Quiz Review and Instructor Availability

- Will be able to review quizzes in class (Thursday) while working on the in-class assignment
- ALSO, I will be available all day tomorrow (1000am-4pm) if you would like to drop in and review quiz/discuss the course (Markin Hall 3047)

iClickers still unregistered

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Class 26:

Social Epidemiology

HLSC 2003

WALKER

FACULTY OF HEALTH SCIENCES

UNIVERSITY OF LETHBRIDGE

Class 22: Learning Objectives

1. Define social epidemiology and the social determinants of health
2. Have a general understanding of key social determinants of health
3. Have a general understanding ***how*** social determinants impact the health of populations via key pathways

Health Outcomes Determined by:

- Genetics (biology)
- Behaviours
- Environment

Or often a combination of the three, and not easily separated.

“In so far as people are simultaneously social and biological organisms, is any biological process ever expressed devoid of the social context?”

N. KRIEGER (2001). A GLOSSARY FOR SOCIAL EPIDEMIOLOGY.

Social Epidemiology Definition:

Social Epidemiology is the branch of epidemiology that studies the **social distribution** and **social determinants** of health.

Research indicates the factors that have the greatest impact on health are **the conditions in which we live**.

Social determinants of health (SDOH): the **conditions** in which people are **born, grow, live, work and age, as well as the social position held within society**.

Some Key Social Determinants of Health

INDIVIDUAL-LEVEL

- Income
- Education
- Employment
- Housing
- Food insecurity
- Early childhood experiences
- Racial discrimination
- Social support

GROUP-LEVEL

- Gross Domestic Product
- Economic Recession
- Unemployment level
- Income inequality
- Neighbourhood quality
- Social capital

History of Social Epidemiology

Although relatively new in its definition (1950s), not actually a new concept…

1845 – "All conceivable evils are heaped upon the poor…they are given damp dwellings…they are supplied bad, tattered, or rotten clothing, adulterated and indigestible food…they are exposed to the most exciting changes of mental condition, the mot violent vibrations between hope and fear…deprived of all enjoyments except sexual indulgence and drunkenness and are worked every day to the point of complete exhaustion of they mental and physical energies." (Engels)

1848 – "Medicine is a social science and politics is just medicine on a large scale…Do we not always find the disease of the populace traceable to defects in society?" (Virchow)

1897 – Research which attempted to show that, although suicide may result from individual acts, the incidence of suicide determined by society (Durkheim)

Social Gradient in Health

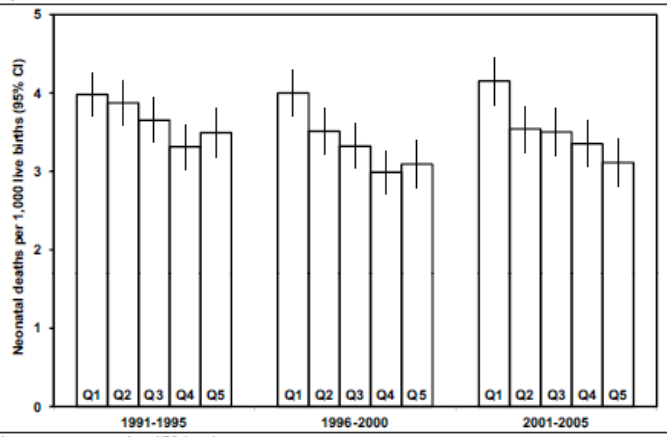
Lower social classes are more likely to suffer from a variety of disease and are more likely to die younger than those in the social classes above them (this is not just confined to those in poverty, but runs from top to bottom in the social hierarchy – position within the hierarchy is important)

THE HIGHER THE SOCIAL POSITION,

THE BETTER THE HEALTH

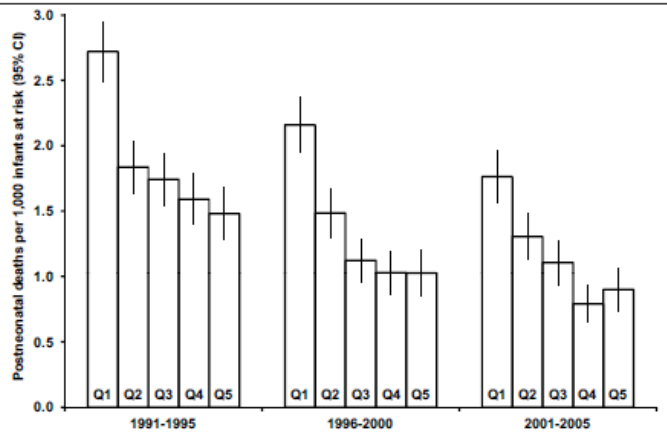
Neighbourhood Income and Neonatal, Post neonatal, and SIDS Mortality in Canada, 1991-2005 (2013) LINK to Reference

Figure 1. Neonatal mortality in urban areas of Canada (excluding Ontario and territories) by period and neighbourhood income quintile



Q1 represents the poorest income quintile and Q5 the richest.

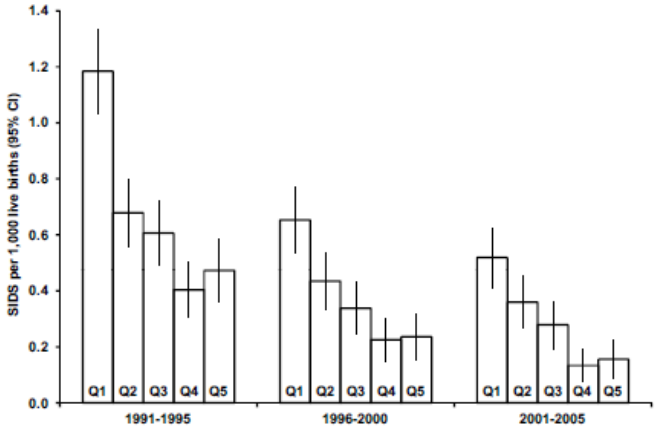
Figure 2. Postneonatal mortality in urban areas of Canada (excluding Ontario and territories) by period and neighbourhood income quintile



Q1 represents the poorest income quintile and Q5 the richest.

ured using neighbourhood income, have narrowed in Canada over a 15-year period but have not disappeared. However, all income quintiles experienced a decline in infant mortality. Unlike previous studies,[6,7] the association between neighbourhood income and infant mortality in our study remained statistically significant after adjustment for covariates, most likely due to the greater statistical power of our larger dataset.

Figure 3. Rate of SIDS in urban areas of Canada (excluding Ontario and territories) by period and neighbourhood income



Q1 represents the poorest income quintile and Q5 the richest.

Reasons why low socio-economic status is associated with infant mortality need further investigation, but higher prevalence of risk factors associated with infant death is likely involved. In Canada, mothers who do not complete high school have significantly higher rates of smoking and alcohol use during pregnancy and lower rates of breastfeeding than women with higher levels of education.[9] In addition, lower maternal education is associated with higher risks of preterm[18] and small-for-gestational-age birth.[10] These inequalities in outcomes were stable or increased over time.[18,19]

In the poorest neighbourhood income quintile, aHRs were higher for SIDS than for neonatal and overall postneonatal death, indicating that socio-economic gradients in relative risks are more important for SIDS than for other types of infant death. The high hazard ratio of SIDS for lower socio-economic strata is consistent with observations from the United Kingdom, where both unemployment and area deprivation were associated with a higher risk of SIDS.[20,21] In Canada, the proportion of mothers placing their infants on their back to sleep (the safest position for SIDS prevention)[22] increases with higher maternal education.[23] Maternal smoking during pregnancy and absence of breastfeeding vary by socio-economic status and are also known to be risk factors for SIDS.[24,25]

As shown in a previous study,[10] changes in the coding of cause of death are unlikely to have substantially affected SIDS counts.

This study has limitations. We did not have individual-level data on income, education and employment, or other potentially relevant characteristics such as smoking. Neighbourhood income was assigned using postal codes and non-differential misclassification errors in the attribution of neighbourhoods may have attenuated the results, especially in small town and rural areas.[26] Neighbourhoods were assigned at time of birth, which may not reflect true residential exposures if mothers had moved either during or after pregnancy. We could not adjust for clustering of births within mothers. Finally, neighbourhood income quintile is only one measure of socio-economic status; we do not know if trends would have been different for other markers of area socio-economic status.

In conclusion, our study shows that despite a decrease in rates of infant mortality and SIDS across all socio-economic strata over time, socio-economic inequalities in those rates have persisted in Canada. This finding highlights the need for effective infant health promotion strategies in vulnerable populations.

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LINK to Article

Table 1

Age-standardized mortality rates (ASMRs) per 100,000 person-years at risk, rate ratios (RRs) and rate differences (RDs), all causes of death, by sex and income adequacy quintile, non-institutional cohort members aged 25 or older at baseline, Canada, 1991 to 2006

Sex and income quintile	Deaths	ASMR	95% confidence Interval		RR	95% confidence Interval		RD	95% confidence Interval	
			from	to		from	to		from	to
Men										
Quintile 1 (lowest)	52,828	1,850.2	1,834.3	1,866.3	1.67*	1.65	1.70	744.4*	724.3	764.5
Quintile 2	62,137	1,494.4	1,482.3	1,506.6	1.35*	1.33	1.37	388.5*	371.3	405.7
Quintile 3	45,962	1,337.7	1,325.1	1,350.3	1.21*	1.19	1.23	231.8*	214.3	249.3
Quintile 4	40,279	1,236.2	1,223.1	1,249.4	1.12*	1.10	1.14	130.3*	112.4	148.2
Quintile 5 (highest)†	39,781	1,105.9	1,093.8	1,118.1	1.00	...	...	0.0	...	...
Women										
Quintile 1 (lowest)	65,032	1,103.7	1,094.1	1,113.4	1.52*	1.50	1.55	378.4*	364.7	392.1
Quintile 2	43,996	907.8	898.9	916.7	1.25*	1.23	1.27	182.5*	169.3	195.6
Quintile 3	29,015	826.6	817.1	836.2	1.14*	1.12	1.16	101.3*	87.6	114.9
Quintile 4	24,411	772.9	763.1	782.9	1.07*	1.12	1.09	47.6*	33.7	61.5
Quintile 5 (highest)†	23,538	725.3	715.7	735.1	1.00	...	...	0.0	...	...

† reference group (RR = 1.00 and RD = 0.0)

* significantly different from quintile 5 ($p < 0.05$)

... not applicable;

Note: Reference population (person-years at risk) for age-standardization was taken from internal cohort age distribution (5-year age group).

Source: 1991 to 2006 Canadian census mortality and cancer follow-up study.

Hamilton, ON. (2010)

Staggering inequalities found…a gap in 21 years between rich and poor neighbourhoods…"if that second neighbourhood were a country, it would rank 165th in the world, alongside Nepal and Below Mongolia…Right here in Hamilton we actually have Third World life expectancy."

(Canadian Medical Association, 2013)

LINK

MON, OCT 26, 2015 | 9 °C
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READERS' CHOICE
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Home > News > Worlds apart

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Aug 25, 2010 | Vote 0 0

Worlds apart

Hamilton Spectator

Two neighbourhoods, separated by just five kilometres as the crow flies. They might as well be worlds apart.

See neighbourhood interactive map

Between these Hamilton neighbourhoods, representing two ends of the spectrum, there's a difference of 21 years in average age at death.

Basically, it's a crude measure of life expectancy across this city, which is important because there's no clearer measure of health than whether you're dead or alive.

Of all the glaring disparities between Hamilton's neighbourhoods uncovered in this groundbreaking health mapping project, the gap in life expectancy is the most disturbing. That 21-year difference represents an entire generation lost between those neighbourhoods at the top and bottom.

At one extreme is the west Mountain neighbourhood bounded by Upper Paradise Road, the Mountain brow, Rice Avenue and Mohawk Road West, where the average age at death was 86.3 years, based on death statistics collected from 2006 to 2008. That's five years better than Canada's average life expectancy.

At the other extreme is the neighbourhood tucked between Wellington Street North, the harbourfront, Sherman Avenue North and the railway tracks near Barton Street, where the average age at death for the same period was 65.5 years.

*By what **pathways** do the Social Determinants influence Health Phenomena?*

1. Material Deprivation Pathway

2. Biologic (Psychosocial) **Pathway**

3. Life course Pathways

3. Epigenetic Pathways

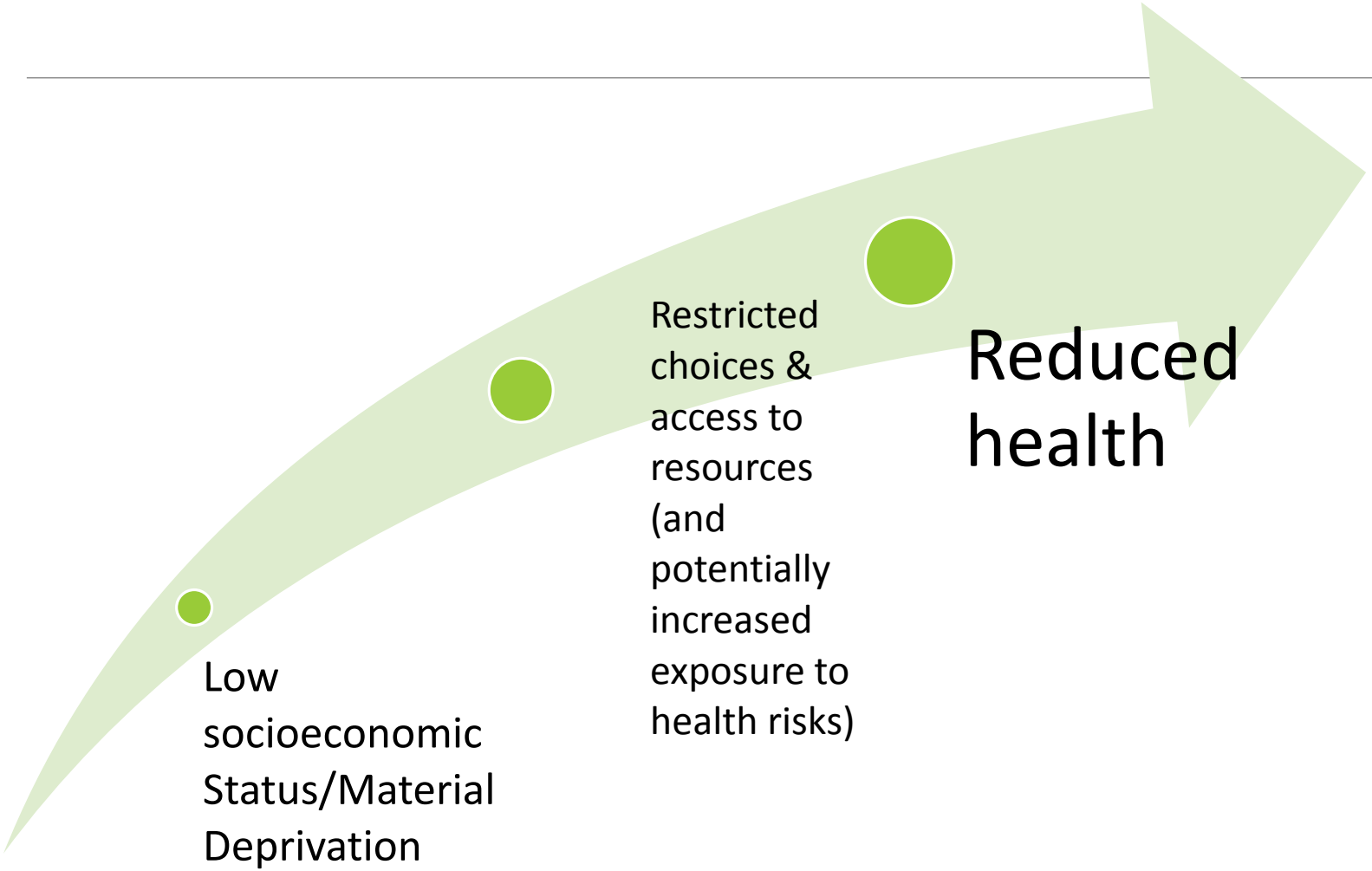
(discussed in previous class)

1. Material Deprivation Pathway

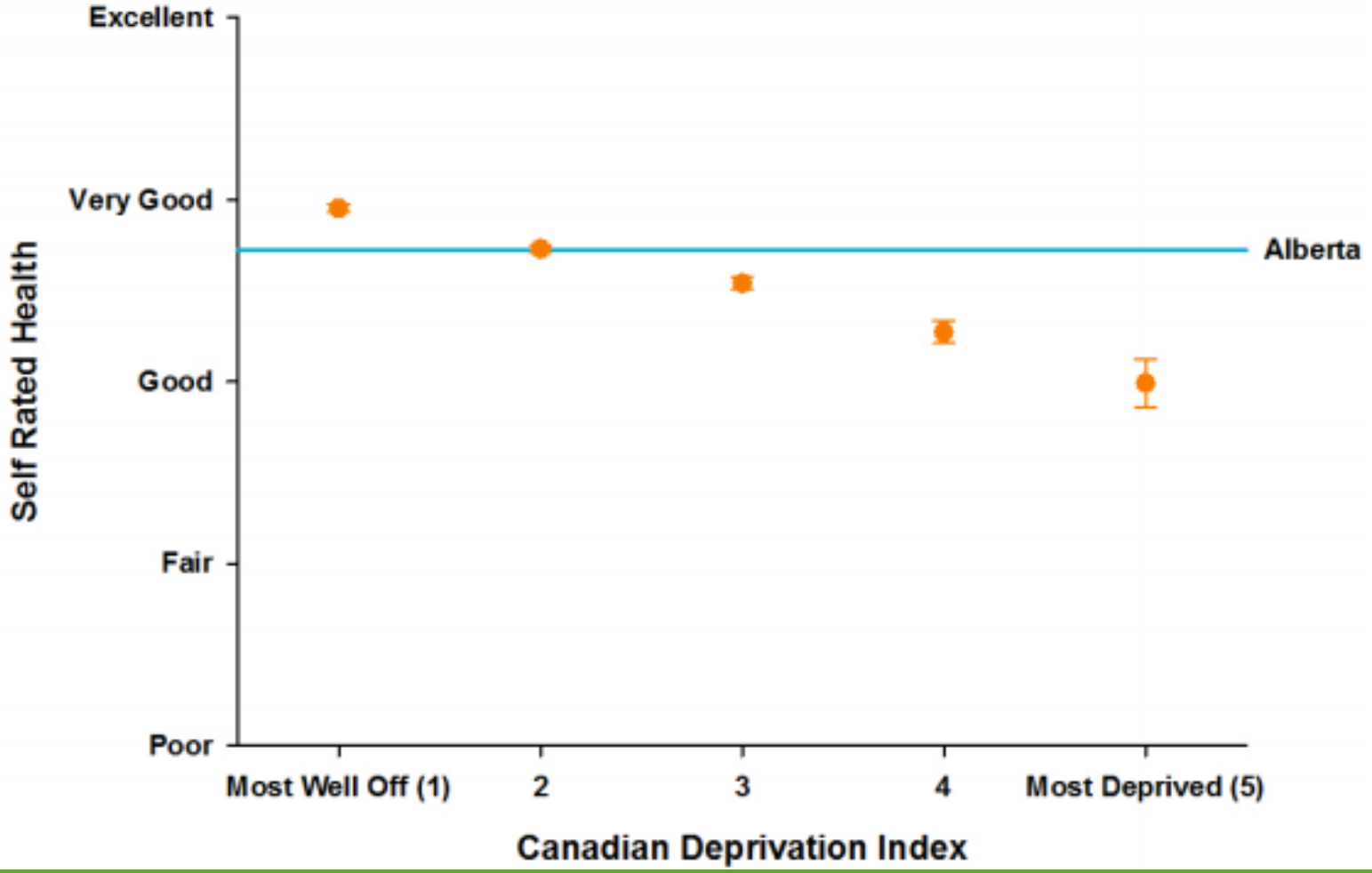
1. Material Deprivation Pathway

- The economic structuring of society controls individual access to material resources
- People experience material deprivation when they do not have the resources or conditions of life to fully participate within society.
- Even though basic needs may be met, each step up the income ladder brings potential material benefits which can produce gains in health (and positively influence social position)
- Social inequalities can best be remedied by **a more equitable distribution of economic resources** across individuals in a society.

1. Material Deprivation Pathway



Material Deprivation and Self-Rated Health in Alberta, 2015



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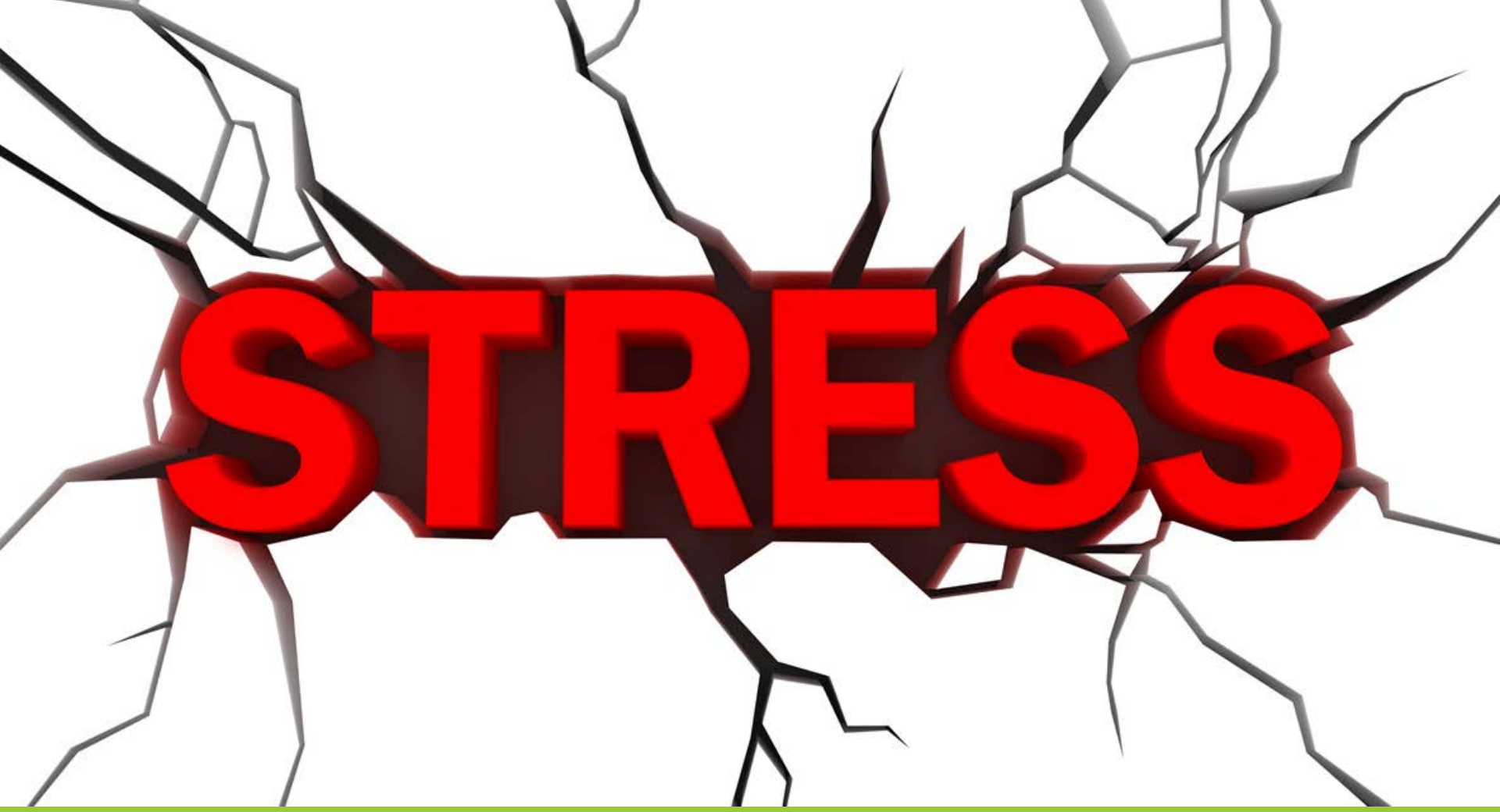


2. Biologic psychosocial Pathway

Biologic (Psychosocial) Pathway

- People who suffer from adverse living conditions experience high levels of stress.
- At the psychological level, unpredictable and uncontrollable stress can cause feelings of anxiety, shame and worthlessness that make coping difficult.
- People may attempt to cope with these pressures through unhealthy coping behaviours which may bring momentary relief.
- Stress also results in physiological and psychological responses that affect health.
- This stress can result from the comparison or discrimination (imposed or personal) of ones position relative to social hierarchy

2. Biologic (psychosocial) Pathway Con't…

Examines how human bodies respond to, and are altered by social conditions over time.

There are various explanations for how biological pathways can change or influence the risk of disease (i.e. Biologic Plausibility)

Embodiment – a concept referring to how we literally incorporate and reflect biologically, the social world in which we live from birth to death (Krieger, 2001).

- In other words, how social factors "**get under the skin**" to influence our health?

Chronic and Acute Social Stressors:

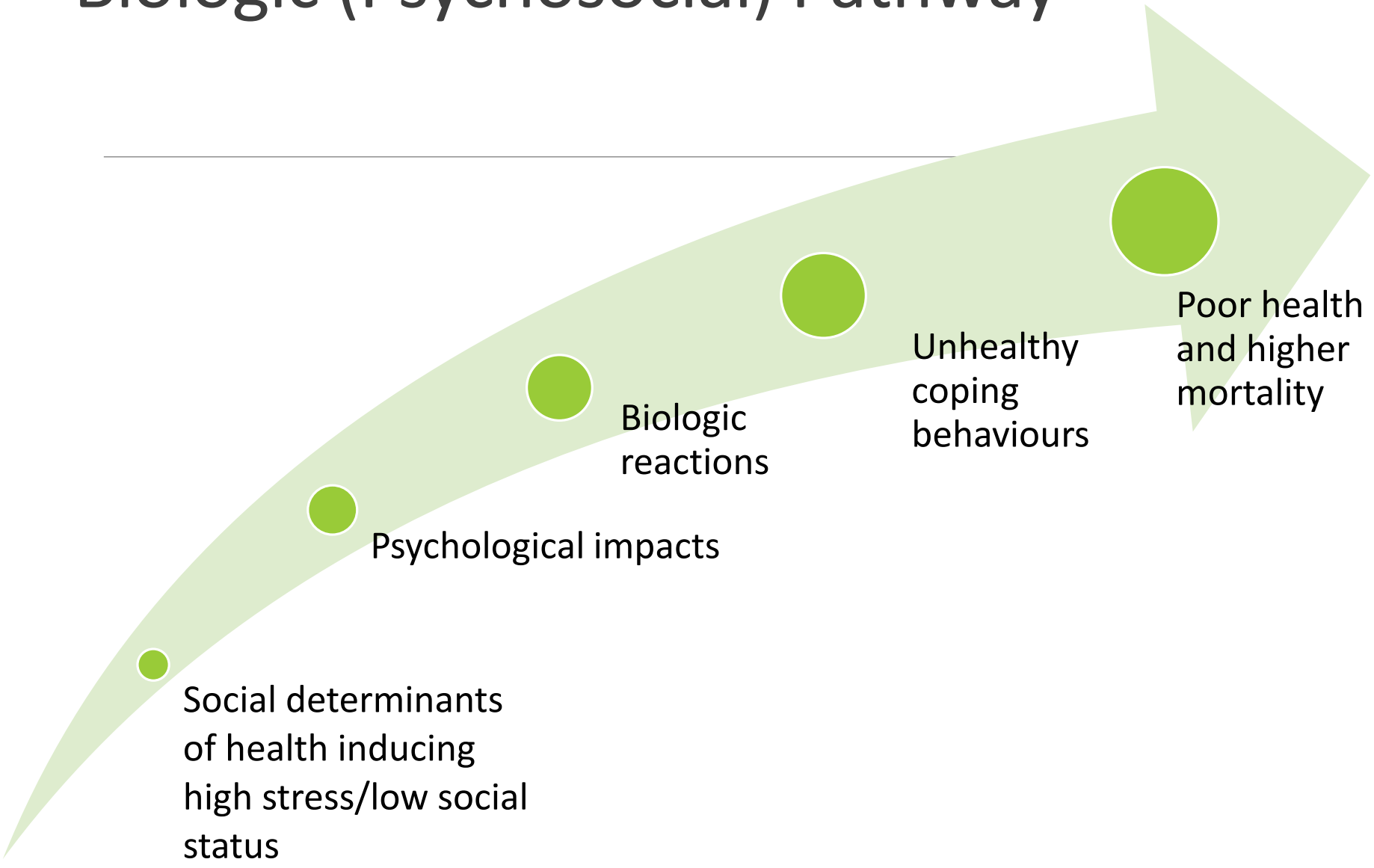
- 1. Alter host susceptibility or are, independently, pathogenic because of their affects on biologic function

AND/OR

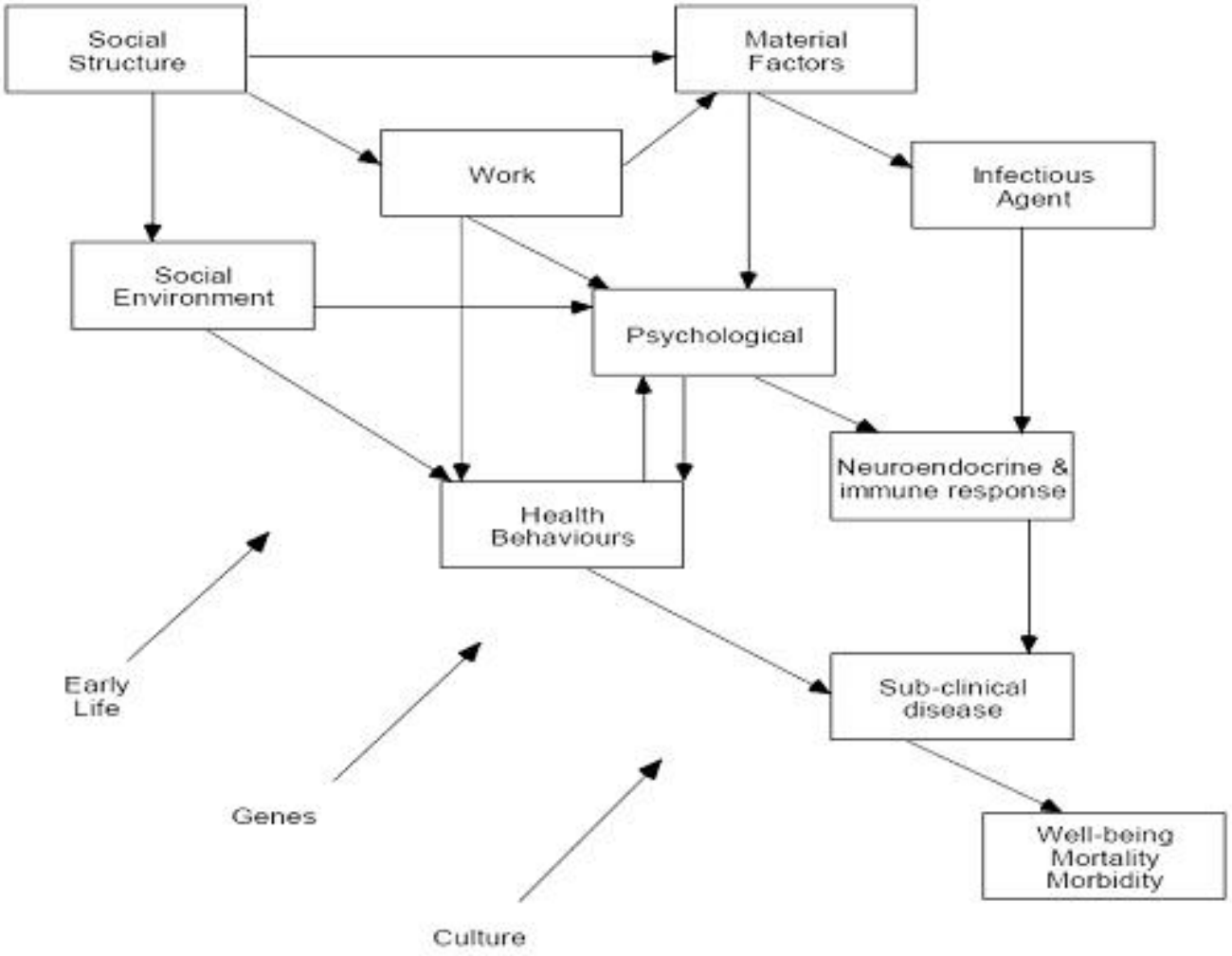
- 2. Induce/Influence health damaging behaviours (diet, sexual engagement, use of psychoactive substances)

-Krieger (2001). A glossary for Social Epidemiology.

Biologic (Psychosocial) Pathway



Brunner and Marmot, 2004

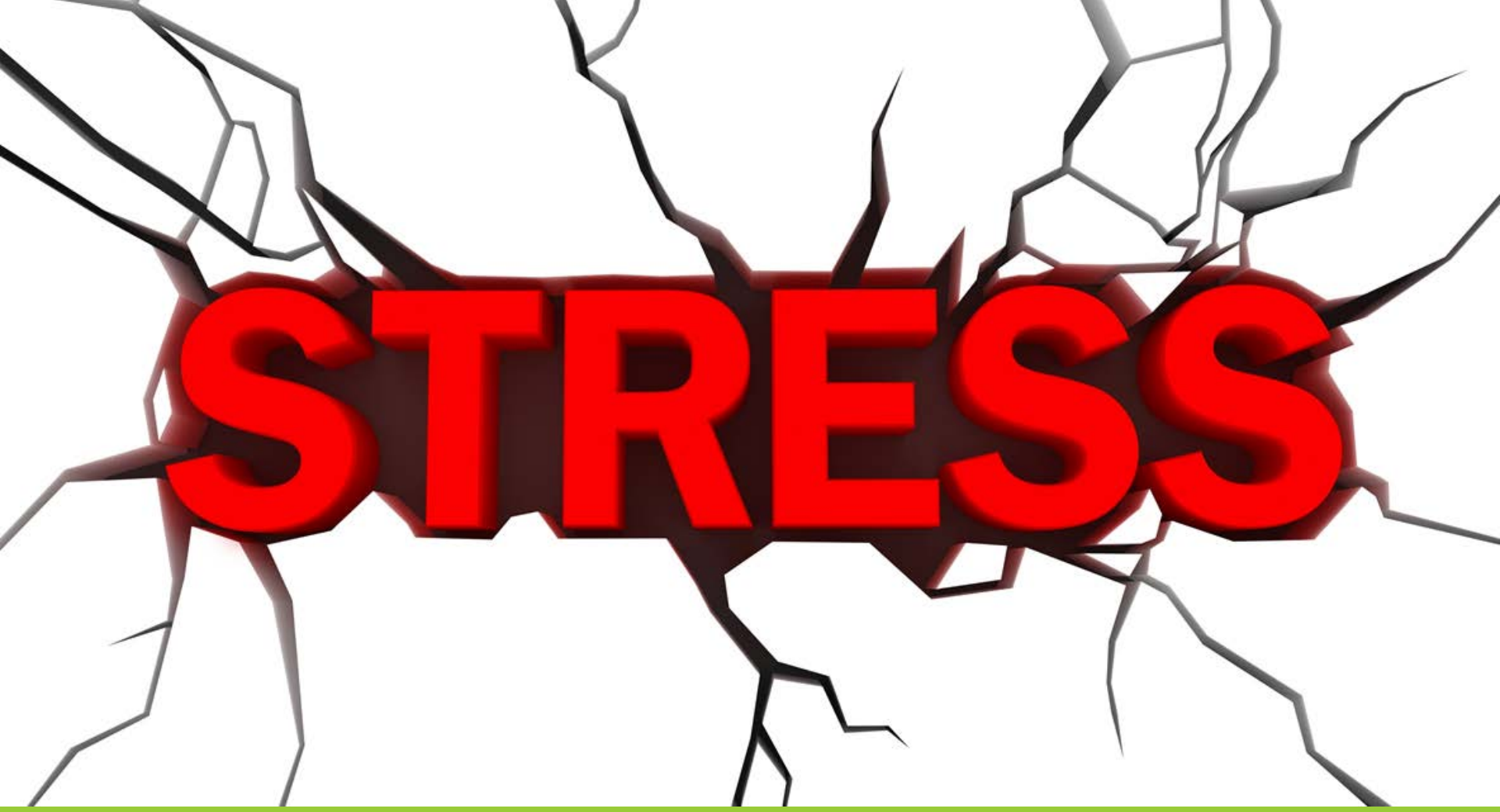


E. Brunner and M. Marmot. (2006). Social organization, stress, and health. In M. Marmot and R.G. Wilkinson (eds) *Social Determinants of Health*. Oxford: Oxford University Press, pp. 6-30.

Is it "biologically plausible" that social determinants could cause poor health? *Do they?*

ASSOCIATION VS. CAUSATION

READING: BRUNNER AND MARMOT, 2006 (POSTED ON MOODLE)



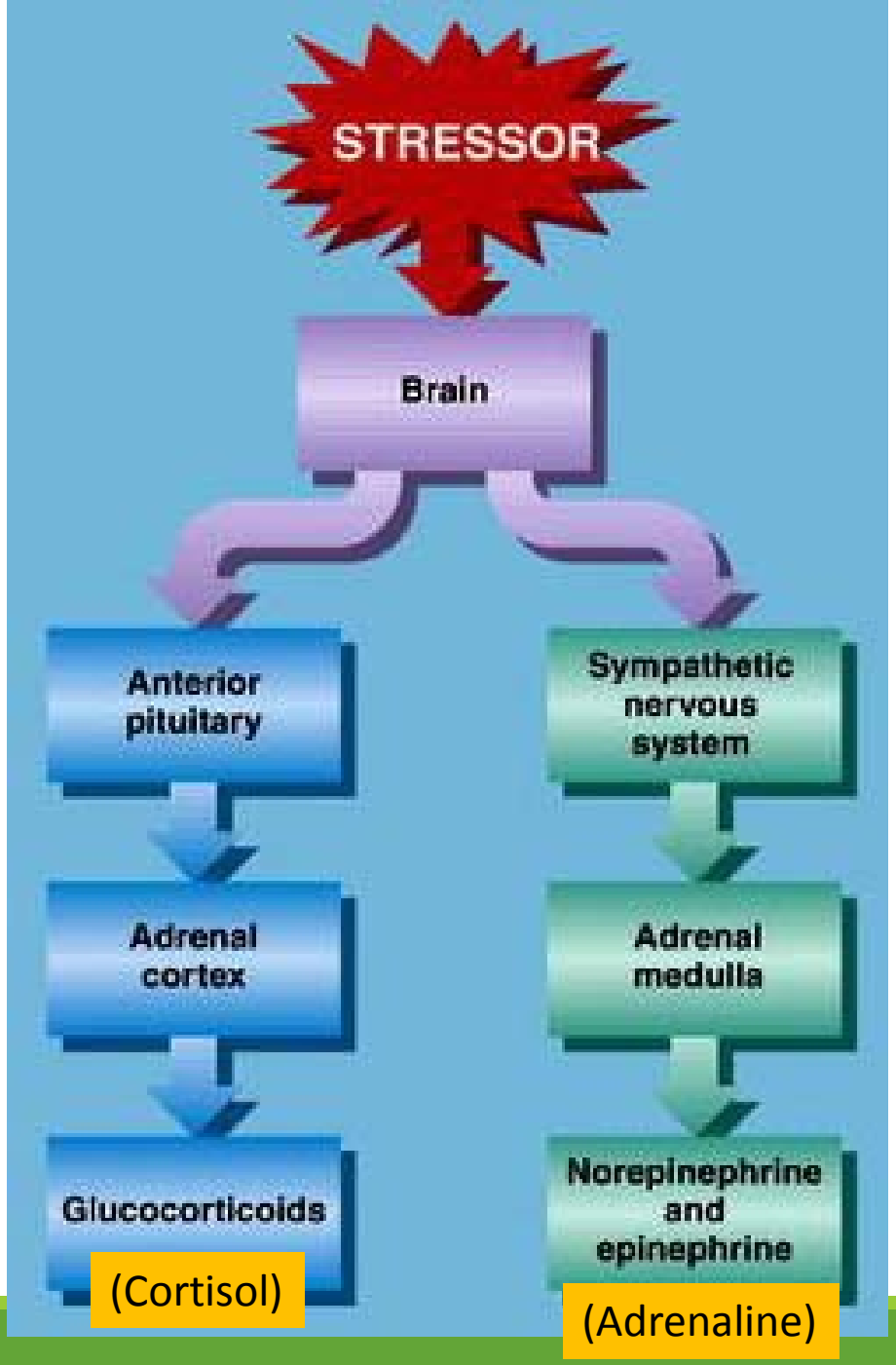
Is stress bad?

The workhorses of the Stress Response:

1. Sympathetic Nervous System

2. Hypothalamic-Pituitary-Adrenal (HPA) Axis

Impacts of toxic stress on the developing brain



Sympathetic Nervous System and Acute Stress

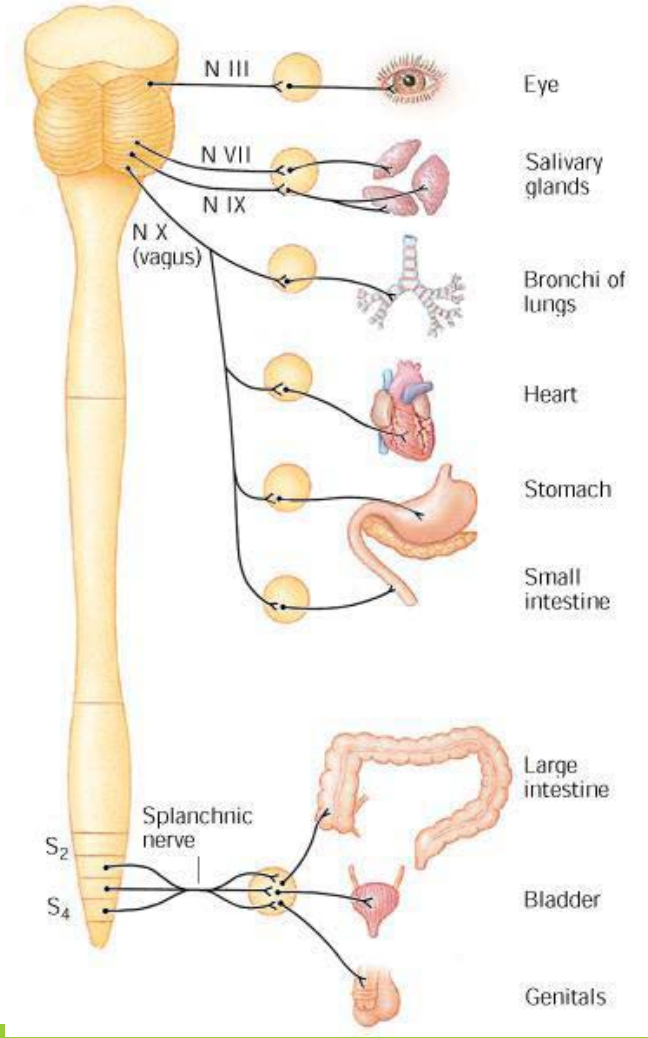
Nerve endings exit spine and branch out to all parts of body

Jumps into action in an emergency

Release epinephrine and norepinephrine in milliseconds – fight or flight

- Heart rate and blood pressure up
- Increased energy availability
- Inhibition of functions not necessary for immediate survival

Can be triggered and stopped rapidly; the effects are short-lived (minutes).

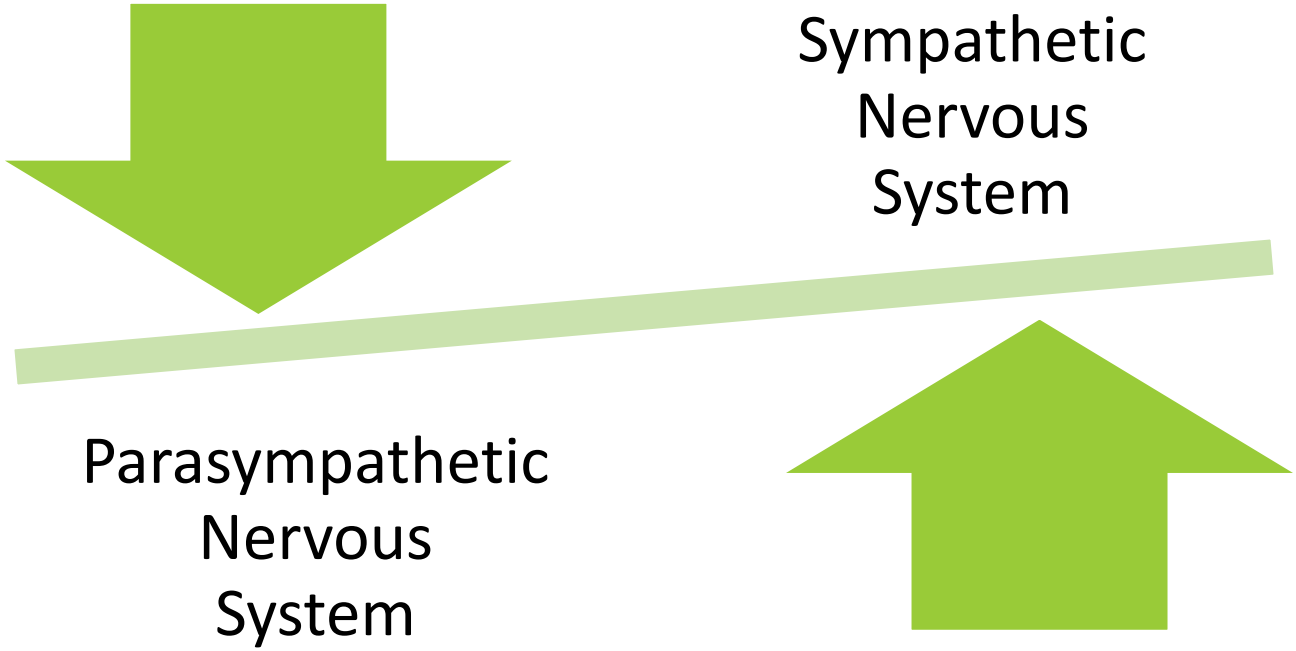


Acute vs. Chronic Stress

We evolved to deal with stressors like this

But today we deal with stressors like this

Autonomic Nervous System and Chronic Stress



If threat is chronic, body has trouble shutting sympathetic nervous system down and returning to homeostasis

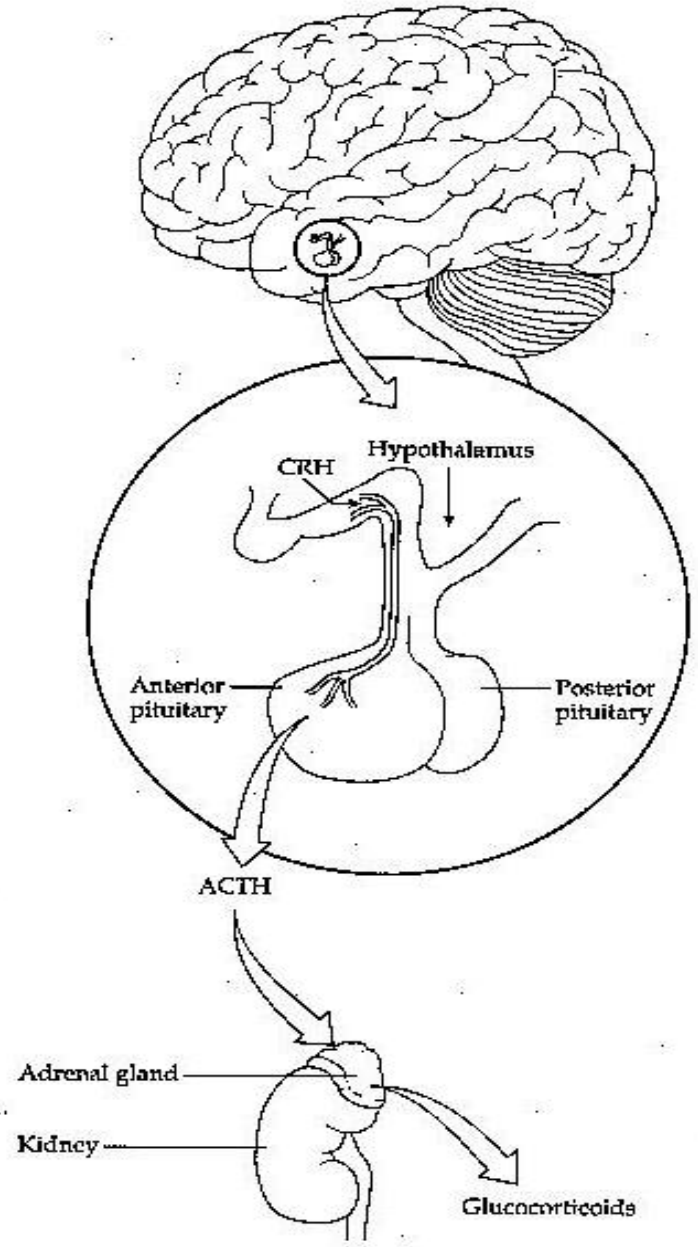
HPA Axis

A Stressful Event Happens

Hypothalamus releases corticotropin-releasing hormone (CRH) to get the ball rolling

15 seconds - Pituitary releases corticotropin (ACTH) into blood

Minutes later the adrenal gland releases cortisol into bloodstream



HPA Axis & Chronic Stress

Adrenal Cortex - 15 minute sustained release of cortisol has a half-life of **100 minutes** in the blood.

Prolonged activation causes the adrenal cortex to increase in size to cope with a greater need for cortisol production.

Cortisol production takes priority over all other hormones!

- Impairs cognitive performance
- Blood sugar / hormone imbalances
- Decreased bone density / muscle tissue
- Chronic high blood pressure
- Lowered immunity, slow wound healing

Impact of Chronic Stress on the Body

Acute Impact

Heart rate, blood pressure increase

Immune system suppressed

Storage of energy stops– insulin resistance

Digestion, elimination process stops

Chronic Impact

Increased risk of heart attacks, strokes

Lowered resistance to infection

Diabetes

GI problems – eg: irritable bowel syndrome

Depression

Epidemiologic research attempting to find and explain biologic and behavioural responses to social stressors: racism and discrimination

BIOLOGIC PLAUSIBILITY

LINK to article

The Relationship of Internalized Racism to Body Fat Distribution and Insulin Resistance among African Adolescent Youth

Earle C. Chambers, PhD; Eugene S. Tull, DrPh, MT; Henry S. Fraser, MBBS, PhD; Nyasha R. Mutunhu, BA; Natasha Sobers, MBBS; and Elisa Niles, BA

New York, New York; Pittsburgh, Pennsylvania; and Barbados, West Indies

This study examined the relationship of internalized racism (INR) and hostility to body fat distribution and insulin resistance in black adolescent children age 14–16 years on the Caribbean island of Barbados. Questionnaire data on psychosocial variables and anthropometric measurements, together with a fasting blood sample, were obtained from 53 low-birthweight and 119 normal-birthweight adolescents. Insulin resistance was calculated using the homeostasis model assessment (HOMA). Spearman correlation analyses showed that both INR ($r=0.244$) and hostility ($r=0.204$) were significantly ($p<0.05$) correlated with waist circumference in girls but not boys. Among girls, age- and birthweight-adjusted mean levels of BMI and waist circumference were greater for those with high levels of INR and hostility compared to those with low levels of both variables. In multiple logistic regression analyses, a high INR remained independently associated [odds ratio=3.30 (95%CI=1.30-8.36); $p=0.012$] with having an elevated HOMA value in models that included age, income, birthweight, hostility, physical activity and family history of diabetes. The results of the current study show that the positive relationship between INR and metabolic health risk seen in African-Caribbean adults also exists in African Caribbean adolescent youth independent of birthweight.

INTRODUCTION

Epidemiological studies have shown that higher levels of psychological distress, measured by indicators, such as anger, anxiety and hostility, are associated with abdominal obesity and an increased risk of the metabolic syndrome.[1-3] It has been proposed that the link between psychological distress and metabolic abnormalities involves the dysfunction of the hypothalamic pituitary adrenal (HPA) axis with cortisol dysregulation and consequent abdominal obesity.[4] A chronic defeat response to environmental stressors is thought to be the initiating event in this pathological cascade leading to HPA-axis dysfunction.[4] Recently, it has also been hypothesized that in western-hemisphere blacks, internalized racism (INR)—the degree to which blacks agree with racist stereotypes about blacks—may be a marker of a mindset which predisposes to a defeat response to environmental stressors.[5] In a series of cross-sectional studies,[5-7] a high level of INR was associated with abdominal obesity and glucose intolerance independent of indicators of socioeconomic status and conventional measures of psychological distress. However, these studies were conducted in adults who have a longer exposure to racist constructs than children. Whether INR is associated with metabolic abnormali-

"Research has found **identifiable cellular pathways** by which racial discrimination ***amplifies*** cardiovascular and other age-related disease risks."

LINK to article

Racial Discrimination Is Associated with a Measure of Red Blood Cell Oxidative Stress: A Potential Pathway for Racial Health Disparities

Sarah L. Szanton · Joseph M. Rifkind · Joy G. Mohanty · Edgar R. Miller III · Roland J. Thorpe · Eneka Nagababu · Elissa S. Epel · Alan B. Zonderman · Michele K. Evans

Abstract

Background There are racial health disparities in many conditions for which oxidative stress is hypothesized to be a precursor. These include cardiovascular disease, diabetes, and premature aging. Small clinical studies suggest that psychological stress may increase oxidative stress. However, confirmation of this association in epidemiological studies has been limited by homogenous populations and unmeasured potential confounders.

Purpose We tested the cross-sectional association between self-reported racial discrimination and red blood cell (RBC) oxidative stress in a biracial, socioeconomically heterogeneous population with well-measured confounders.

Methods We performed a cross-sectional analysis of a consecutive series of 629 participants enrolled in the Healthy Aging in Neighborhoods of Diversity across the Life Span (HANDLS) study. Conducted by the National Institute on Aging Intramural Research Program, HANDLS is a prospective epidemiological study of a socioeconomically diverse cohort of 3,721 Whites and African Americans aged 30–64 years. Racial discrimination was based on self-report. RBC oxidative stress was measured by fluorescent heme degradation products. Potential confounders were age, smoking status, obesity, and C-reactive protein.

Results Participants had a mean age of 49 years (SD=9.27). In multivariable linear regression models, racial discrimination was significantly associated with RBC oxidative stress (Beta=0.55, $P<0.05$) after adjustment for age, smoking, C-reactive protein level, and obesity. When stratified by race, discrimination was not associated with RBC oxidative stress in Whites but was associated significantly for African Americans (Beta=0.36, $P<0.05$).

Conclusions These findings suggest that there may be identifiable cellular pathways by which racial discrimination amplifies cardiovascular and other age-related disease risks.

Keywords Health disparities · Racial discrimination · Oxidative stress · Accelerated aging

Abbreviations

RBC Red blood cells
CRP C-reactive protein

Introduction

Discrimination, Racial Bias, and Telomere Length in African-American Men

David H. Chae, ScD, MA, Amani M. Nuru-Jeter, PhD, Nancy E. Adler, PhD, Gene H. Brody, PhD, Jue Lin, PhD, Elizabeth H. Blackburn, PhD, Elissa S. Epel, PhD

Background: Leukocyte telomere length (LTL) is an indicator of general systemic aging, with shorter LTL being associated with several chronic diseases of aging and earlier mortality. Identifying factors related to LTL among African Americans may yield insights into mechanisms underlying racial disparities in health.

Purpose: To test whether the combination of more frequent reports of racial discrimination and holding a greater implicit anti-black racial bias is associated with shorter LTL among African-American men.

Methods: Cross-sectional study of a community sample of 92 African-American men aged between 30 and 50 years. Participants were recruited from February to May 2010. Ordinary least squares regressions were used to examine LTL in kilobase pairs in relation to racial discrimination and implicit racial bias. Data analysis was completed in July 2013.

Results: After controlling for chronologic age and socioeconomic and health-related characteristics, the interaction between racial discrimination and implicit racial bias was significantly associated with LTL (b=−0.10, SE=0.04, p=0.02). Those demonstrating a stronger implicit anti-black bias and reporting higher levels of racial discrimination had the shortest LTL. Household income-to-poverty threshold ratio was also associated with LTL (b=0.05, SE=0.02, $p<0.01$).

Conclusions: Results suggest that multiple levels of racism, including interpersonal experiences of racial discrimination and the internalization of negative racial bias, operate jointly to accelerate biological aging among African-American men. Societal efforts to address racial discrimination in concert with efforts to promote positive in-group racial attitudes may protect against premature biological aging in this population.

(Am J Prev Med 2014;46(2):103–111)

Study Link

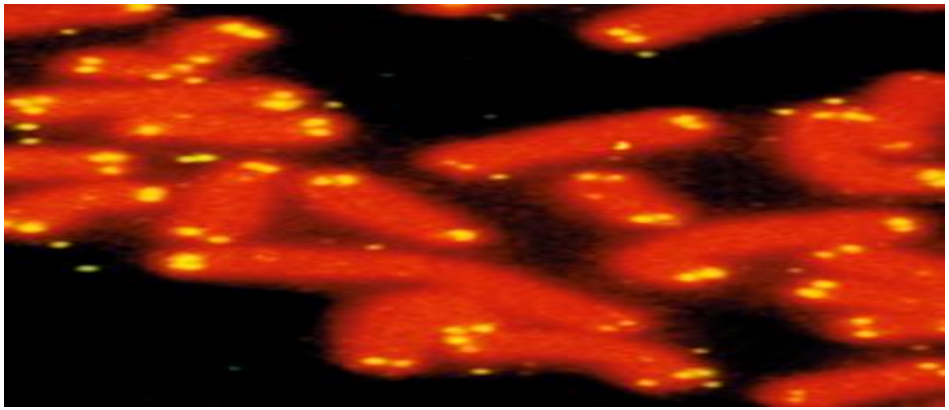
Discrimination Reduces Telomere Length

Findings indicate racial discrimination has pernicious effects on biological aging.

Telomeres highly susceptible to oxidative stress – resulting in their shortening

Shortened telomere length is one pathway through which discrimination may generate greater disease.

Chae et al., 2014



Social dominance in monkeys: dopamine D_2 receptors and cocaine self-administration

Drake Morgan[1], Kathleen A. Grant[1], H. Donald Gage[2], Robert H. Mach[1,2], Jay R. Kaplan[3], Osric Prioleau[1], Susan H. Nader[1], Nancy Buchheimer[2], Richard L. Ehrenkaufer[2] and Michael A. Nader[1,2]

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Disruption of the dopaminergic system has been implicated in the etiology of many pathological conditions, including drug addiction. Here we used positron emission tomography (PET) imaging to study brain dopaminergic function in individually housed and in socially housed cynomolgus macaques ($n = 20$). Whereas the monkeys did not differ during individual housing, social housing increased the amount or availability of dopamine D_2 receptors in dominant monkeys and produced no change in subordinate monkeys. These neurobiological changes had an important behavioral influence as demonstrated by the finding that cocaine functioned as a reinforcer in subordinate but not dominant monkeys. These data demonstrate that alterations in an organism's environment can produce profound biological changes that have important behavioral associations, including vulnerability to cocaine addiction.

Social rank and vulnerability to drug abuse

Michael J. Kuhar

Figure 1. Social dominance in monkeys.
Pictured here is a juvenile rhesus demonstrating the submissive facial expression (silent, with bared teeth) to an approaching dominant animal. Photograph courtesy of Frans de Waal, Living Links Center of Yerkes Primate Center.

A lack of opportunities to achieve personal goals in society will unbalance the brain's reward centres toward whatever rewards are available including reliable, sources such as drugs and alcohol (West, 2006)

"There is unequivocal evidence that social status exerts a profound impact on nonhuman primate physiology and can influence the behavioural effects of dopaminergic drugs" (Czoty, 2005)

The Impact of Internalized Oppression on Health

Major psychological effects

Self doubt, identity confusion

Internalize messages of inferiority about their group at young age - Clark Doll Experiment: Video

Internalized hatred, denial of oppression, self-sabotage

Violence against others in racial group– verbal & physical

"There is a difference between being in an inferior status and being an inferior person, but these differences may be lost in translation; disadvantaged statuses may come to be mirrored in disadvantaged selves."

–Pearlin et al. (2005, p. 208)

"Life circumstances that deny a sense of dignity, increase feelings of insecurity about personal worth and competence, and carry connotations of inferiority in which few people can feel respected, valued and confident will lead to adverse psychosocial states that accumulate over the life course and contribute to increased illness."

-Wilkinson

3. Life Course Pathways

3. The Life Course pathways

A. Latency Model – Exposures during *critical* or *sensitive times can* cause biological changes that will impact disease outcomes, often regardless of later life circumstances.

These exposures may occur prior to birth or in early childhood development (i.e. infection, lack of nutrients, tobacco exposure) and cause changes in biologic function which ultimately leads to disease outcomes later in life.

International Journal of Epidemiology 2002;**31**:1235–1239

FETAL ORIGINS

Fetal origins of adult disease: strength of effects and biological basis

DJP Barker,[a] JG Eriksson,[b] T Forsén[b,c] and C Osmond[a]

Background	Low birthweight has been consistently shown to be associated with coronary heart disease (CHD) and its biological risk factors. The effects of low birthweight are increased by slow infant growth and rapid weight gain in childhood. To quantify the importance of developmental processes in the genesis of CHD it is necessary to establish the impact of fetal, infant and childhood growth on major pathological events in later life—death, hospital treatment and the need for medication.
Methods	Longitudinal study of 13 517 men and women who were born in Helsinki University Hospital during 1924–1944, whose body sizes at birth and during childhood were recorded, and in whom deaths, hospital admissions, and prescription of medication for chronic disease are documented.
Results	The combination of small size at birth and during infancy, followed by accelerated weight gain from age 3 to 11 years, predicts large differences in the cumulative incidence of CHD, type 2 diabetes and hypertension.
Conclusions	Coronary heart disease and type 2 diabetes may originate through two widespread biological phenomena—developmental plasticity and compensatory growth.

Keywords	Fetal growth, childhood growth, type 2 diabetes, hypertension, coronary heart disease
Accepted	11 June 2002

LINK

Birth weight, mid-life hypertension, and late-life brain tissue loss: A life-course approach

Majon Muller, Sigurdur Sigurdsson, Olafur Kjartansson, Ingibjorg Gunnarsdottir, Inga Thorsdottir, Mark van Buchem, Vilmundur Gudnason, Lenore Launer

O5-03-01

DOI: http://dx.doi.org/10.1016/j.jalz.2014.04.488

Abstract | Full Text | Images

Background: Brain tissue loss is not determined in any single time period, but results from the complex interplay of exposures during critical periods throughout life. The "fetal origins of adult disease" hypothesis proposes that an unfavorable intrauterine environment, deduced from small birth-size, may induce permanent changes in fetal brains. These changes in combination with effects of (cardiovascular) exposures during adult life may condition the later risk of brain atrophy. In this study we investigated the combined effect of small birth-size and mid-life hypertension on late-life brain volumes.

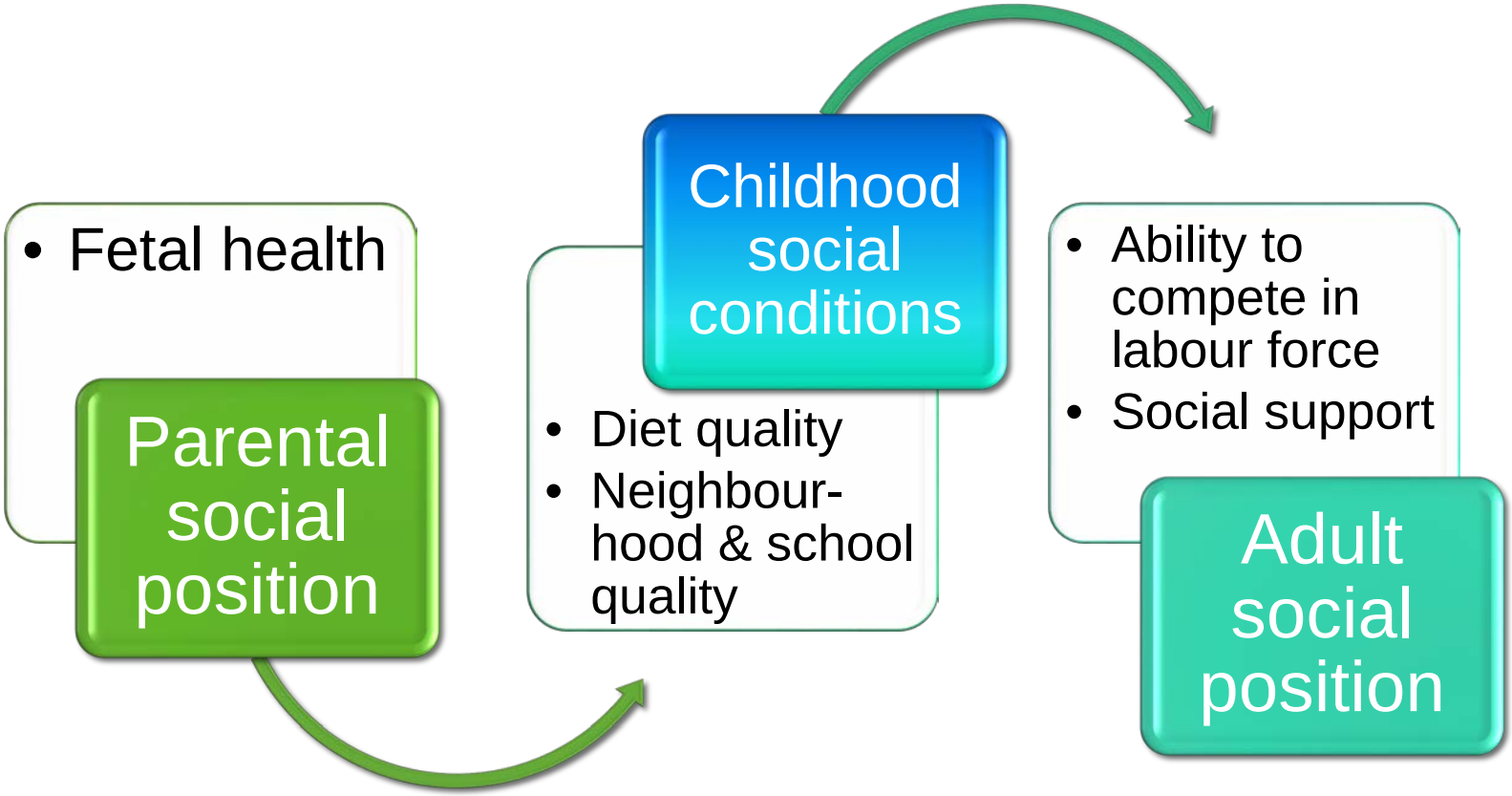
LINK

3. The Life Course pathways

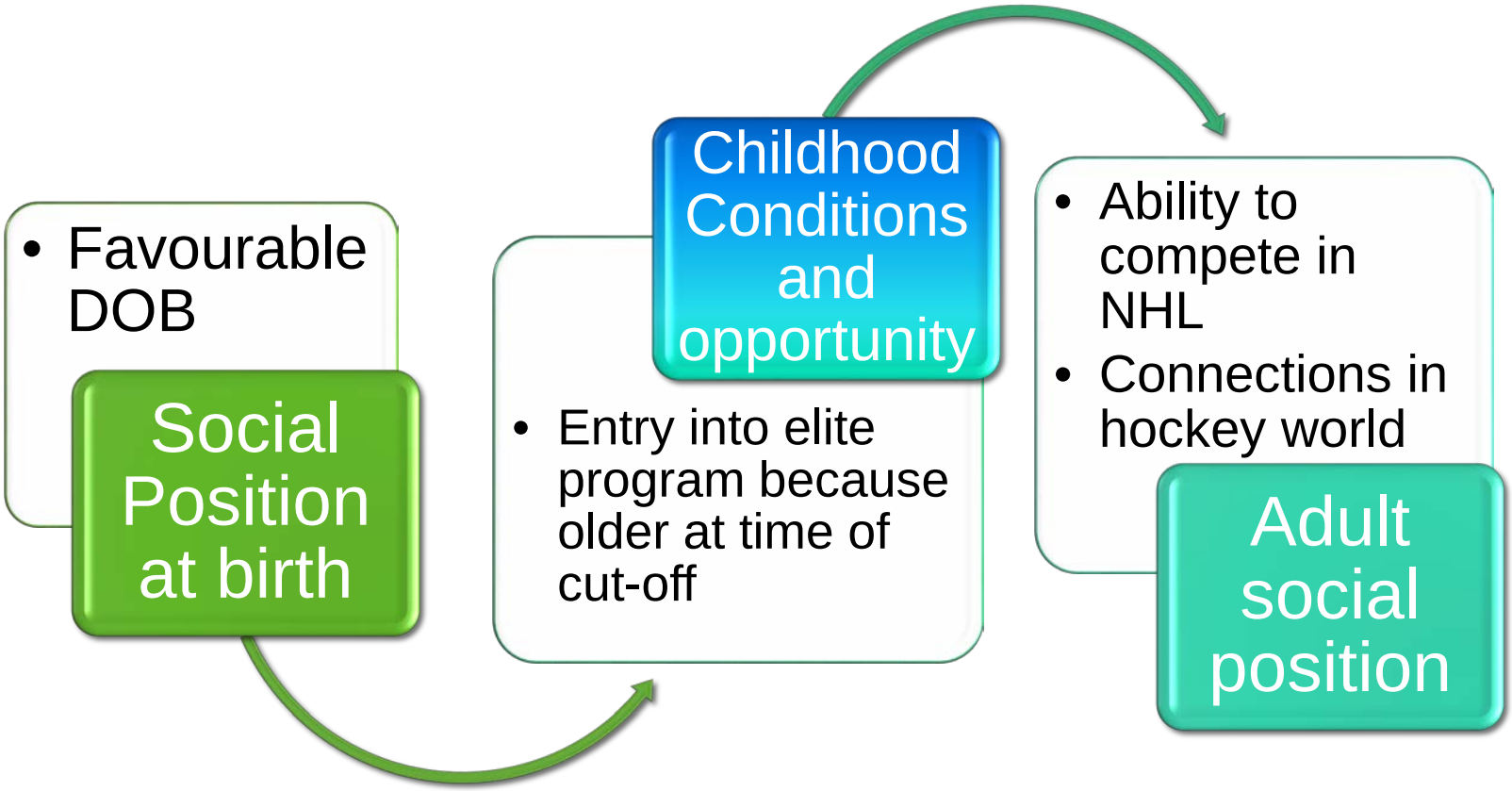
B. Cumulative and Chain of Risk Model(s) –

- The idea that there is a continuum of exposures across the entire life course which accumulate and may cause long term damage or impact other spheres of life.
- Not necessarily any one single factor that has a major impact on health outcomes, but accumulation of multiple or repeated independent risk factors, or a clustering of these factors
- It is a collective culmination of advantage or disadvantage (risks or protective factors) over the entire life course which determine health outcomes
- May exist a chain of advantage or disadvantage (risks or protective factors) over the entire life course which are linked to certain exposures

Cumulative Pathway



Cumulative Pathway (e.g. Chains of Advantage)



An Example: the Great One

Malcolm Gladwell (2006): “How does a skinny, not terribly fast hockey player set records that will probably never be broken?”

In Canada, the best players at age 8-9 are streamed into programs where they practice more, play more games & get better coaching.

The eligibility cut off for elite hockey programs is **January 1**.

But who tends to be the "best" at age 8 or 9?

Birthdate and success in minor hockey: The key to the NHL

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ABSTRACT

Previous research (Barnsley, Thompson, & Barnsley, 1985) demonstrated an extremely strong linear relationship between the month of birth (from January to December) and the likelihood of playing in the National Hockey League and two major developmental Junior hockey leagues. The present study analyzes the birthdates of 7313 hockey players participating in the Edmonton Minor Hockey Association in the 1983-1984 season. The main findings are that players possessing a relative age advantage, i.e., born in the months of January to June, are more likely to participate in minor hockey and are more likely to play for "top tier," or "rep" teams than are players who are born in the months of July to December and thereby are disadvantaged by their relative age. Proposals by which these relative age effects could be reduced are considered.

Barnsley, Thompson, and Barnsley (1985) reported on the month of birth of hockey

Journal of Sports Sciences, June 2005; 23(6): 629–636

Taylor & Francis
Taylor & Francis Group

The relative age effect in youth soccer across Europe

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(Accepted 24 July 2004)

Abstract

The potential asymmetries in the birth-date distributions of youth soccer players across ten European countries (2175 age citations) were considered. First, we examined the birth-dates of players representing national youth teams in international competitions. Second, the birth-dates of players representing professional club teams in international youth tournaments were analysed. Kolmogorov-Smirnov tests were used to assess differences between observed and expected birth-date distributions. Regression analyses were employed to examine the relationship between month of birth and number of players in the different samples. The results showed an over-representation of players born in the first quarter of the selection year (from January to March) for all the national youth selections at the under-15 (U-15), U-16, U-17 and U-18 age categories, as well as for the UEFA U-16 tournaments and Meridian Cup. Players with a greater relative age are more likely to be identified as "talented" because of the likely physical advantages they have over their "younger" peers. Some options for reducing the relative age effect are offered.

Keywords: *Performance, player selection, seasonal variation, talent identification*

LINK

A Proposal to Reduce the Age Discrimination in Canadian Minor Hockey

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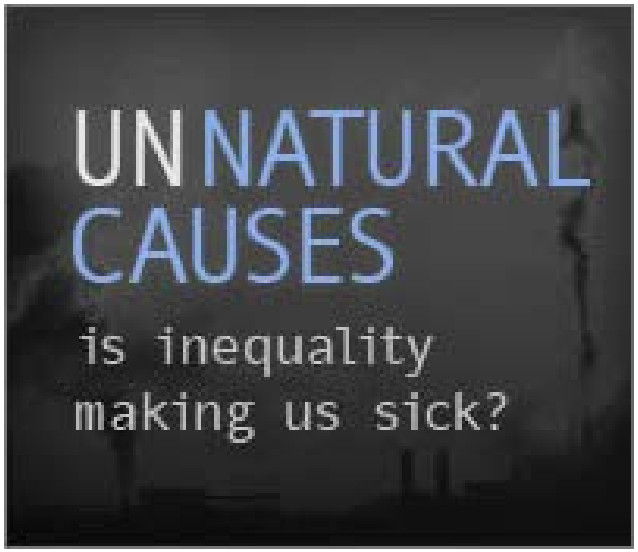
Pour un joueur canadien de hockey mineur, il existe un rapport étroit entre son mois de naissance et les chances qu'il aura de jouer dans une équipe de très haut niveau. Les joueurs nés dans les premiers mois de l'année sont avantagés. On attribue généralement ce fait au système de classification, dans le hockey mineur, qui groupe les joueurs en catégories selon l'âge. Dans cet article, nous commençons par un examen de la situation actuelle. Ensuite, nous montrons que le système de classification basé sur l'âge ne constitue pas une explication suffisante; il faut aussi compter avec une répartition qui s'opère très tôt (c-à-d. la division des joueurs en équipes représentatives et en équipes de ligue-maison.) Nous suggérons un système de classification plus équitable et, finalement, nous examinons les implications de politiques d'intérêt public.

There is strong relationship between birthmonth and the chance that a Canadian minor hockey player will play at an elite level. Players born in the early months of the year have an advantage. This is generally attributed to the slotting system: the way in which minor hockey groups players into age divisions. In this paper we first review the evidence. We then argue that there is more to the explanation of this relative age effect than just the slotting system; it also depends on early streaming (i.e., the partitioning of players into representative and house league teams). We suggest a more equitable slotting system, and finally, we discuss public policy implications.



Unnatural Causes

WHEN THE BOUGH BREAKS (29 MINUTES)