppbak](#)

Profesjonsstudium

## Medisin

For å bli lege må du kjenne kroppsmaskineriet helt inn til den minste celle. Du må utvikle eyno til kontinuerlig oppdatering i et fagfelt med rivende kunnskapsutvikling.

## Nobelprisen 2014 «Hjernens GPS»

[Ta del av studieprogrammet](#)

Gradsnivå: Profesjonsstudium

Adgangsbegrensning: Ja

Karakterkrav i 2013: Ordinær: 66,7

Førstegangsvitnemål: 60,0

Opptak: [Samordna opptak](#)

Søknadsfrist: 15. april/1. mars

Fakultet: Det medisinske fakultet

Informasjonens gyldighet: Studieåret 2013/2014

**“For å bli lege, må du kjenne kroppsmaskineriet helt inn til den minste celle”**

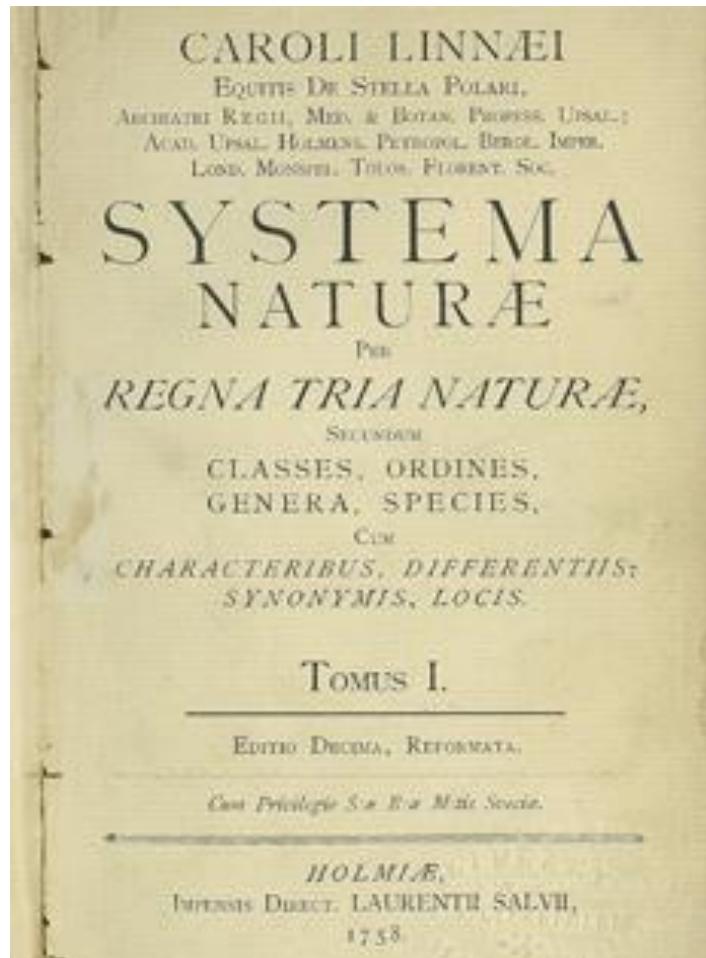


Medisin er studiet av den menneskelige organisme, fra den minste celle til større organer. Du vil lære om hvordan kroppen fungerer optimalt og hva som skjer ved sykdom.

Medisinsk forskning fører til nye spennende behandlingsteknikker. Vi får stadig ny kunnskap om sykdomsprosesser.

Som medisinstudent ved NTNU får du mulighet til å studere ved et nytt og moderne universitetssykehus. Det medisinske fakultet er samlokalisert med St. Olavs hospital. Studentene har tilgang til moderne og funksjonelle arealer til forelesninger, gruppearbeid og individuell lesing. Høsten 2013 tok vi i bruk det nye kunnskapsenter for medisinsk forskning og undervisning. Bygget inneholder og et nytt stort [medisinsk bibliotek](#).

# HOMO SAPIENS: Nosce te ipsum (**Know yourself**)



ANTROPOMORPHA. Dentes primores utrinque  
quatuor, aut nulli.

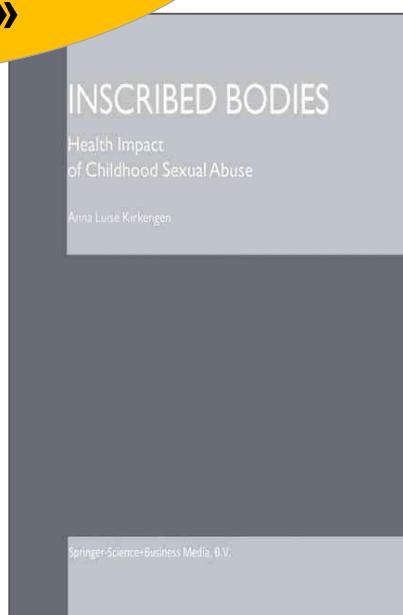
1. *HOMO. Nosce te ipsum.*

*Homo* variat: *Europaeus albus* l'HOMME.  
*Americanus rubescens.*  
*Asiacus fuscus.*  
*Africanus niger.*

2. *SIMIA. Os dentatum. Pedes pentadactyli scandentes;*  
*cum pollice.*  
*Mamma pectorales.*

*Simia* mammis quaternis, capite ad aures crinito. LE SINGE.  
*Animal cynocephalum*, tardigradum dictum, *Simii species. Seb. 1. p.*  
*55. t. 35. f. 1. 2.*  
*Simiarum species descripte non sunt, nec earum differentiae verò de-*  
*tectae. E. gr. *Papio*, *Satyrus*, *Cercopithecus*, *Cynocephalus*.*

**«Vi behøver  
medisinsk  
kunnskap som yter  
menneskenaturen  
rettferdighet»**





## Medicine's perception of reality – a split picture: critical reflections on apparent anomalies within the biomedical theory of science

Anna Luise Kirkengen MD PhD,<sup>1,2</sup> Tor-Johan Ekeland PhD,<sup>3</sup> Linn Getz MD PhD,<sup>1</sup> Irene Hetlevik MD PhD,<sup>1</sup> Edvin Schei MD PhD,<sup>2,4</sup> Elling Ulvestad MD PhD<sup>5,6</sup> and Arne Johan Vetlesen PhD<sup>7</sup>

<sup>1</sup>Professor, General Practice Research Unit, Department of Public Health and General Practice, Norwegian University of Science and Technology, Trondheim, Norway

<sup>2</sup>Professor, Department of Community Medicine, The Arctic University Tromsø, Tromsø, Norway

<sup>3</sup>Professor, Faculty of Social Science and History, Volda University College, Volda, Norway

<sup>4</sup>Professor, Department of Community Medicine, University of Bergen, Bergen, Norway

<sup>5</sup>Professor, Department of Microbiology, Haukeland University Hospital, Bergen, Norway

<sup>6</sup>Professor, Department of Clinical Science, University of Bergen, Bergen, Norway

<sup>7</sup>Professor, Department of Philosophy, University of Oslo, Oslo, Norway

### Keywords

dichotomies, general practice, lived body, medical anomalies, phenomenology

### Correspondence

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Norway  
E-mail: anlui-k@online.no

Accepted for publication: 15 March 2015

doi:10.1111/jep.12369

### Abstract

Escalating costs, increasing multi-morbidity, medically unexplained health problems, complex risk, poly-pharmacy and antibiotic resistance can be regarded as artefacts of the traditional knowledge production in Western medicine, arising from its particular worldview. Our paper presents a historically grounded critical analysis of this view. The materialistic shift of Enlightenment philosophy, separating subjectivity from bodily matter, became normative for modern medicine and yielded astonishing results. The traditional dichotomies of mind/body and subjective/objective are, however, incompatible with modern biological theory. Medical knowledge ignores central tenets of human existence, notably the physiological impact of subjective experience, relationships, history and socio-cultural contexts. Biomedicine will not succeed in resolving today's poorly understood health problems by doing 'more of the same'. We must acknowledge that health, sickness and bodily functioning are interwoven with human meaning-production, fundamentally personal and biographical. This implies that the biomedical framework, although having engendered 'success stories' like the era of antibiotics, needs to be radically revised.

# 1998

**Research Article**

## Relationship of Childhood Abuse and Household Dysfunction to Many of the Leading Causes of Death in Adults

The Adverse Childhood Experiences (ACE) Study

Vincent J. Felitti, MD, FACP, Robert F. Anda, MD, MS, Dale Nordenberg, MD, David F. Williamson, MS, PhD, Alison M. Spitz, MS, MPH, Valerie Edwards, BA, Mary P. Koss, PhD, James S. Marks, MD, MPH

**Background:** The relationship of health risk behavior and disease in adulthood to the breadth of exposure to childhood emotional, physical, or sexual abuse, and household dysfunction during childhood has not been described.

**Methods:** A nationally representative sample of adverse childhood experiences was mailed to 13,494 adults who had completed a standardized medical evaluation at a large HMO. 9,508 (70.5%) responded. Seven categories of adverse childhood experiences were studied: psychological, physical, or sexual abuse; violence against mother; or living with household members who were substance abusers, mentally ill or suicidal, or ever imprisoned. The number of categories of these adverse childhood experiences was used as continuous measures of adult risk behavior, health status, and mortality. Logistic regression was used to adjust the effects of demographic factors on the association between the cumulative number of categories of childhood exposures (range 0–7) and risk factors for the leading causes of death in adult life.

# The Adverse Childhood Experiences (ACE) study

## V. Felitti & R. Anda

### EPIDEMIOLOGI

Am J Prev Med 1998;14(4)  
© 1998 American Journal of Preventive Medicine

# EMBODIMENT

## N Krieger 2005

### INSCRIBED BODIES

Health Impact  
of Childhood Sexual Abuse  
Anna Lise Kirkengen

# «Kroppsinnskrifter» AL Kirkengen KVALITATIVE DYBDEINTERVJUER

Seminars in Medicine of the  
Beth Israel Deaconess Medical Center



JEFFREY S. FLIER, M.D., Editor  
LISA H. UNDERHILL, Assistant Editor

### PROTECTIVE AND DAMAGING EFFECTS OF STRESS MEDIATORS

BRUCE S. McEWEN, PH.D.

### THE PHYSIOLOGIC RESPONSE TO STRESS

Stressful experiences include major life events, trauma, and abuse and are sometimes related to the environment in the home, workplace, or neighborhood. Acute stress (in the sense of “fight or flight” or major life events) and chronic stress (the cumulative load of minor, day-to-day stresses) can both have long-term consequences. The effects of chronic stress may be exacerbated by a rich diet and the use of tobacco and alcohol and reduced by moderate exercise.

Genetic factors do not account for all the individual variability in sensitivity to stress, as evinced by the lack of concordance between identical twins in many disorders.<sup>4,5</sup> Moreover, genetic factors do not explain the gradients of health across socioeconomic levels in Western societies.<sup>6</sup> Two factors largely determine individual responses to potentially stressful situations: the way a person perceives a situation<sup>7</sup>

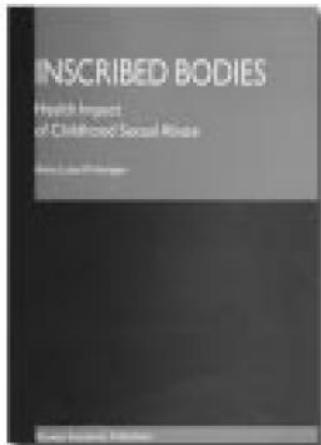
# «Allostatic load» BS McEwen FYSIOLOGI

The New England Journal of Medicine

OVER 60 years ago, Selye recognized a paradox that the physiologic systems induced by stress can not only protect and but also damage the body. What links these seemingly contradictory roles? How does stress influence the pathogenesis of disease, and what accounts for the variation in vulnerability to stress-related diseases among people with similar life experiences? Can stress-induced damage be quantified? These many other questions still challenge investigation.

# *book reviews*

The Permanente Journal 2003  
<https://www.thepermanentejournal.org/files/Spring2003/inscribed.pdf>



Boston: Kluwer Academic;  
2001. ISBN: 0-792370198.  
462 pages. \$139.

## *Inscribed Bodies: Health Impact of Childhood Sexual Abuse*

by Anna Luise Kirkengen, MD, PhD

---

Review by Vincent J Felitti, MD

**A** “Such a profound and insightful work as “*Inscribed Bodies*” comes into print only once every decade or two”

In this book, however, we are shown the profoundly destructive effects of familiarly and socially imposed secrecy and of being unable to speak openly, even to physicians, about childhood sexual abuse. Indeed, according to the author, “Violated humans are made

- Unfolding the impact of sexual violation; and
- Exploring the medical making of patients.

A short final section, *Impressions*, consists of a series of the author’s reflections expressed in the form of poetry.

*Inscribed Bodies* deserves attention as the work of

## Research Article

# Relationship of Childhood Abuse and Household Dysfunction to Many of the Leading Causes of Death in Adults

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multiple categories of childhood exposure were likely to have later in life.

**Conclusions:** We found a strong graded relationship between the breadth of exposure to abuse or household dysfunction during childhood and multiple risk factors for several of the leading causes of death in adults.

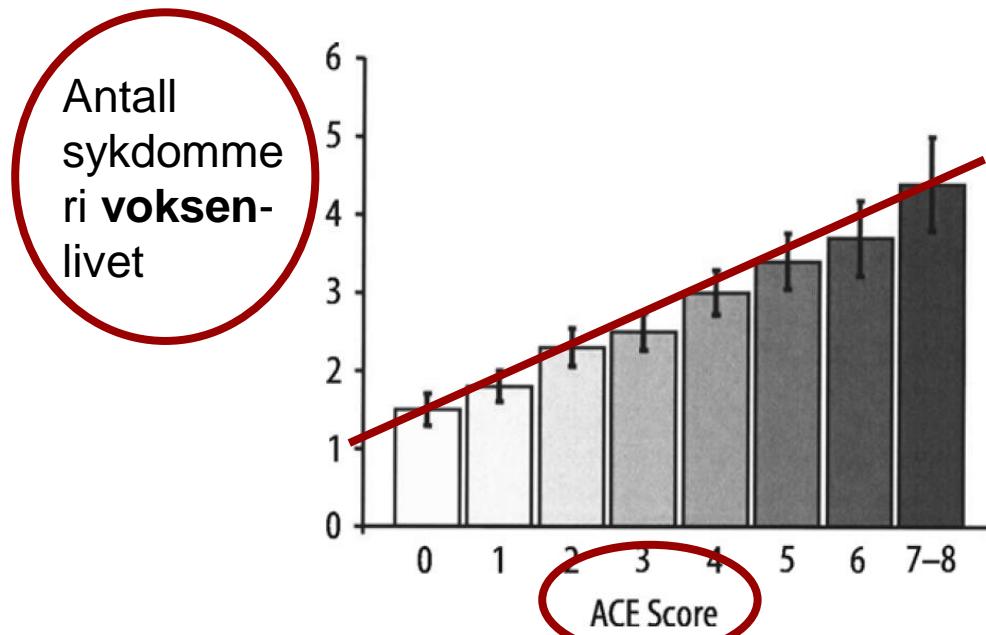
**Medical Subject Headings (MeSH):** child abuse, sexual, domestic violence, spouse abuse, children of impaired parents, substance abuse, alcoholism, smoking, obesity, physical activity, depression, suicide, sexual behavior, sexually transmitted diseases, chronic obstructive pulmonary disease, ischemic heart disease. (Am J Prev Med 1998;14:245-258) © 1998 American Journal of Preventive Medicine

**ACE studien**  
Kaiser Permanente / CDC

13.494 amerikanere  
med helseforsikring

<http://www.cdc.gov/ncccdphp/ACE/>

## Et dose-reponsforhold mellom antall typer av negative barndoms-erfaringer og sykdommer i voksenlivet



**Fig. 1** The mean number of comorbid outcomes in the study sample was 2.1 (range: 0–14); means are adjusted for age, sex, race, and educational attainment. The trend in the means is significant ( $P < 0.0001$ ); vertical error bars represent 95% confidence intervals

*The ACE study, Felitti & Anda og co, Kaiser Permanente og CDC*

Review Article

*Seminars in Medicine of the  
Beth Israel Deaconess Medical Center*



JEFFREY S. FLIER, M.D., *Editor*  
LISA H. UNDERHILL, *Assistant Editor*

**PROTECTIVE AND DAMAGING EFFECTS  
OF STRESS MEDIATORS**

BRUCE S. McEWEN, PH.D.

OVER 60 years ago, Selye<sup>1</sup> recognized the paradox that the physiologic systems activated by stress can not only protect and restore but also damage the body. What links these seemingly contradictory roles? How does stress influence the pathogenesis of disease, and what accounts for the variation in vulnerability to stress-related diseases among people with similar life experiences? How can stress-induced damage be quantified? These and many other questions still challenge investigators.

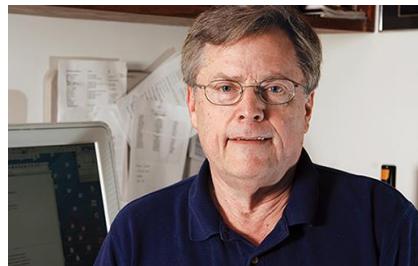
This article reviews the long-term effect of the physiologic response to stress, which I refer to as allostatic load.<sup>2</sup> Allostasis — the ability to achieve stability through change<sup>3</sup> — is critical to survival. Through allostasis, the autonomic nervous system, the hypothalamic-pituitary-adrenal (HPA) axis, and the cardiovascular, metabolic, and immune systems

**THE PHYSIOLOGIC RESPONSE TO STRESS**

Stressful experiences include major life events, trauma, and abuse and are sometimes related to the environment in the home, workplace, or neighborhood. Acute stress (in the sense of “fight or flight” or major life events) and chronic stress (the cumulative load of minor, day-to-day stresses) can both have long-term consequences. The effects of chronic stress may be exacerbated by a rich diet and the use of tobacco and alcohol and reduced by moderate exercise.

Genetic factors do not account for all the individual variability in sensitivity to stress, as evinced by the lack of concordance between identical twins in many disorders.<sup>4,5</sup> Moreover, genetic factors do not explain the gradients of health across socioeconomic levels in Western societies.<sup>6</sup> Two factors largely determine individual responses to potentially stressful situations: the way a person perceives a situation<sup>7</sup> and a person's general state of physical health, which is determined not only by genetic factors but also by behavioral and lifestyle choices (Fig. 1). Whether one perceives a situation as a threat, either psychological or physical, is crucial in determining the behavioral response — whether it is fleeing, fighting, or cowering in fear — and the physiologic response — calmness or heart palpitations and elevated cortisol levels.

The ability to adjust or habituate to repeated stress is also determined by the way one perceives a situation. For example, most people react initially to the challenge of public speaking with activation of the HPA axis. After repeated public speaking, however, most people become habituated and their cortisol secretion no longer increases with the challenge. But approximately 10 percent of subjects continue to find public speaking stressful, and their cortisol secretion increases each time they speak in



**1998**

**«Allostatic  
overload»**

**STRESSFYSIOLOGI**

**Hvordan «toksisk»  
stress overbeskatter  
kroppens kapasitet for  
tilpasning**

# **Allostase**

**beskriver kroppens fysiologiske tilpasning til utfordringer på godt og vondt, slik personen selv erfarer dem**

**Homeo-stasis => ‘stå’ stabilt**

**Allo-stasis => ‘bestå’ under endring**

Hjernen og  
nervesystemet

Hormonsystem  
et

Immunsystemet

Telomerer

Genregulering/  
Epigenetikk

....

Tidsskr Nor Legeforen nr. 7, 2011; 131: 683–7

ORIGINALARTIKKEL

## Menneskets biologi – mettet med erfaring

### Sammendrag

Bakgrunn. Mennesket er en selvreflekterende, meningssøkende, relasjonell og målrettet organisme. Det å samordne og videreføre kunnskapen om hvordan erfaringer knyttet til selvværssthet, relasjoner og verdier kan bidra til utvikling av helse og sykdom, er en stor medisinteoretisk utfordring.

Materiale og metode. Vi presenterer en teoriveiledet syntese av ny vitenskapelig kunnskap fra flere fagfelter, inkludert epigenetikk, psykoneuroendokrinoimmunologi, stressforskning og systembiologi, basert på artikler i anerkjente tidsskrifter og fagbøker. De er utvalgt for å gi innsikt i samspillet mellom eksistensielle betingelser i vid forstand (biografi) og biomolekulære forhold i kroppen (biologi).

Resultater. Forskning viser at menneskeorganismen bokstavelig talt inkorporerer biografisk (livshistorisk) informasjon, som omfatter opplevd mening og relasjoner. Epigenetikken illustrerer det grunnleggende biologiske potensialet for kontekstavhengig tilpasning. Videre er det dokumentert hvordan ulike typer eksistensielle påkjennninger kan lede til sårbarthet for sykdom gjennom forstyrrelser i mannskapsfisiologiske tilpasningsprosesse.

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Akershus universitetssykehus

Elling Ulvestad

Mikrobiologisk avdeling  
Haukeland universitetssykehus  
og  
Gades Institutt  
Universitetet i Bergen

### Materiale og metode

Organismer og deres ulike aspekter har lenge vært utforsket innen mange fag, f.eks. evolusjonsbiologi, økologi, embryologi og fysiologi. Det resulterte lenge i ulike faglige oppfatninger av organismens betydning, men ingen enhetlig teori. I de senere år har forståelsen av organismen som *et levende hele*, det være seg en bakterie eller et menneske, fått en mer sentral plass i biologisk tenkning (3). Med dette som utgangspunkt presenterer vi her kunnskap som synliggjør samspillet mellom menneskets kropp (biologi) og livserfaring (biografi) i vid forstand.

Utvæltet av referanser er styrt av en forståelse av den menneskelige organismen som *integrert med sine omgivelser og relasjonell i sin natur*. Det har sin bakgrunn i flere års personlig samling av litteratur på feltet. Vi inkluderer studier fra epidemiologi, somatisk og psykiatrisk klinisk medisin, genetikk, mikrobiologi, immunologi og nevrovitenskapene. I tillegg inngår arbeider fra nye, fagovergripende disipliner som nevroendokrinologi og psykoneuroendokrinoimmunologi samt analyser innen biosemiotikk, evolusjonsbiologi, vitenskapsteori og filosofi. Temaets art og bredde har i liten grad egnet seg for bruk av systematiske litteratursøk.

### Resultater

En erkjennelse av at enhver organisme aktivt fortolkjer betydningsfulle signaler fra det ytre og indre miljø finnes i biologen Jakob von Uexkülls (1864–1944) økologiske

## „Toksisk stress“

Høy belastning (overveldende påkjenninger, evt. kronisk stress) har biologiske ‘kostnader’ på sikt:

### **Allostatisk overbelastning**

Et integrativt begrep som tar høyde for at **mentale og fysiske belastninger** ‘overbeskatter’ **de samme** fysiologiske systemene.

*Bruce McEwen, 1998*



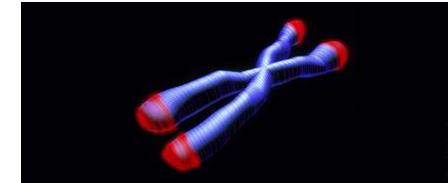
The NEW ENGLAND  
JOURNAL of MEDICINE

# Allostatisk (over)belastning

autonom-nervøse, hormonelle, immunologiske variable, m.m.

**ENHETLIG IDE – LITT ULIKE ALGORITMER**

- Kardiovaskulære mål (blodtrykk, pulsfrekvens, m.fl.)
- Lungefunksjonstester (PEF, spirometri)
- Hormonnivåer (cortisol, DHEA-S, A, NA, m.fl.)
- Inflammasjonsmarkører (CRP, IL-6, TNF, Fibrinogen m.fl.)
- Glucose-metabolisme (blodsukker, HbA1c)
- Lipid-metabolisme (TG, HDL, LDL, m.fl.)
- Kroppsmål (WHR, BMI m.fl.)
- *Epigenetisk profil*
- *Telomerlengde og telomerasenivå*
- *Mitokondriefunksjon...*



Science Photo Library

# Hjernen og nervesystemet

## Hormonsystemet

### et

## Immunsystemet

## Telomerer

## Genregulering/ Epigenetikk

....

Tidsskr Nor Legeforen nr. 7, 2011; 131: 683–7

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### Materiale og metode

Organismer og deres ulike aspekter har lenge vært utforsket innen mange fag, f.eks. evolusjonsbiologi, økologi, embryologi og fysiologi. Det resulterte lenge i ulike faglige oppfatninger av organismens betydning, men ingen enhetlig teori. I de senere år har forståelsen av organismen som *et levende hele*, det være seg en bakterie eller et menneske, fått en mer sentral plass i biologisk tenkning (3). Med dette som utgangspunkt presenterer vi her kunnskap som synliggjør samspillet mellom menneskets kropp (biologi) og livserfaring (biografi) i vid forstand.

Utvæltet av referanser er styrt av en forståelse av den menneskelige organismen som *integrert med sine omgivelser og relasjonell i sin natur*. Det har sin bakgrunn i flere års personlig samling av litteratur på feltet. Vi inkluderer studier fra epidemiologi, somatisk og psykiatrisk klinisk medisin, genetikk, mikrobiologi, immunologi og nevrovitenskapene. I tillegg inngår arbeider fra nye, fagovergripende disipliner som nevroendokrinologi og psykoneuroendokrinoimmunologi samt analyser innen biosemiotikk, evolusjonsbiologi, vitenskapsteori og filosofi. Temaets art og bredde har i liten grad egnet seg for bruk av systematiske litteratursøk.

### Resultater

En erkjennelse av at enhver organisme aktivt fortolkjer betydningsfulle signaler fra det ytre og indre miljø finnes i biologen Jakob von Uexkülls (1864–1944) økologiske

# Eksistensielle grunnvilkår gjenspeiles i biologien

## “Nærer” (salutogenese)

Tillit

Tilhørighet

Respekt

Omsorg

Ære og stolthet

Mening

Innflytelse

## “Tærer” (patogenese)

Trusler og svik

Isolasjon og  
forlatthet

Krenkelse av  
integritet

Vanskjøtsel

Skyld og skam

Meningsløshet

Avmakt

“Verdighetens og  
tilhørighetens fysiologi”?

Allostatisk overbelastning  
“avmaktens patofysiologi”

# Allostatisk overbelastning

*“For lite av det som nærer,  
for mye av det som tærer”*

- Anna Luise Kirkengen

- Overveldende ansvar
- Isolasjon, ensomhet
- Integritetskrenkeler
- Slitsomme relasjoner
- Urettferdighet, svik
- AVMAKT

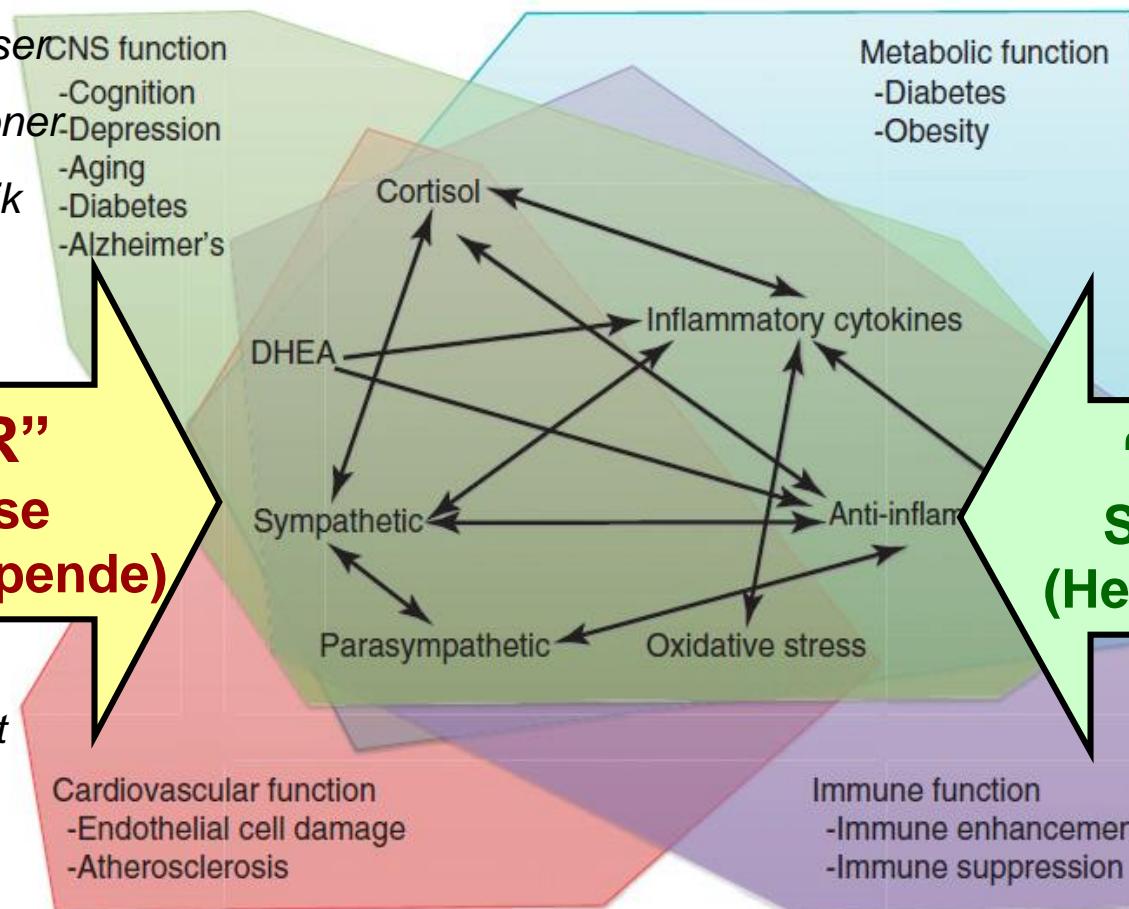
**“Mentale”**

**“TÆRER”**  
Patogenese  
(Sykdomsskapende)

**“Fysiske”**

- Søvn mangel/skift
- Fysisk slit
- Inaktivitet
- Usunn mat
- Sykdom og smerte (onde sirkler), medikamenter (bivirkninger)
- Røyking og andre rusmidler
- Forurensing

# Allostase – et liv i balanse?



• Tillit, trygghet, støtte

• Anledninger til å vokse og utvikle seg

• Opplevd mening (ex. samtaler, kunst)

**“Mentale”**

**“NÆRER”**  
Salutogenese  
(Helsefremmende)

**“Fysiske”**

- God søvn
- Fysisk aktivitet og rekreasjon
- Sunn mat
- Relevante medikamenter
- Osv.

**CNS: kognitive og  
psykiatriske lidelser**

**„Sensitisering“**

**Metabske  
forstyrrelser**

# MULTIMORBIDITET

**„TÆRE“**

**„NÆRER“**

Cardiovascular function  
-Endothelial cell damage  
-Atherosclerosis

Immune function  
-Immune enhancement  
-Immune suppression



**Hjerte/kar-  
sykdommer**

**Immunologisk  
relaterte lidelser**

## Epidemiology of multimorbidity and implications for health care, research, and medical education: a cross-sectional study

Karen Barnett, Stewart W Mercer, Michael Norbury, Graham Watt, Sally Wyke, Bruce Guthrie

### Summary

**Background** Long-term disorders are the main challenge facing health-care systems worldwide, but health systems are largely configured for individual diseases rather than multimorbidity. We examined the distribution of multimorbidity, and of comorbidity of physical and mental health disorders, in relation to age and socioeconomic deprivation.

Published Online  
May 10, 2012  
DOI:10.1016/S0140-6736(12)60240-2  
See Online/Comment  
DOI:10.1016/S0140-

**Methods** In a cross-sectional study we extracted data on 40 morbidities from a database of 1751841 people registered

# Grensen fysisk/psykisk viskes ut En klar sosial gradient

deprived areas compared with the most affluent, with socioeconomic deprivation particularly associated with multimorbidity that included mental health disorders (prevalence of both physical and mental health disorder 11·0%, 95% CI 10·9–11·2% in most deprived area vs 5·9%, 5·8%–6·0% in least deprived). The presence of a mental health disorder increased as the number of physical morbidities increased (adjusted odds ratio 6·74, 95% CI 6·59–6·90 for five or more disorders vs 1·95, 1·93–1·98 for one disorder), and was much greater in more deprived than in less deprived people (2·28, 2·21–2·32 vs 1·08, 1·05–1·11).

**Interpretation** Our findings challenge the single-disease framework by which most health care, medical research, and medical education is configured. A complementary strategy is needed, supporting generalist clinicians to provide personalised, comprehensive continuity of care, especially in socioeconomically deprived areas.

**Funding** Scottish Government Chief Scientist Office.

### Introduction

Management of the rising prevalence of long-term disorders is the main challenge facing governments and health-care systems worldwide.<sup>1</sup> Although individual diseases dominate health-care delivery, medical research, and medical education, people with multimorbidity—those with two or more chronic morbidities—need a broader approach. Use of many services to manage individual diseases can become duplicative and inefficient, and is burdensome and unsafe for patients because of poor coordination and integration.<sup>2,4</sup> Multimorbidity becomes progressively more common with age<sup>5–7</sup> and is associated with high mortality,<sup>8</sup> reduced functional status,<sup>9,10</sup> and increased use of both inpatient and ambulatory health care.<sup>11</sup> Estimates of the prevalence

have life expectancies 13 years shorter, and women 9 years shorter than do those in the most affluent 10%. The most deprived people spend twice as many years in poor health before they die than do the most affluent (10·3 years vs 5·5 years for men; 14·4 years vs 6·0 years for women).<sup>15</sup>

Better understanding of the epidemiology of multimorbidity is necessary to develop interventions to prevent it, reduce its burden, and align health-care services more closely with patients' needs. We aimed to use a large, representative primary medical care electronic database to examine the distribution of multimorbidity in relation to age and socioeconomic deprivation, and the relation between comorbidity of physical and mental health disorders and deprivation.

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# Epidemiology of multimorbidity and implications for health care, research, and medical education: a cross-sectional study

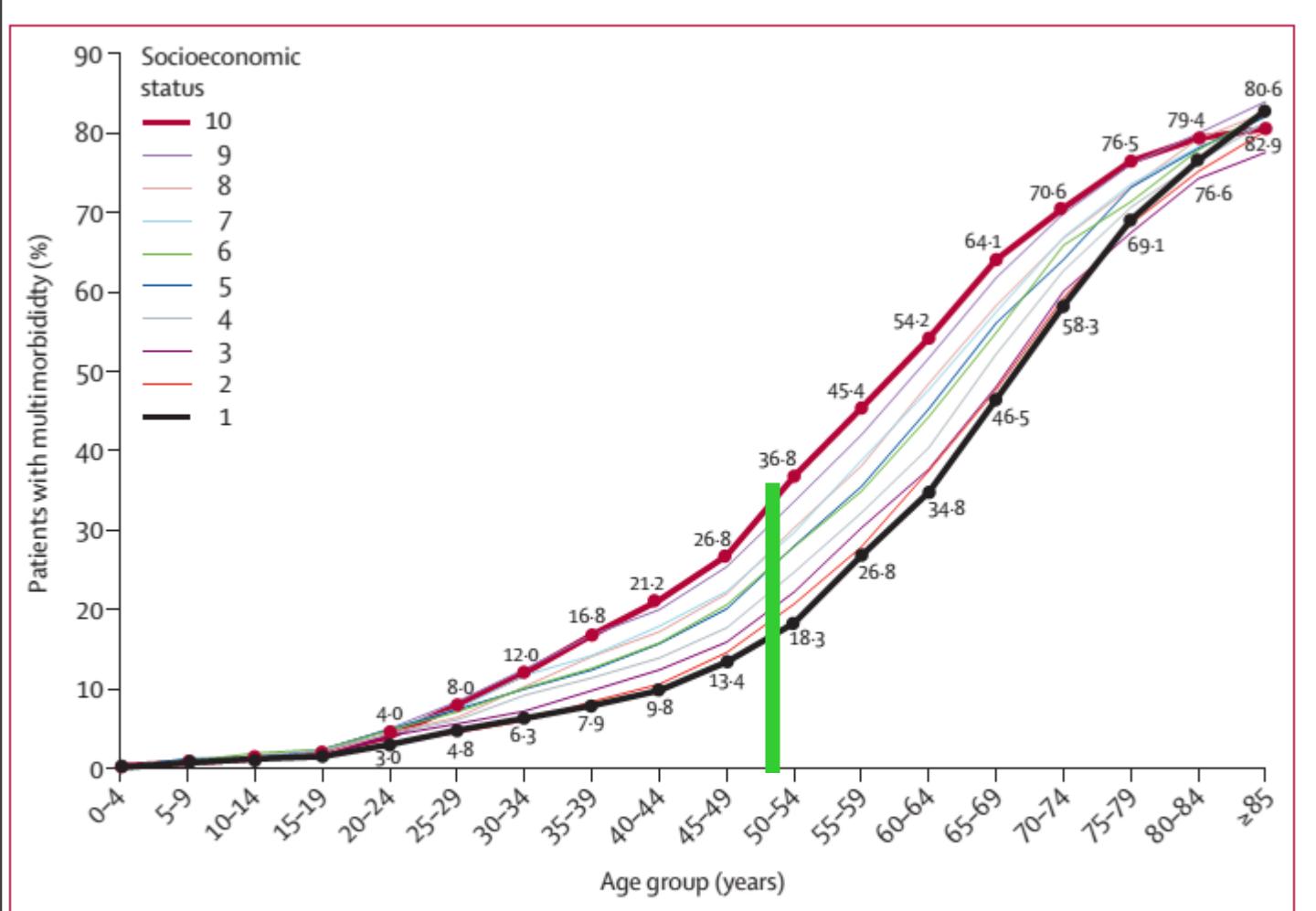


Karen Barnett, Stewart W Mercer, Michael Norbury, Graham Watt, Sally Wyke, Bruce Guthrie

## Summary

**Background** Long-term health largely configured for risk and of comorbidity of

**Methods** In a cross-sectional study with 314 medical practices, by disorder type (physical and mental) and two or more disorders



**Figure 2: Prevalence of multimorbidity by age and socioeconomic status**  
On socioeconomic status scale, 1=most affluent and 10=most deprived.

# Do socioeconomic inequalities in pain, psychological distress and oral health increase or decrease over the life course? Evidence from Sweden over 43 years of follow-up

Roger Keller Celeste,<sup>1,2</sup> Johan Fritzell<sup>2</sup>

► Additional material is published online only. To view please visit the journal online (<http://dx.doi.org/10.1136/jech-2017-209123>).

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Received 26 February 2017  
Revised 3 October 2017  
Accepted 16 October 2017  
Published Online First  
24 November 2017

## ABSTRACT

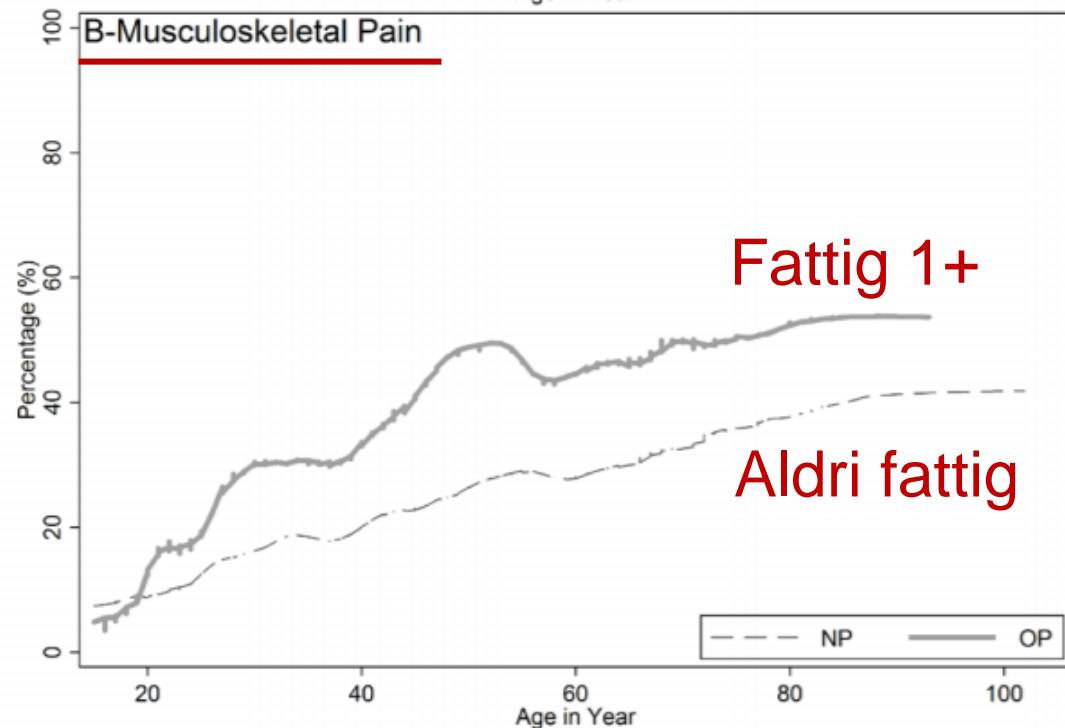
**Background** Inequalities over the life course may increase due to accumulation of disadvantage or may decrease because ageing can work as a leveller. We report how absolute and relative socioeconomic inequalities in musculoskeletal pain, oral health and psychological distress evolve with ageing.

**Methods** Data were combined from two nationally representative Swedish panel studies: the Swedish Level-of-Living Survey and the Swedish Panel Study of Living Conditions of the Oldest Old. Individuals were followed up to 43 years in six waves (1968, 1974, 1981, 1991/1992, 2000/2002, 2010/2011) from five cohorts: 1906–1915 (n=899), 1925–1934 (n=906), 1944–1953 (n=1154), 1957–1966 (n=923) and 1970–1981 (n=1199). The participants were 15–62 years at baseline. Three self-reported outcomes were measured as dichotomous variables: teeth not in good conditions, psychological distress and musculoskeletal pain. The fixed-income groups were: (A) never poor and (B) poor at least once in life. The relationship between ageing and the outcomes was smoothed with locally weighted ordinary least squares, and the relative and absolute gaps were calculated with Poisson regression using generalised estimating equations.

**Results** All outcomes were associated with ageing, birth cohort, sex and being poor at least once in life. Absolute inequalities increased up to the age of 45–64 years, and then they decreased. Relative inequalities were large already in individuals aged 15–25 years, showing a declining trend over the life course. Selective mortality did not change the results. The socioeconomic gap was larger for current poverty than for being poor at least once in life.

**Conclusion** Inequalities persist into very old age, though they are more salient in midlife for all three outcomes observed.

The gap may evolve differently when comparing mortality and morbidity indicators. Most mortality studies have shown either decreasing or stable inequalities across the lifespan.<sup>3–9</sup> Age-as-leveler has been considered an explanation for converging inequalities, for example, welfare programmes and



on morbidity, and studied inequalities in three different health outcomes that have consistently been associated with socioeconomic conditions

# Teicher & Samson (2016)

## The effects of childhood maltreatment on brain structure, function and connectivity

Martin H. Teicher<sup>1,2</sup>, Jacqueline A. Samson<sup>1,2</sup>, Carl M. Anderson<sup>1,2</sup> and Kyoko Ohashi<sup>1,2</sup>

**Abstract** | Maltreatment-related childhood adversity is the leading preventable risk factor for mental illness and substance abuse. Although the association between maltreatment and psychopathology is compelling, there is a pressing need to understand how maltreatment increases the risk of psychiatric disorders. Emerging evidence suggests that maltreatment alters trajectories of brain development to affect sensory systems, network architecture and circuits involved in threat detection, emotional regulation and reward anticipation. This Review explores whether these alterations reflect toxic effects of early-life stress or potentially adaptive modifications, the relationship between psychopathology and brain changes, and the distinction between resilience, susceptibility and compensation.

Brain development is directed by genes but sculpted by experiences, particularly those occurring during early sensitive or critical periods. Studies suggest that the onset of regional critical periods may be triggered when GABAergic inhibitory influences develop to the point that they come into balance with excitatory influences<sup>1</sup>. This balance allows experience to shape and fine-tune connectivity patterns and network architecture. Contrary to earlier notions, plasticity is not lost but seems to be dampened by molecular ‘brakes’ that draw critical periods to a close, although these brakes can be lifted through pharmacological manipulations or epigenetic modifications<sup>1</sup>. Together, these developmental processes provide a highly adaptive mechanism for the formation of optimally sculpted neural representations to guide future actions based on early experience, while allowing possible revisions<sup>1</sup>.

There are few early experiences as consequential as abuse and neglect. Studies on the effects of childhood maltreatment typically include physically, sexually and emotionally abusive acts and episodes of both physical and emotional neglect. Emotional maltreatment includes intentionally eliciting feelings of guilt, shame or fear to serve the emotional needs of the perpetrator; persuading children to perform inappropriate acts; denigrating or destroying things they value; or placing them in harmful situations, such as witnessing interparental violence<sup>2</sup>. Physical neglect is defined as failure to provide basic needs such as food, clean clothing, shelter, supervision, and dental and paediatric care<sup>2</sup>. Emotional neglect is the failure to provide for fundamental emotional needs, by

being emotionally unresponsive to children’s distress, failing to attend to their social needs or expecting children to manage situations that are beyond their maturity level or unsafe<sup>2</sup>. Some studies also include exposure to various forms of household dysfunction, such as living with substance-abusing parents.

According to the Adverse Childhood Experiences (ACEs) study, a collaboration between Kaiser Permanente and US Centers for Disease Control and Prevention, exposure to one or more maltreatment-related ACEs accounts for 54% of the population attributable risk (PAR) for depression<sup>3</sup>, 67% of the PAR for suicide attempts<sup>3</sup> and 64% of the PAR for addiction to illicit drugs<sup>4</sup>. Exposure to five or more ACEs was associated with a 2-, 3-, 10- or 17-fold increase in risk for receiving a prescription of an anxiolytic, antidepressant, antipsychotic or mood-stabilizing medication, respectively<sup>5</sup>. Individuals exposed to six or more ACEs were found to have a 20-year reduction in lifespan<sup>6</sup>, which may be due to accelerated telomere shortening<sup>7</sup>. Understanding how maltreatment increases risk of various psychiatric and medical disorders is of crucial importance to prevent, pre-empt or treat the consequences of abuse and neglect.

We hypothesized several years ago that maltreatment acts as a stressor to produce a cascade of physiological and neurohumoral reactions that alter brain-development trajectories, setting the stage for the emergence of psychiatric symptoms in genetically susceptible individuals<sup>8–10</sup>. Since then, there have been more than 180 original reports showing an association between childhood maltreatment and alterations in brain structure, function,

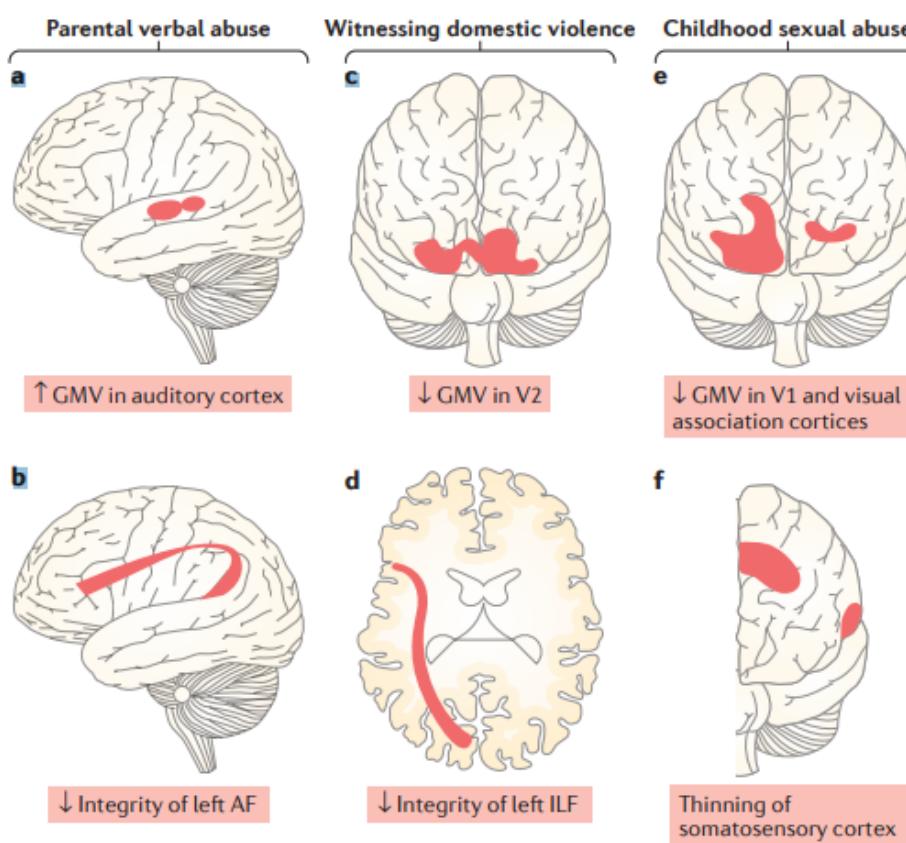
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doi:10.1058/nrn.2016.111

Published online 19 Sep 2016

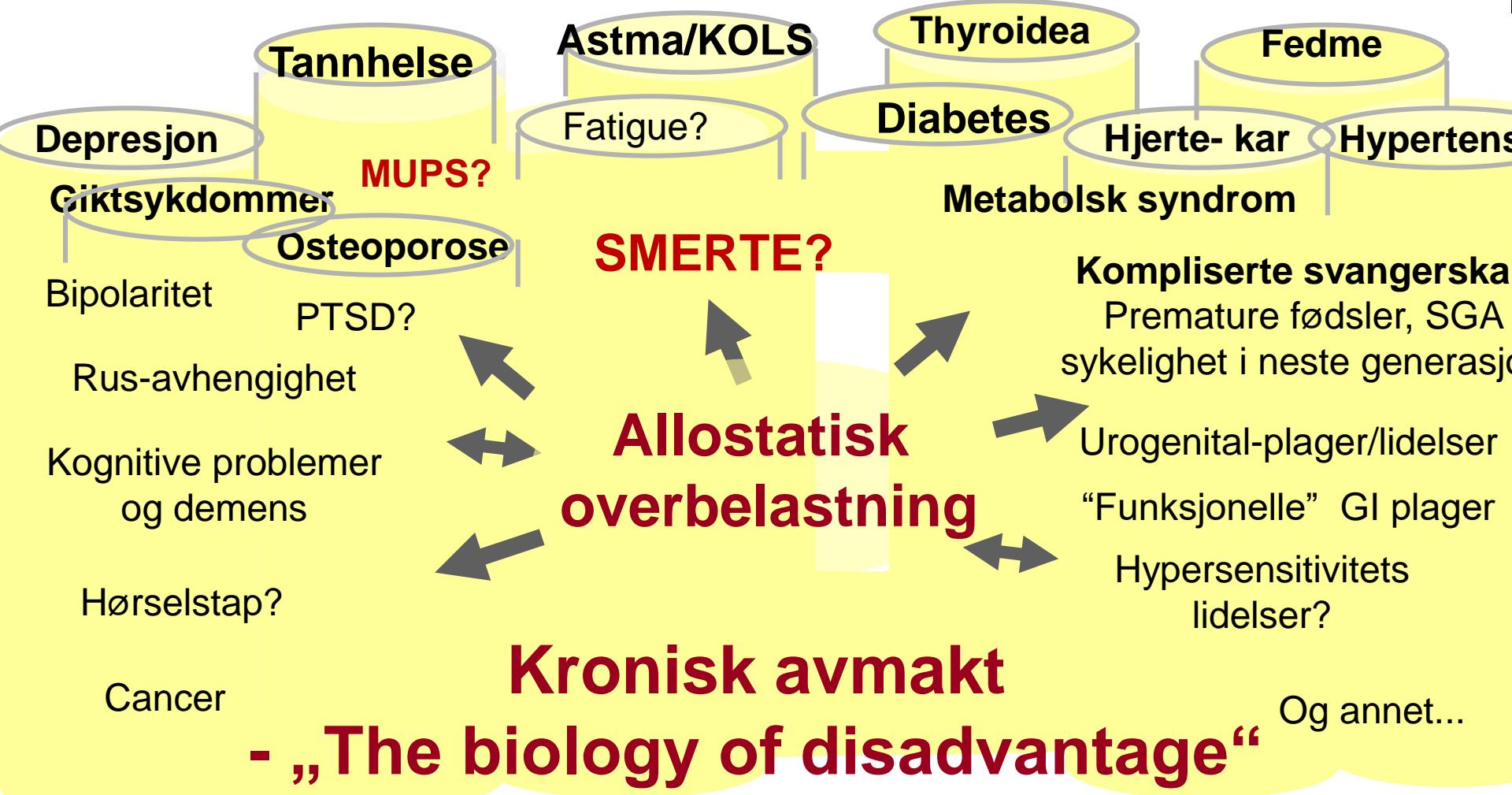


**Figure 1 | Abuse type-specific effects on the developing brain.** Images depicting the potential effects of exposure to specific types of childhood maltreatment on grey-matter volume (GMV) or thickness and fibre-tract integrity. Exposure to parental verbal abuse was associated with increased GMV in the auditory cortex portion of the left superior temporal gyrus<sup>25</sup> (part a) and decreased integrity of the left arcuate fasciculus (AF) interconnecting Wernicke's area and Broca's area<sup>26</sup> (part b). Visually witnessing multiple episodes of domestic violence was associated with reduced GMV in right lingual gyrus, left occipital pole and bilateral secondary visual cortex (V2)<sup>27</sup> (part c) and decreased integrity of the left inferior longitudinal fasciculus (ILF), which serves as a visual-limbic pathway<sup>28</sup> (part d). Adults reporting exposure to multiple episodes of childhood forced-contact sexual abuse were found to have reduced GMV in right and left primary visual cortex (V1) and visual association cortices, as well as reduced thickness in right lingual, left fusiform and left middle occipital gyri<sup>29</sup> (part e) and portions of the somatosensory cortex representing the clitoris and surrounding genital area<sup>30</sup> (part f). Part a is adapted with permission from REF. 25, Elsevier. Part b is adapted with permission from REF. 26, Elsevier. Part c is adapted from REF. 27. Part d is adapted with permission from REF. 28, Elsevier. Part e is adapted with permission from REF. 29, Elsevier. Part f is adapted from an image courtesy of C. Heim, Charité Universitätsmedizin Berlin, Germany, and J. Pruessner, McGill University, Canada.

**Teicher & Samson  
2016 (Review)**

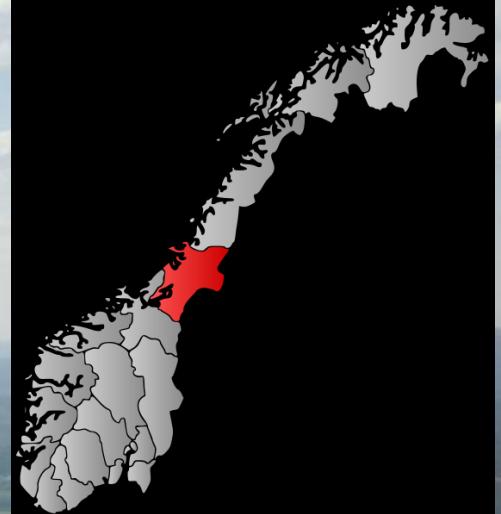
**Sansespesifikke  
strukturelle og  
funksjonelle endringer  
i CNS s.f.a  
traumatiske erfaringer**

# «VULKAN-MEDISIN»?



# Hvordan er det i NORGE?

Multimorbiditet  
Barndom og helse



# HUNT3 2006-8, Ca. 48 000, 20-79 år

## ARTICLE

### **Co- and multi-morbidity patterns in an unselected Norwegian population: cross-sectional analysis based on the HUNT Study and theoretical reflections concerning basic medical models**

Margret Olafia Tomasdottir MD<sup>a</sup>, Linn Getz MD PhD<sup>b</sup>, Johann A. Sigurdsson MD Dr med<sup>c</sup>, Halfdan Petursson MD PhD<sup>d</sup>, Anna Luise Kirkengen MD Dr med<sup>e</sup>, Steinar Krokstad MD PhD<sup>f</sup>, Bruce McEwen PhD<sup>g</sup> and Irene Hetlevik MD Dr med<sup>h</sup>

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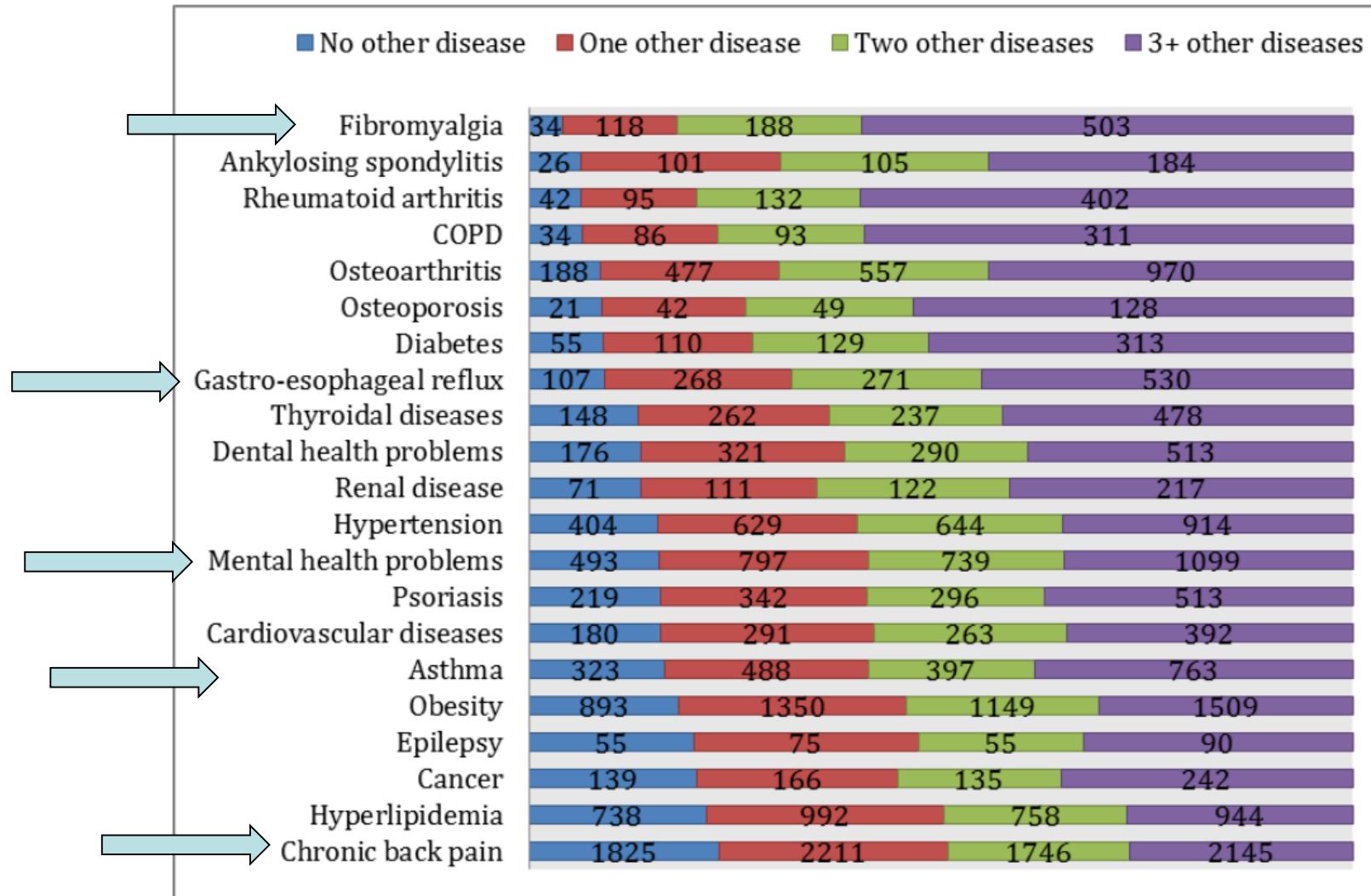
f Director, HUNT Research Centre, The Nord-Trøndelag Health Study Centre (HUNT), Department of Public Health and General Practice, Norwegian University of Science and Technology (NTNU), Trondheim, Norway

g Professor & Head of Laboratory, Laboratory of Neuroendocrinology, The Rockefeller University, New York, NY, USA

h Professor, General Practice Research Unit, Department of Public Health and General Practice, Norwegian University of Science and Technology (NTNU), Trondheim, Norway

# Multimorbiditet ut fra 21 index-sykdommer i HUNT 3

**Figure 3 Number and distribution of disease clustering / multimorbidity by index diseases in the age-group 40-59 years. COPD = Chronic obstructive pulmonary disease**



## RESEARCH ARTICLE

# Self Reported Childhood Difficulties, Adult Multimorbidity and Allostatic Load. A Cross-Sectional Analysis of the Norwegian HUNT Study

Margret Olafia Tomasdottir<sup>1,2\*</sup>, Johann Agust Sigurdsson<sup>1,2</sup>, Halfdan Petursson<sup>2</sup>, Anna Luise Kirkengen<sup>2,3</sup>, Steinar Krokstad<sup>4</sup>, Bruce McEwen<sup>5</sup>, Irene Hetlevik<sup>2</sup>, Linn Getz<sup>2</sup>

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## OPEN ACCESS

**Citation:** Tomasdottir MO, Sigurdsson JA, Petursson H, Kirkengen AL, Krokstad S, McEwen B, et al. (2015) Self Reported Childhood Difficulties, Adult Multimorbidity and Allostatic Load. A Cross-Sectional Analysis of the Norwegian HUNT Study. PLoS ONE 10(6): e0130591. doi:10.1371/journal.pone.0130591

**Academic Editor:** Chang-Qing Gao, Central South University, CHINA

**Received:** August 28, 2014

## Abstract

## Background

Multimorbidity receives increasing scientific attention. So does the detrimental health impact of adverse childhood experiences (ACE). Aetiological pathways from ACE to complex disease burdens are under investigation. In this context, the concept of *allostatic over-*

**51** Hvor mye melk eller yoghurt drakk du vanligvis?

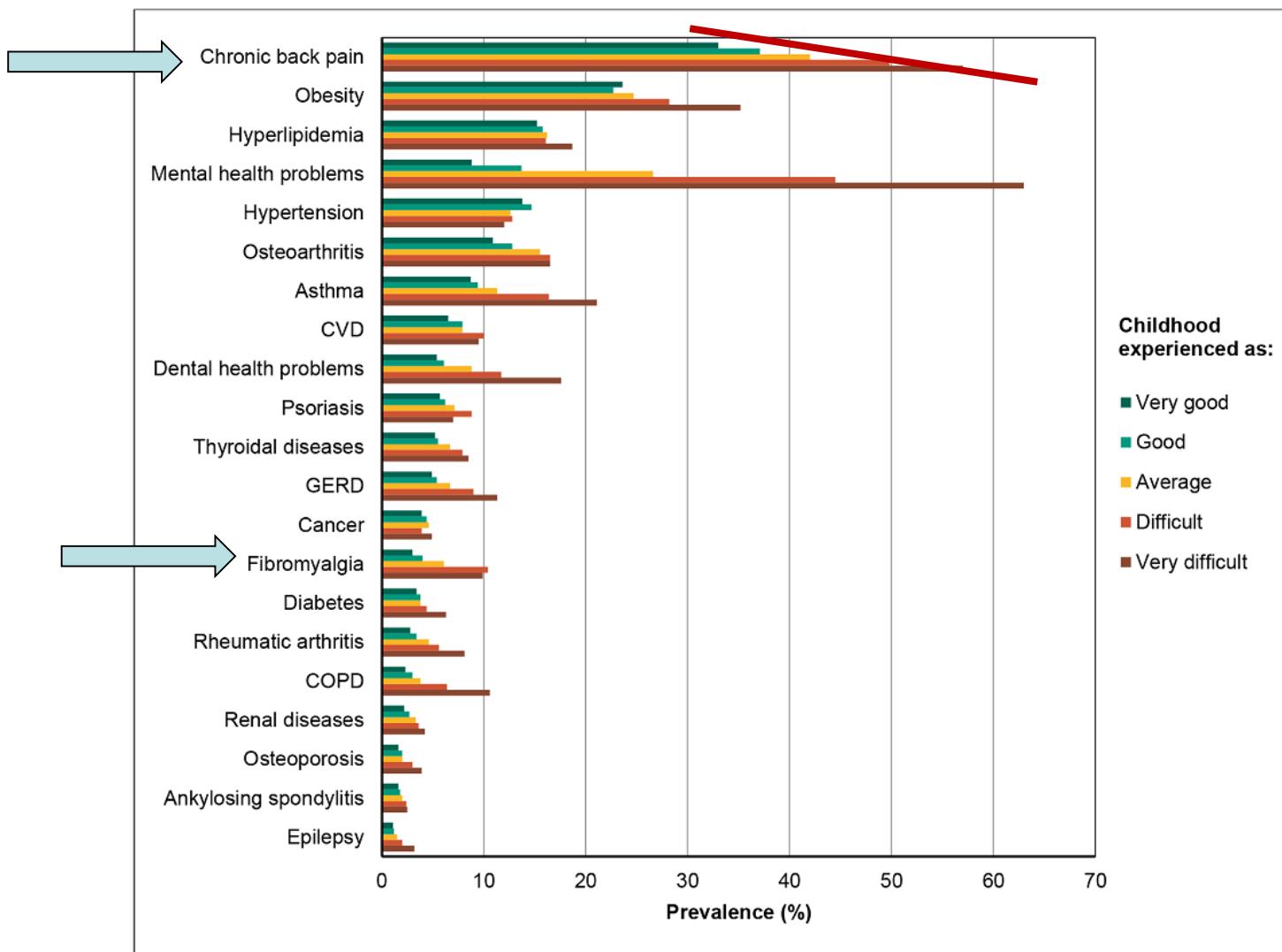
Sjeldent/ aldri	1-6 gl. pr. uke	1 glass pr. dag	2-3 gl. pr. dag	Mer enn 3 glass pr. dag
<input type="checkbox"/>				

**52** Vokste du opp på gård med husdyr?

Ja  Nei

**53** Når du tenker på barndommen/oppveksten din,  
vil du beskrive den som:

Svært god .....	<input type="checkbox"/>	Vansklig .....	<input type="checkbox"/>
God .....	<input type="checkbox"/>	Svært vanskelig.....	<input type="checkbox"/>
Middels .....	<input type="checkbox"/>		



**Fig 3. Prevalence of diseases/conditions according to childhood experience for adults (30–69y) in the HUNT3 Study.**

doi:10.1371/journal.pone.0130591.g003

OK, Einstein –  
Nye forsøk på  
problemanalyse!





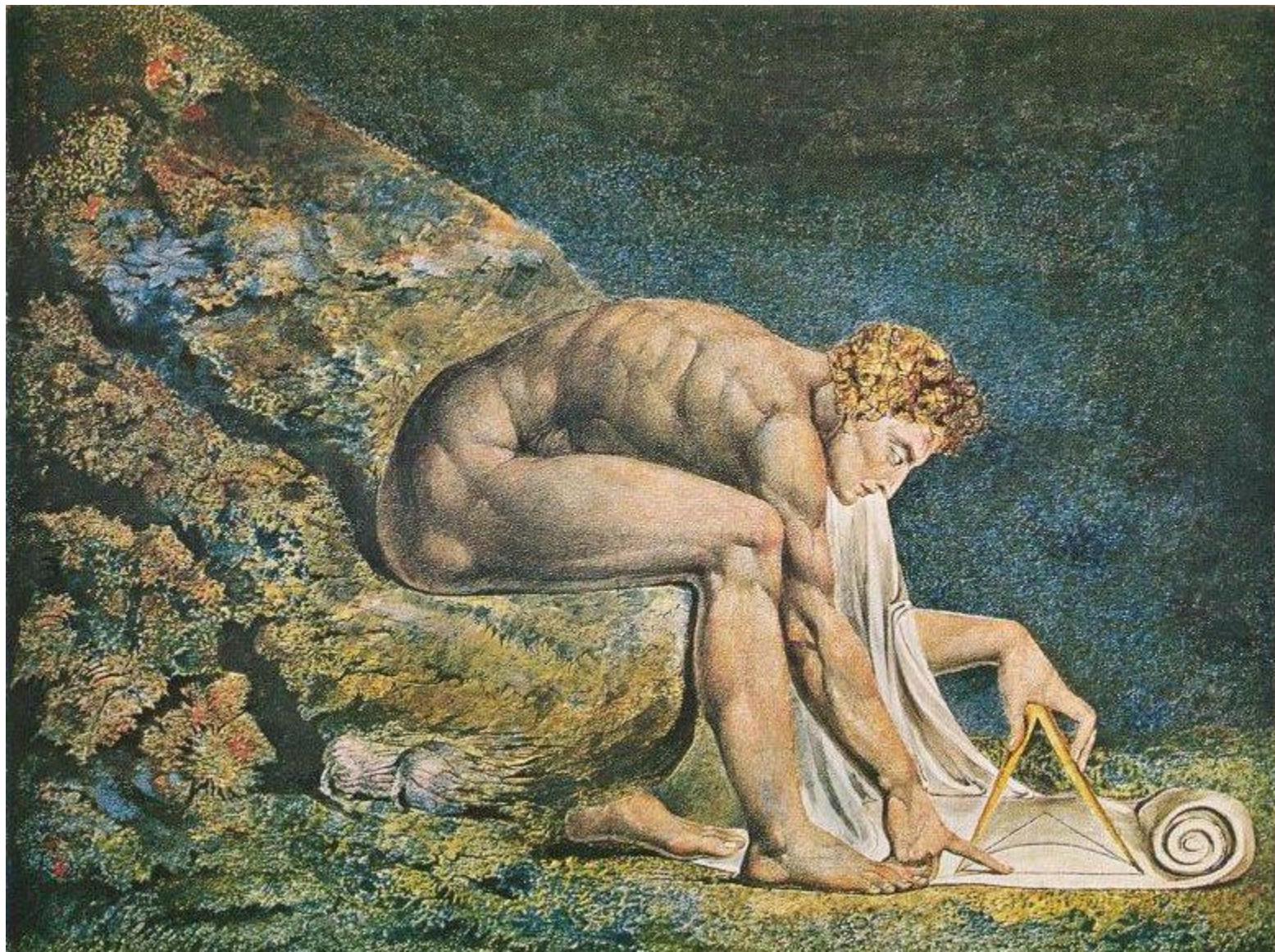
# "My Story Is Broken; Can You Help Me Fix It?"

The joint construction of  
narrative in medicine

Howard Brody

1994





*William Blake: Newton (1795/1805).*

# Embodiment: a conceptual glossary for epidemiology

Nancy Krieger

*J Epidemiol Community Health* 2005;59:350–355. doi: 10.1136/jech.2004.024

Recognising that we, as humans, are simultaneously social beings and biological organisms, the notion of “embodiment” advances three critical claims:

- (1) bodies tell stories about—and cannot be studied divorced from—the conditions of our existence;
- (2) bodies tell stories that often—but not always—match people’s stated accounts; and
- (3) bodies tell stories that people cannot or will not tell, either because they are unable, forbidden, or choose not to tell.

Just as the proverbial “dead man’s bones” do in fact tell tales, via forensic pathology and historical anthropometry,<sup>5–7</sup> so too do our living bodies tell stories about our lives, whether or not these are ever consciously expressed.

# BMJ Open Does 'existential unease' predict adult multimorbidity? Analytical cohort study on embodiment based on the Norwegian HUNT population

Margret Olafia Tomasdottir,<sup>1,2</sup> Johann Agust Sigurdsson,<sup>1,2</sup> Halfdan Petursson,<sup>2</sup> Anna Luise Kirkengen,<sup>2</sup> Tom Ivar Lund Nilsen,<sup>2</sup> Irene Hetlevik,<sup>2</sup> Linn Getz<sup>2</sup>

**To cite:** Tomasdottir MO, Sigurdsson JA, Petursson H, et al. Does 'existential unease' predict adult multimorbidity? Analytical cohort study on embodiment based on the Norwegian HUNT population. *BMJ Open* 2016;6:e012602. doi:10.1136/bmjopen-2016-012602

► Prepublication history and additional material is available. To view please visit the journal (<http://dx.doi.org/10.1136/bmjopen-2016-012602>).

Received 11 May 2016  
Revised 16 September 2016  
Accepted 5 October 2016

## ABSTRACT

**Objectives:** Multimorbidity is prevalent, and knowledge regarding its aetiology is limited. The general pathogenic impact of adverse life experiences, comprising a wide-ranging typology, is well documented and coherent with the concept *allostatic overload* (the long-term impact of stress on human physiology) and the notion *embodiment* (the conversion of sociocultural and environmental influences into physiological characteristics). Less is known about the medical relevance of subtle distress or unease. The study aim was to prospectively explore the associations between *existential unease* (coined as a meta-term for the included items) and multimorbidity.

**Setting:** Our data are derived from an unselected Norwegian population, the Nord-Trøndelag Health Study, phases 2 (1995–1997) and 3 (2006–2008), with a mean of 11 years follow-up.

**Participants:** The analysis includes 20 365 individuals aged 20–59 years who participated in both phases and was classified without multimorbidity (with 0–1 disease) at baseline.

**Methods:** From HUNT2, we selected 11 items indicating 'unease' in the realms of self-esteem, well-being, sense of coherence and social relationships. Poisson regressions were used to generate relative risk (RR) of developing multimorbidity according to the

## Strength and limitations of this study

- This large, prospective study explores subtle aetiological factors of multimorbidity, a fairly new area of investigation.
- The study shows that relatively subtle, existentially demanding life circumstances are associated with the development of multimorbidity.
- The data come from a large, homogenous and relatively affluent population. Finding effect of subtle unease on future health even in this population highlights its importance.
- The basic science concept *allostatic load* is key to our hypothesis. We described the participants' allostatic load at the level of tertiary outcomes (established diseases/conditions) in accordance with the literature. Our findings suggest that a subjective experience of existential unease is associated with allostatic load in a long-term perspective.
- The findings have relevance for general practice/primary healthcare and raise the question whether attentive, person-centred dialogues can contribute to treatment and prevention of complex disease within the frame of an established doctor–patient relationship.



CrossMark

## **Child maltreatment and adult multimorbidity: results from the Canadian Community Health Survey.**

[England-Mason G<sup>1,2</sup>](#), [Casey R<sup>3</sup>](#), [Ferro M<sup>4,5</sup>](#), [MacMillan HL<sup>2,5</sup>](#), [Tonmyr L<sup>6</sup>](#), [Gonzalez A<sup>7,8</sup>](#).

### **⊕ Author information**

#### **Abstract**

**OBJECTIVES:** This study investigated associations between three types of child maltreatment (exposure to intimate partner violence, sexual, and physical abuse) and multimorbidity (chronic physical conditions, pain conditions, and mental disorders) in adults.

**METHODS:** Multinomial logistic regression was used to analyze weighted data from the 2012 Canadian Community Health Survey (CCHS - MH 2012), a representative population sample ( $N = 23,846$ ) of respondents ages 18+.

**RESULTS:** All three subtypes of child maltreatment independently predicted increased odds of experiencing multimorbidity as an adult, while adjusting for covariates (adjusted odds ratios ranged from 1.34 (95% CI = 1.00, 1.80) to 4.87 (95% CI = 2.75, 8.63)). A dose-response relationship between the number of child maltreatment subtypes and risk for multimorbidity was also observed (adjusted odds ratios ranged from 1.38 (95% CI = 1.11, 1.73) to 10.96 (95% CI = 6.12, 19.64)).

**CONCLUSION:** The current results highlight the importance of considering a range of childhood adversities and suggest that public health approaches that aim to decrease the prevalence and severity of child maltreatment have the potential to ameliorate adult multimorbidities. Future research is encouraged to investigate these issues using longitudinal population-level data.

**KEYWORDS:** Child abuse; Chronic disease; Intimate partner violence; Mental disorder; Public health

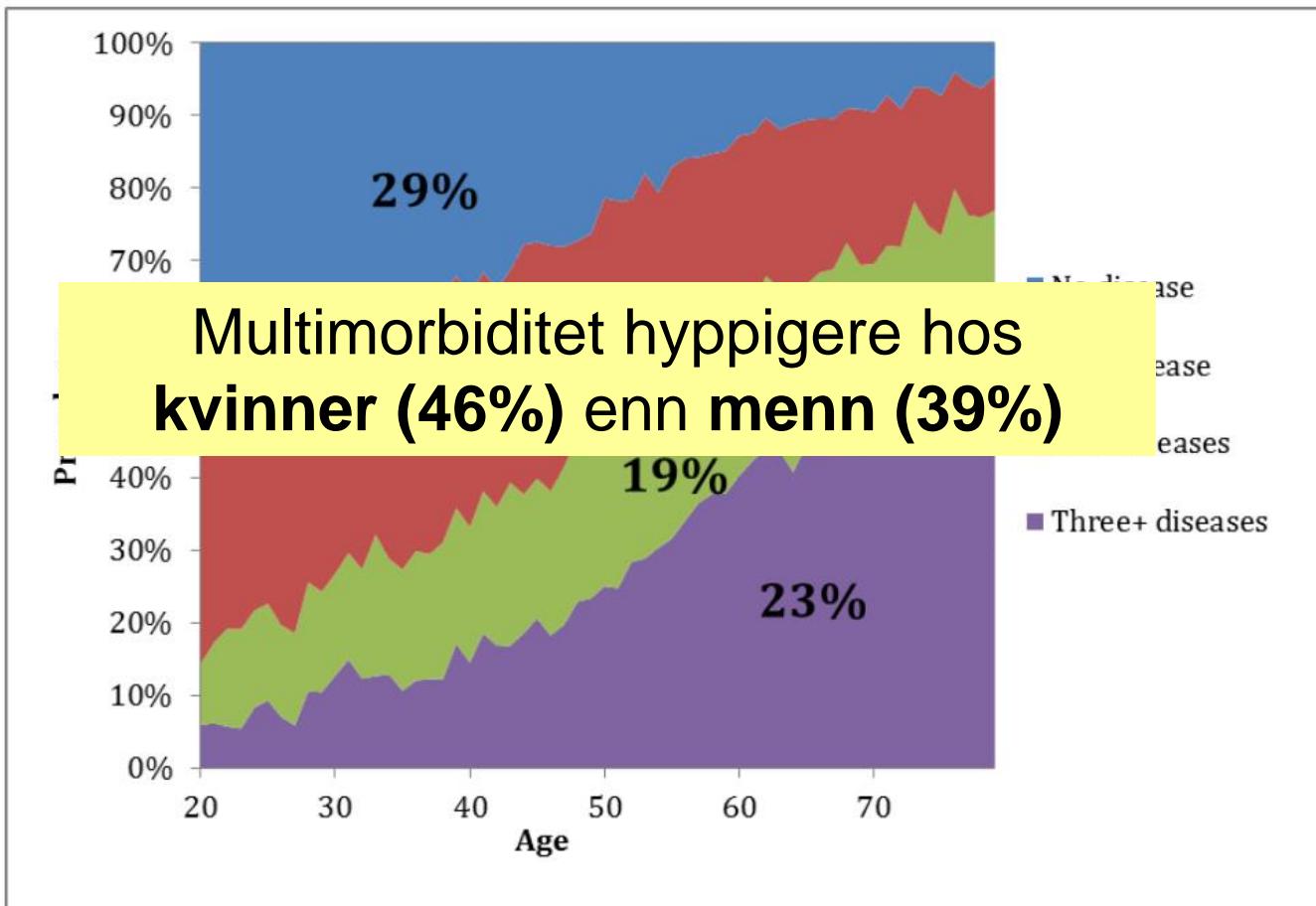
PMID: 29981095 DOI: [10.17269/s41997-018-0069-y](https://doi.org/10.17269/s41997-018-0069-y)



# Prevalens HUNT 3

(ca 48000 deltagere 29-79 år)

**Figure 1 Age distribution and prevalence of multimorbid diseases/conditions among participants aged 20-79 years in the HUNT 3 Study. Bold prevalence numbers are age standardized**



**Figure 2 Prevalence of mental health problems in relation to increasing number of somatic health conditions in the age groups 20-39 years; 40-59 years; and 60-79 years in the HUNT 3 study**