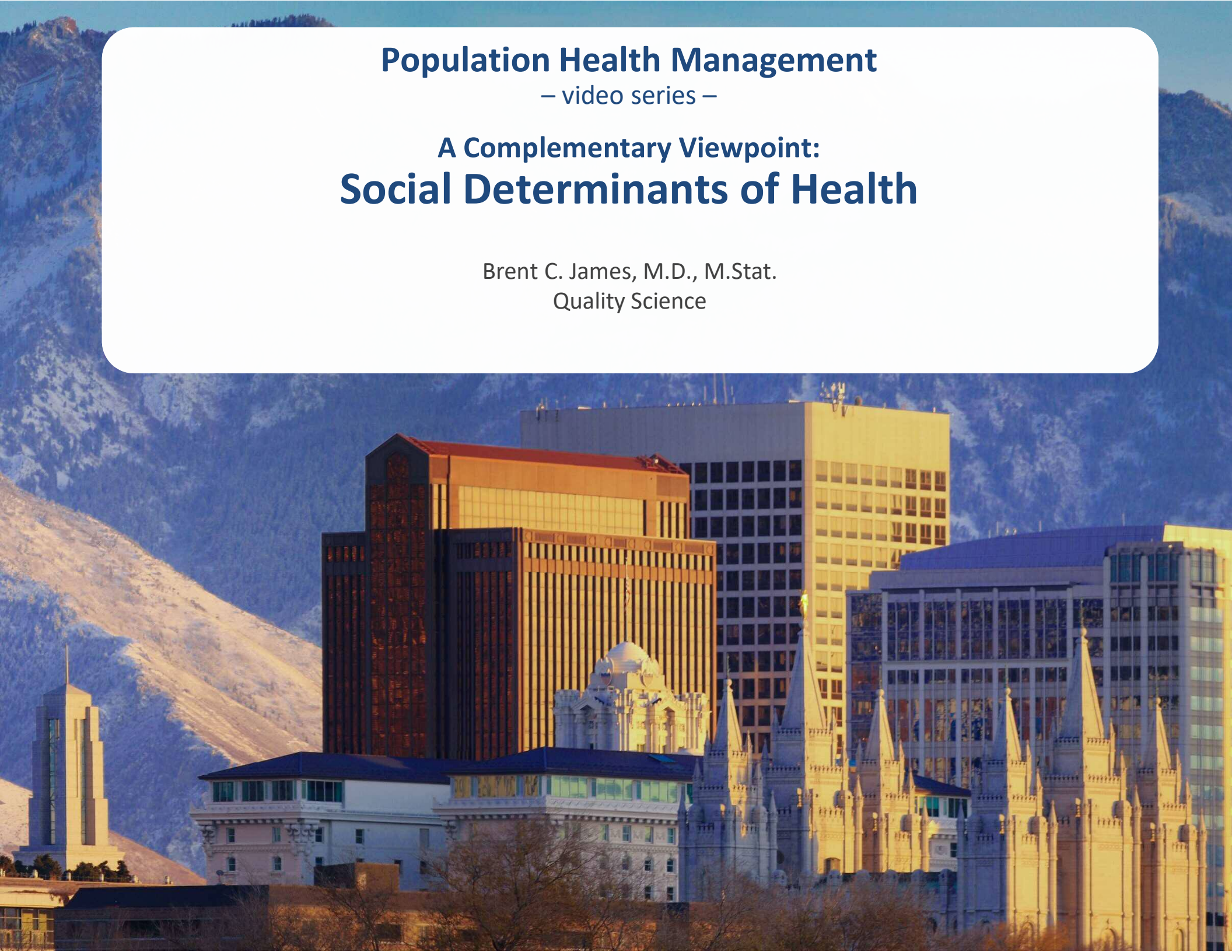
Population Health Management

– video series –

A Complementary Viewpoint:

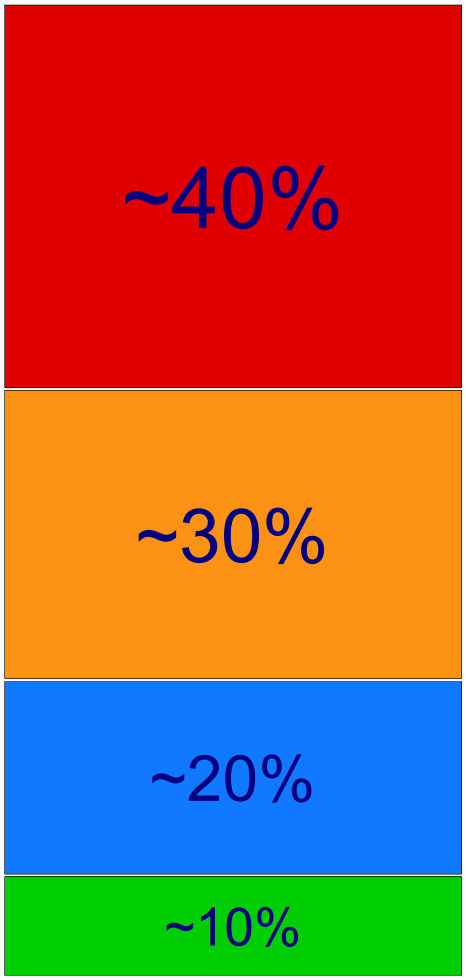
Social Determinants of Health

Brent C. James, M.D., M.Stat.
Quality Science



Video and slides

Health: How long, how well we live



Behavior: ***Tobacco***
Ethanol *(and other recreational drugs)*
Obesity *(diet and exercise)*
Sexually-transmitted disease *(AIDS)*
Unwed pregnancy *(weak support network)*
Suicide, violence, & accidents *(young men)*

Genetics

Physical environment, social environment, public health *(control of epidemic infectious disease through immunization & sanitation)*

Health care delivery *(hospitals and clinics)*

McGinnis JM & Foege WH. Actual causes of death in the United States. *JAMA* 1993; 270(18):2207-12 (Nov 10).

McGinnis JM, Williams-Russo P, & Knickman JR. The case for more active policy attention to health promotion. *Health Affairs* 2002; 21(2):78-93 (Mar).

Kaplan RM & Milstein A. Contributions of health care to longevity: A review of 4 estimation methods. Ann Fam Med 2019; 17(3):267-72 (May/June).

Definition – *social determinants of health:*

1. ***WHO*** – ***The conditions in which people are born, grow, live, work, and age.*** *These circumstances are shaped by the distribution of money, power, and resources at global, national, and local levels.* www.who.int/gender-equity-rights/understanding/sdh-definition/en/

2. ***CDC*** – ***life-enhancing resources, such as food supply, housing, economic and social relationships, transportation, education, and health care,*** *whose distribution across populations effectively determines length and quality of life.* en.wikipedia.org/wiki/Social_determinants_of_health

3 lines of research

1. ***Rabbits*** *with coronary artery disease*

2. ***Human children***
   - *orphanages*
   - *the Adverse Childhood Experiences (ACE) study*
   - *Werner: at-risk children in Kauai* *(longitudinal study, each decade)*

3. ***Social stress***
   - *Michael Marmot: the Status Syndrome*

Study: diet's impact on heart disease

- *Genetically similar line of New Zealand rabbits*
- *Controlled high cholesterol diet (2%)*
- *Tracked blood pressure, heart rate, and atherosclerosis*
  (fatty deposits in the aorta and coronary arteries)
- *Unexpected, very significant outlier result:*
  *one subgroup had **60%** lower levels of atherosclerosis*

- ***Why? What's going on here?***

The difference:

- *All the rabbits with lower rates of atherosclerosis received care from one technician.*
- *She fed the rabbits per protocol, but "she was an unusually kind and caring individual."**
- *As she fed the rabbits (their very unhealthy, high cholesterol diet), "she talked to them, cuddled and petted them."**
- *Subsequent randomized trials confirmed these findings:*

loving care** (high rates of positive contact) **produced a significantly different, much better, health result.

*Harding, Kelli. *The Rabbit Effect.* New York, NY: Atria Books, 2019; pp. xxiii – xxiv.

Image from: Stevenson, Gary E. *Hearts Knit Together.* The Church of Jesus Christ of Latter-day Saints General Conference, Saturday morning session, 3 April 2021.

tive, although the specific fluorescence appeared to be stronger in the P_2 and P_3 segments. Distal tubules and collecting ducts were negative. The glomeruli, except for the parietal layer of Bowman's capsule, were negative. There was a marked reduction in fluorescence from the outer stripe to the inner stripe of the outer medulla (not shown). Low-lying cells, showing a faint fluorescence and probably representing thin limbs of the loop of Henle, were seen in the inner stripe, but all other segments of the nephron in the inner stripe and inner medulla were essentially negative. Minipig kidneys (Fig. 2h) resemble the rat kidneys with one exception: occasional brightly staining cells were seen among essentially negative cells in cortical collecting ducts; these cells may represent the "dark" cells of the pig collecting duct.

We found no differences in the distribution or intensity of specific fluorescence between the lungs of phenobarbital- and saline-treated rats (Fig. 2, c and f). Positive fluorescence was seen in bronchi and bronchioles and in an additional cell type in the lung parenchyma. Minipig lungs (Fig. 2i) showed a similar distribution of NADPH-cytochrome *c* (P-450) reductase.

Our results concerning the distribution of NADPH–cytochrome *c* (P-450) reductase in the liver agree with those of Baron *et al.* (6). To our knowledge, the immonohistochemical demonstration of NADPH–cytochrome *c* (P-450) reductase in kidney or lung has not been reported previously. Our results agree with morphologic and biochemical evidence suggesting that the proximal tubule is the site of drug metabolism in the kidney (*11*). Boyd (*12*) has shown by autoradiography that a metabolite of the pulmonary toxin, 4-ipomeanol, is localized in pulmonary nonciliated bronchiolar (Clara) cells, and has postulated that the Clara cell is the primary location of P-450–mediated mixed function oxidase enzymes in the lung. Our results are consistent with the presence of the enzyme in the bronchioles, but indicate that additional cell types in the bronchi and bronchioles, and possibly a parenchymal cell type, are positive for NADPH–cytochrome *c* (P-450) reductase. Although the Clara cell may be the predominant cell metabolizing ipomeanol, the more general method of determining the distribution of NADPH–cytochrome *c* (P-450) reductase, which (according to current knowledge) exists in only one form and is presumably present wherever any of the multiple forms of cytochrome P-450 occur, may be a more sensitive way to identify cells containing cytochrome P-450–specific mixed function oxidase enzymes.

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Social Environment as a Factor in Diet-Induced Atherosclerosis

Abstract. *Rabbits on a 2 percent cholesterol diet were individually petted, held, talked to, and played with on a regular basis. Measurements of aortic affinity for a Sudan stain, serum cholesterol levels, heart rate, and blood pressure were made at the end of the experimental period. Compared to control groups, which were given the same diet and normal laboratory animal care, the experimental groups showed more than a 60 percent reduction in the percentage of aortic surface area exhibiting sudanophilic lesions, even though serum cholesterol levels, heart rate, and blood pressure were comparable.*

The apparent relation between stress and cardiovascular disease is based on a variety of evidence implicating physical, emotional, and behavioral factors (*1*–*6*). Data from animal studies link psychosocial disruption to pathological changes in the cardiovascular system (*7*–*14*). These studies include several in which states of severe emotional disturbance were produced with negative stressors. To our knowledge, however, there have not been any studies in which the effects of positive factors were investigated.

We designed a series of studies to investigate the influence of social environment on diet-induced atherosclerosis in rabbits. In the experimental groups (groups A, B, and D), the animals experienced social interaction with an experimenter (M.J.L.). In the control groups (groups C and E), the animals received normal laboratory animal care. Group A was studied during late 1977 without a concurrent control, although earlier control studies had been carried out. The results for group A, although initially considered anomalous, ultimately led us to conduct two additional studies. One was carried out in early 1978 and involved experimental group B and control group C. The other was carried out in late 1978 and involved experimental group D and control group E.

It should be noted that the essence of the experimental environment studied here was to establish a one-to-one relationship between each animal and the experimenter. This was achieved through an early morning, half-hour visit during which each animal was handled, stroked, talked to, and played with; an hour-long feeding period during which the animal was also touched and talked to; and a number of 5-minute visits during the day. Through this daily process, the animals quickly learned to recognize the experimenter, and when present, many even sought her personal attention. They were left alone for 10 hours each night.

The animals used in this study were young male New Zealand White rabbits. Upon being received, they were separated and subjected to a 2-week adaptation period during which they and the experimenter became acquainted. All experiments were carried out by the same experimenter with the same protocol.

After the adaptation period, the ani-

0036-8075/80/0627-1475$00.50/0

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Basic Science Reports

Social Environment Influences the Progression of Atherosclerosis in the Watanabe Heritable Hyperlipidemic Rabbit

Philip M. McCabe, PhD; Julie A. Gonzales, PhD; Julia Zaias, DVM; Angela Szeto, BA; Mahendra Kumar, PhD; Alan J. Herron, DVM; Neil Schneiderman, PhD

Background—Although there is evidence that emotionally stressful behavior can accelerate the progression of atherosclerosis, there is less data to support the notion that affiliative social behavior can slow disease progression. The present study examines the influence of social environment on the progression of atherosclerosis in the Watanabe Heritable Hyperlipidemic (WHHL) rabbit, a model that spontaneously develops lesions because of a genetic defect in lipoprotein clearance.

Methods and Results—WHHL rabbits were assigned to 1 of 3 social or behavioral groups: an unstable group, in which unfamiliar rabbits were paired daily, with the pairing switched each week; a stable group, in which littermates were paired daily for the entire study; and an individually caged group. The stable group exhibited more affiliative social behavior and less agonistic behavior than the unstable group and significantly less aortic atherosclerosis than each of the other 2 groups. Although the unstable and individually caged groups had comparable aortic lesion areas, the severity of the disease progressed faster in the unstable group, as indexed by a larger area of calcification and increased fibrous cap thickness in complex lesions. The unstable group showed increased agonistic behavior and signs of chronic adrenocortical and gonadal activation, whereas the individually caged group was relatively sedentary, had low glucocorticoid levels, and was hyperinsulinemic compared with the other groups.

Conclusions—The present study demonstrates that social environment can slow, as well as accelerate, the progression of atherosclerosis. It also emphasizes the importance of behavioral factors in atherogenesis, even in a model of disease with strong genetic determinants. **(*Circulation*. 2002;105:354-359.)**

Key Words: behavior ■ social environment ■ atherosclerosis ■ rabbits

Behavioral factors have been implicated in the etiology of atherosclerosis and coronary heart disease (CHD).[1] It has been established that cynomolgus macaques, fed an atherogenic diet and exposed to an emotionally stressful social environment, develop greater CHD than monkeys in a stable social environment or animals fed a low-fat diet.[1–4] These studies suggest that behavioral factors can promote disease progression; however, there is far less evidence that manipulation of emotional behavior can slow the progression of atherosclerosis. Notably, it was demonstrated that repeated handling and petting of albino rabbits decreased aortic atherosclerosis relative to controls.[5] The paucity of research relating to this topic may be attributable to the fact that in most of these models, atherosclerosis does not develop spontaneously, and, therefore, it is difficult to demonstrate an experimental attenuation of the disease process.

The Watanabe Heritable Hyperlipidemic (WHHL) rabbit is an inbred strain of rabbit that exhibits hypercholesterolemia, elevated plasma LDL levels, and severe atherosclerosis.[6,7] This abnormality is inherited as a single-gene mutation, with a genotype and phenotype that is strikingly similar to human familial hypercholesterolemia.[8] The WHHL rabbits spontaneously develop observable atherosclerotic lesions in the aortic arch beginning at 2 months of age, and by 6 months of age, 100% of WHHL rabbits have severe atherosclerotic lesions at all levels of the aorta.[9] Although many diseases, such as familial hypercholesterolemia, have strong genetic determinants, the expression of disease can vary as a result of environmental factors. To date, there have been no published studies that have examined the relationship of behavioral factors to the progression of atherosclerosis in the WHHL rabbit. This may be attributable, in part, to the belief that genetic variables in this model are so predominant that behavior does not have an appreciable influence on disease progression.

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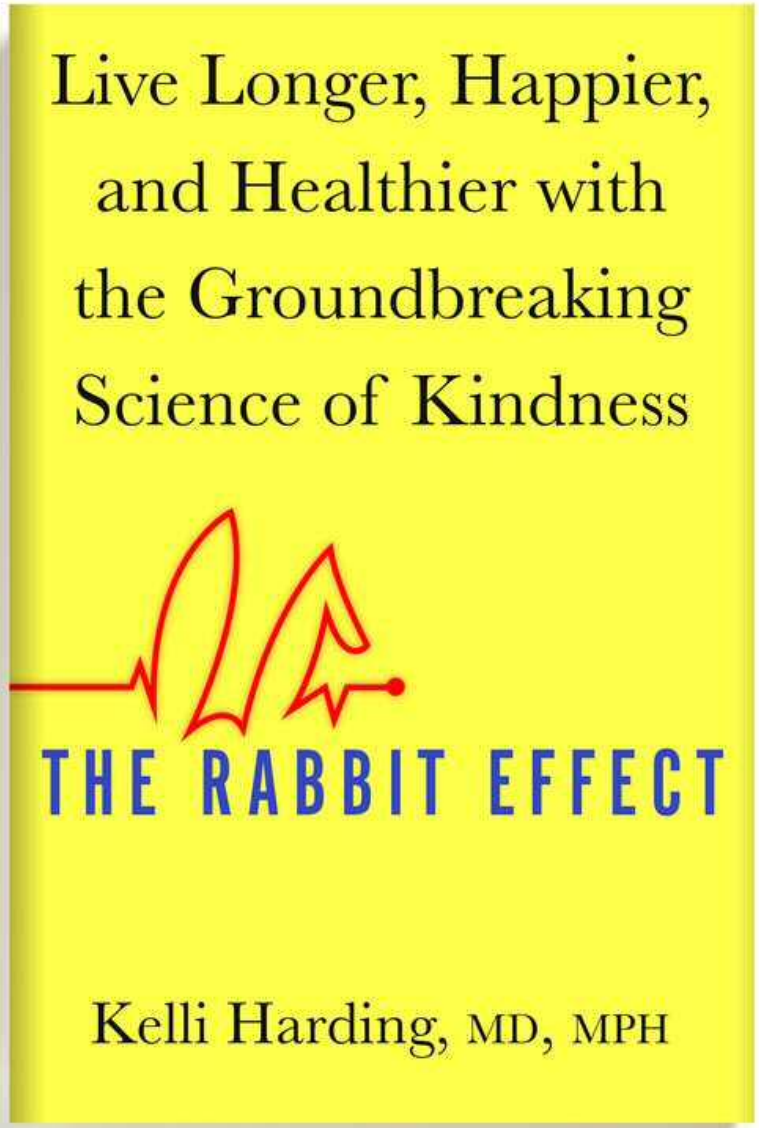
McCabe PM, Gonzales JA, Zaias J, *et al*. Social environment influences the progression of atherosclerosis in the Watanabe heritable hyperlipidemic rabbit. *Circulation 2002*; 105:354-9 (Jan 22).



Harding, Kelli. *The Rabbit Effect.* New York, NY: Atria Books, 2019.

"Take a rabbit with an unhealthy lifestyle. Talk to it, hold it, give it affection. …

"The relationship made a difference. …

"Ultimately, what affects our health in the most meaningful ways has as much to do with how we treat one another, how we live, and how we think about what it means to be human."

Harding, Kelli. *The Rabbit Effect.* New York, NY: Atria Books, 2019; pp. xxiv, xxv.

Humans need social contact to thrive

A series of studies:

- ***Assessing infants in orphanages***
- ***Sufficient physical sustenance*** *(shelter, food, water, etc.)*
- ***Very limited human contact***
- ***Failure to thrive – the "runt syndrome"***
- ***Significantly lower IQ*** *(reduced brain development)*
- ***As many as 1 in 3 simply waste away and die***

Same effect observed with elderly patients experiencing social isolation during the COVID-19 pandemic
(and with other groups of humans in general)

How Orphanages Kill Babies – and Why No Child Under 5 Should Be in One

For most people, the word orphanage conjures cold Dickensian images of cruelty to children -- and yet whenever I write that children under five should never be kept in institutional care, I hear from people who vigorously defend such facilities.

By

Maia Szalavitz, Contributor

Author, Journalist

06/23/2010 05:12am EDT | **Updated** November 17, 2011

For most people, the word orphanage conjures cold Dickensian images of cruelty to children -- and yet whenever I write that children under five should never be kept in institutional care, I hear from people who vigorously defend such facilities.
With public attention focused on the horrifying case of Artyom Savelyev who was sent home alone to Russia after being briefly adopted from an orphanage, more people need to know why orphanages for infants are indefensible and can safely and economically be shuttered.

Indeed, that has already quietly happened in the U.S. and Western Europe over the last few decades. Baby orphanages here have gone extinct because experts now understand the profound dangers they pose for infants. But to help more kids and future adoptive families, these facilities for infants need to be abolished in the rest of the world as well.

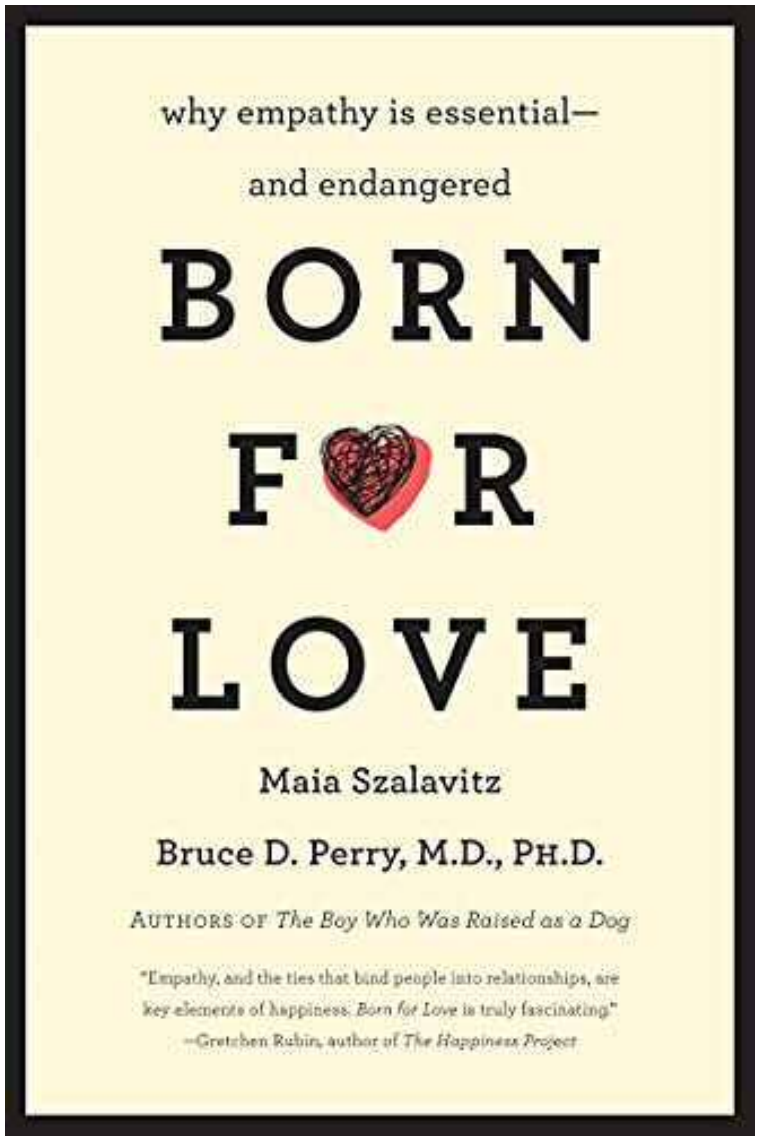
Unfortunately, the myth of the good orphanage for little ones lives on in the popular imagination and in Eastern Europe and China. Even the *New York Times* recently promoted it, with an article that claimed that research shows orphanages are fine for kids. Sadly, the article failed to note that the research was conducted on children over six-- and so doesn't apply to orphanages for babies.

More recently, in an editorial on the Savelyev case, the *Times* said that Russia's orphanages were merely "overcrowded, with too few staff members and resources." Instead, it should have called for replacing baby orphanages entirely with foster care.

Here's why. As we discuss in our book, *Born for Love: Why Empathy Is Essential -- and Endangered*, about 1/3 of babies placed in the barest orphanages can actually die as a result (one very early study found this death rate).

Half of the rest -- at least double the rate seen in the general population -- will suffer from mental illness. Each month spent in an orphanage in early life reduces IQ and increases risk of behavioral and psychological problems--and this has been proved by the highest level of scientific proof we have, a randomized controlled trial.

Szalavitz M. How orphanages kill babies – and why no child under 5 should be in one. *Huff Post Blog June 23,* 2010; at https://www.huffpost.com/entry/how-orphanages-kill-babie_b_549608



Szalavitz, Maia, and Perry, Bruce D. *Born For Love: Why Empathy Is Essential – and Endangered.* New York, NY: William Morrow, 2010.

from neuronal synchrony. Synchrony may have contributed to these effects, and lack of synchrony during another epoch may have resulted in an absent effect [see, however, (*14*)].

Lack of synchrony was unlikely to account for absent effects when the highly significant effects were consistent with monosynaptic connections (onset latency > 5 ms and PWHM < 9 ms, lower right quadrants). Particularly for the eight SpikeTA effects in the lower right quadrant of Fig. 3C (from eight different neurons recorded in eight different sessions, three in monkey E and five in monkey W), the loss of throughput that resulted in absent effects cannot be attributed simply to lower neuron firing rate, lower ongoing EMG, and/or loss of synchronized inputs. Additional factors may have changed the throughput from these M1 neurons to their target muscles.

Although M1 output, particularly that from CM cells, dominates control of distal upper extremity musculature during voluntary activity, our results show that the throughput from individual M1 neurons to muscle activity can be changed rapidly and dramatically. For about half of the neuron-muscle pairs that produced highly significant SpikeTA effects, throughput evident during some behavioral epochs was absent during other epochs. In most cases, differences in intracortical excitability and the resulting changes in excitation of motoneuron pools reflected by the firing rate of the trigger neuron, the level of ongoing EMG activity, and/or synchrony in the SpikeTA effect contributed to the presence of effective throughput during some behavioral epochs and not others.

In about 10% (8 of 82) of cases, however, none of these factors could account for the presence versus absence of throughput from the M1 neuron to the muscle's EMG activity. We therefore speculate that three subcortical factors may have contributed as well. First, some SpikeTA effects may be mediated through disynaptic linkages that involve rubrospinal neurons, reticulospinal neurons, or spinal interneurons (*20*–*22*). Such effects may have been blocked during some epochs by inactivity of the interposed neuron. This mechanism seems likely for suppressive effects, all of which are mediated through inhibitory interneurons, and may have contributed to the absence of some facilitative effects as well. Second, single CM cell EPSPs in motoneurons may be relatively small (*23*, *24*). Within motoneuron dendrites, small synaptic inputs may have been amplified by persistent inward currents during some behavioral epochs but not during others (*25*). Third, the synaptic input from an M1 neuron to a motoneuron pool commonly is assumed to remain constant. Although synaptic efficacy might be altered by presynaptic inhibition, available evidence indicates that this mechanism does not affect corticospinal terminals (*26*, *27*). Plastic changes can occur in spinal cord synapses which might have played a role in the 10% of cases lacking differences in intracortical excitability, also could have contributed to the rapid change in throughput in many of the other 90%.

Our findings indicate that M1 neurons, even those with relatively direct connections to α-motoneurons, are not always effective in driving their target motoneurons. Rather, throughput can be changed rapidly such that an individual M1 neuron, which is ineffective in eliciting motoneuron discharge during certain motor behaviors, does elicit discharge of the same motoneurons during other behaviors.

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Supporting Online Material

www.sciencemag.org/cgi/content/full/318/5858/1934/DC1
Materials and Methods
Figs. S1 and S2
Table S1
References

27 August 2007; accepted 16 November 2007
10.1126/science.1149774

Cognitive Recovery in Socially Deprived Young Children: The Bucharest Early Intervention Project

Charles A. Nelson III,[1]* Charles H. Zeanah,[2] Nathan A. Fox,[3] Peter J. Marshall,[4] Anna T. Smyke,[2] Donald Guthrie[5]

In a randomized controlled trial, we compared abandoned children reared in institutions to abandoned children placed in institutions but then moved to foster care. Young children living in institutions were randomly assigned to continued institutional care or to placement in foster care, and their cognitive development was tracked through 54 months of age. The cognitive outcome of children who remained in the institution was markedly below that of never-institutionalized children and children taken out of the institution and placed into foster care. The improved cognitive outcomes we observed at 42 and 54 months were most marked for the youngest children placed in foster care. These results point to the negative sequelae of early institutionalization, suggest a possible sensitive period in cognitive development, and underscore the advantages of family placements for young abandoned children.

For normal development, mammalian brains require an optimal level of environmental input, a so-called "expectable" environment (*1*, *2*). Examples of an expectable environment might include exposure to patterned light information, normal language exposure, and access to mented routines (e.g., all children eat, sleep, and toilet at the same time); impoverished sensory, cognitive, and linguistic stimulation; and unresponsive caregiving practices. These issues af-

[1]Harvard Medical School and Children's Hospital, Boston, MA

Nelson CA, Zeanah CH, Fox NA, Marshal PJ, Smyke AT, Guthrie D. Cognitive recovery in socially deprived children: The Bucharest Early Intervention Project. *Science* 2007; 381(5858):1937-40 (Dec 21).

from neuronal synchrony. Synchrony may have contributed to these effects, and lack of synchrony during another epoch may have resulted in an absent effect [see, however, (*14*)].

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In a randomized controlled trial, we compared abandoned children reared in institutions to abandoned children placed in institutions but then moved to foster care. Young children living in institutions were randomly assigned to continued institutional care or to placement in foster care, and their cognitive development was tracked through 54 months of age. The cognitive outcome of children who remained in the institution was markedly below that of never-institutionalized children and children taken out of the institution and placed into foster care. The improved cognitive outcomes we observed at 42 and 54 months were most marked for the youngest children placed in foster care. These results point to the negative sequelae of early institutionalization, suggest a possible sensitive period in cognitive development, and underscore the advantages of family placements for young abandoned children.

of spinal interneurons (20–22). Such effects may have been blocked during some epochs by inactivity of the interposed neuron. This mechanism seems likely for suppressive effects, all of which are mediated through inhibitory interneurons, and may have contributed to the absence of some facilitative effects as well. Second, single CM cell EPSPs in motoneurons may be relatively small (*23*, *24*). Within motoneuron dendrites, small synaptic inputs may have been amplified by persistent inward currents during some behavioral epochs but not during others (*25*). Third, the synaptic input from an M1 neuron to a motoneuron pool commonly is assumed to remain constant. Although synaptic efficacy might be altered by presynaptic inhibition, available evidence indicates that this mechanism does not affect corticospinal terminals (*26*, *27*). Plastic changes can occur in spinal cord synapses

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For normal development, mammalian brains require an optimal level of environmental input, a so-called "expectable" environment (*1*, *2*). Examples of an expectable environment might include exposure to patterned light information, normal language exposure, and access to

mented routines (e.g., all children eat, sleep, and toilet at the same time); impoverished sensory, cognitive, and linguistic stimulation; and unresponsive caregiving practices. These issues af-

[3]Harvard Medical School and Children's Hospital, Boston, MA

Nelson CA, Zeanah CH, Fox NA, Marshal PJ, Smyke AT, Guthrie D. Cognitive recovery in socially deprived children: The Bucharest Early Intervention Project. *Science* 2007; 381(5858):1937-40 (Dec 21).

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 previously been described.

Methods: A questionnaire about 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 then compared to measures of adult risk behavior, health status, and disease. Logistic regression was used to adjust for 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.

Results: More than half of respondents reported at least one, and one-fourth reported ≥2 categories of childhood exposures. We found a graded relationship between the number of categories of childhood exposure and each of the adult health risk behaviors and diseases that were studied ($P < .001$). Persons who had experienced four or more categories of childhood exposure, compared to those who had experienced none, had 4- to 12-fold increased health risks for alcoholism, drug abuse, depression, and suicide attempt; a 2- to 4-fold increase in smoking, poor self-rated health, ≥50 sexual intercourse partners, and sexually transmitted disease; and a 1.4- to 1.6-fold increase in physical inactivity and severe obesity. The number of categories of adverse childhood exposures showed a graded relationship to the presence of adult diseases including ischemic heart disease, cancer, chronic lung disease, skeletal fractures, and liver disease. The seven categories of adverse childhood experiences were strongly interrelated and persons with multiple categories of childhood exposure were likely to have multiple health risk factors 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)

Department of Preventive Medicine, Southern California Permanente Medical Group (Kaiser Permanente), (Felitti) San Diego, California 92111. National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention, (Anda, Williamson, Spitz, Edwards, Marks) Atlanta, Georgia 30333. Department of Pediatrics, Emory University School Medicine, (Nordenberg) Atlanta, Georgia 30333. Department of Family and Community Medicine, University of Arizona Health Sciences Center, (Koss) Tucson, Arizona 85727.

Address correspondence to: Vincent J. Felitti, MD, Kaiser Permanente, Department of Preventive Medicine, 7060 Clairemont Mesa Boulevard, San Diego, California 92111.

Felitti VJ, Anda RF, Nordenberg D, *et al*. Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults – the Adverse Childhood Experiences (ACE) study. *Am J Prev Med* 1998; 14(4):245-58.

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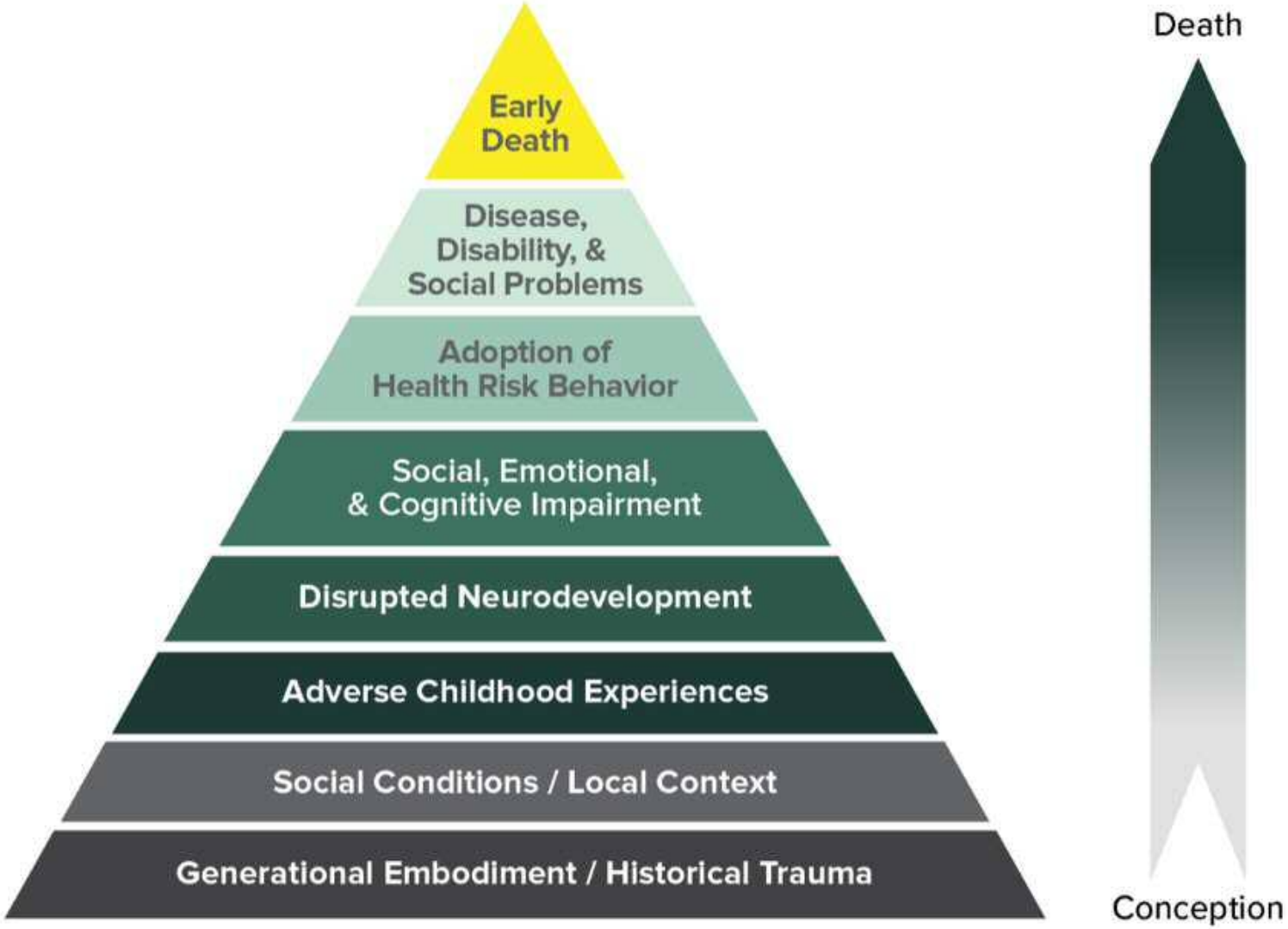


Mechanism by which Adverse Childhood Experiences Influence Health and Well-being Throughout the Lifespan

https://cdc.gov/violenceprevention-aces-about.html (+The ACE Pyramid)

The ACE trial – key take-aways

The first 5 years of life predicts an individual's life expectancy across their entire remaining life arc.

The effect is mediated through health-related behaviors.

Helping children succeed *despite challenging barriers*

Emmy Werner studied children in Kauai with risk factors that put them on a trajectory for failure (e.g., father absence or criminality, mother with mental health problems). Some overcame those barriers to succeed regardless. "The resilient youngsters in our study had at least one person in their lives who accepted them unconditionally, regardless of temperamental idiosyncrasies, physical attractiveness, or intelligence."

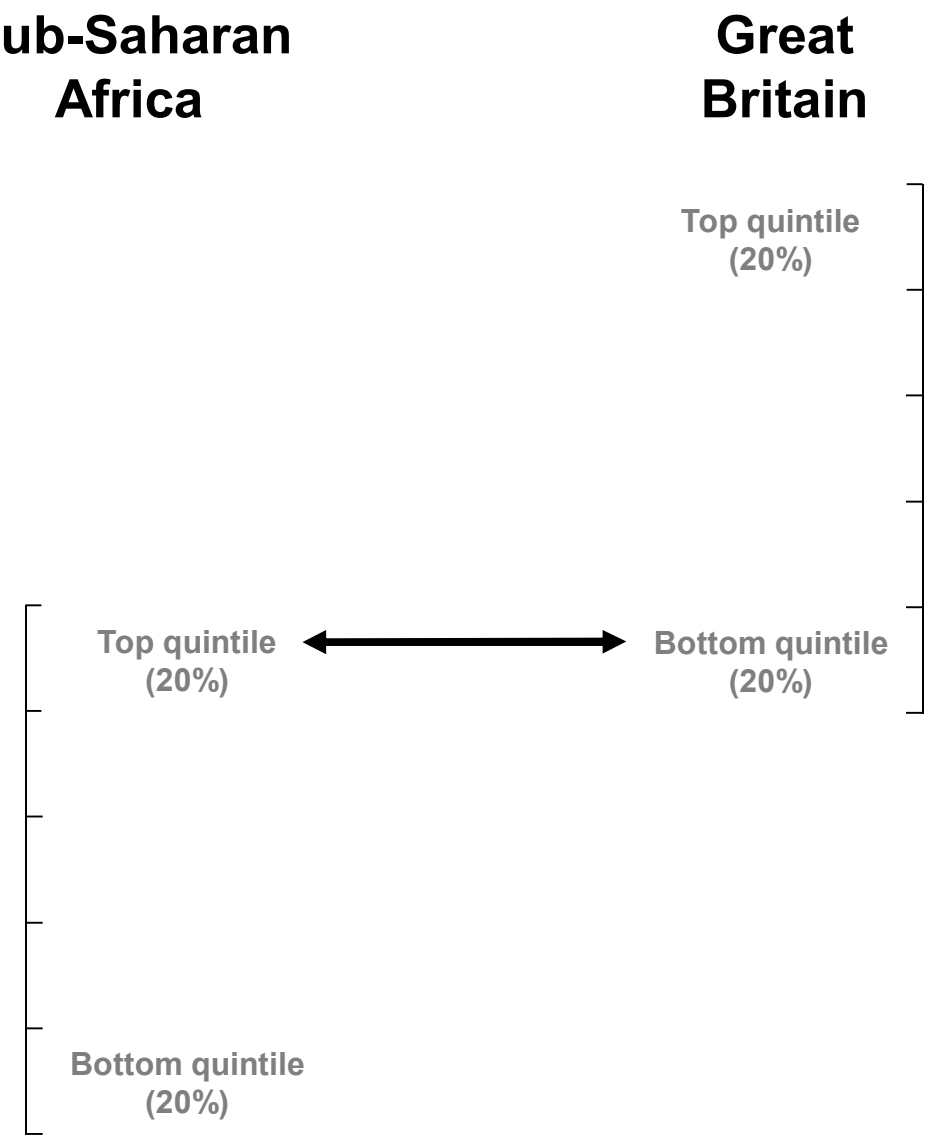
Werner EE, Smith RS. *Overcoming the Odds: High-Risk Children from Birth to Adulthood*. Ithaca, NY: Cornell University Press, 1992; pg. 205.

"Every child should spend a substantial amount of time with someone who's crazy about him or her. There has to be at least one person who has an irrational involvement with that child, who thinks that kid is more important than other people's kids, someone who's in love with him or her and whom he or she loves in return."

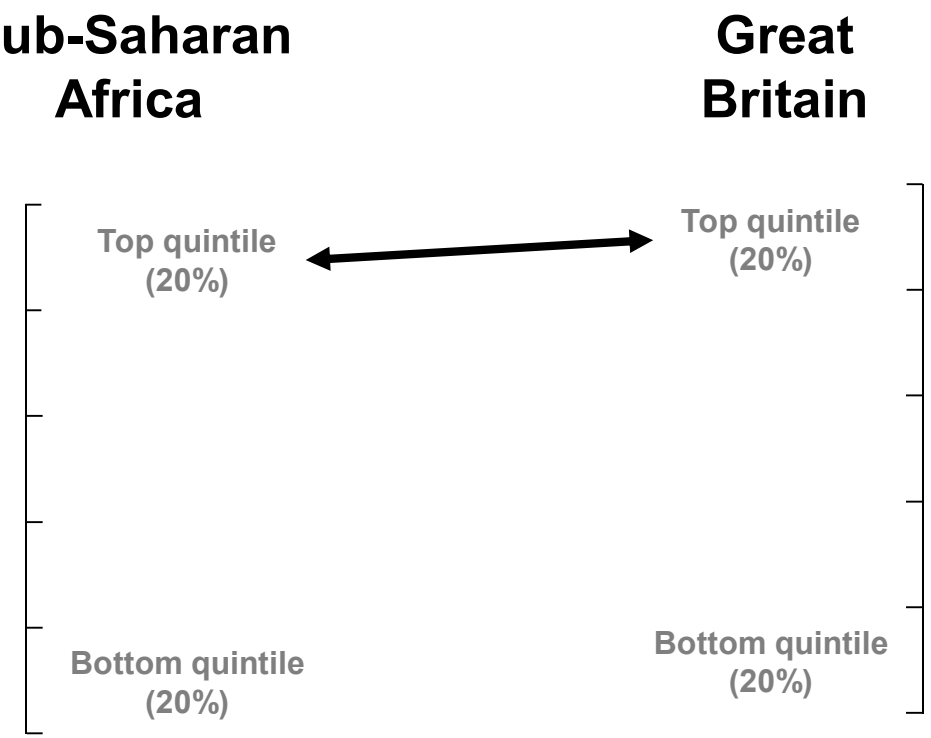
Bronfenbrenner U. Nobody home: The erosion of the American family. *Psychology Today* 1977; 10(12):40 (May).

Courtesy of Dr. Wallace Goddard, University of Arkansas.

Wealth *as measured by purchasing power*



Health *as measured by life expectancy*



The idea of rank within a society …

Michael Marmot, *professor of epidemiology and public health, University College London,* ***cites 3 studies****:*

Whitehall study *(Great Britain)*

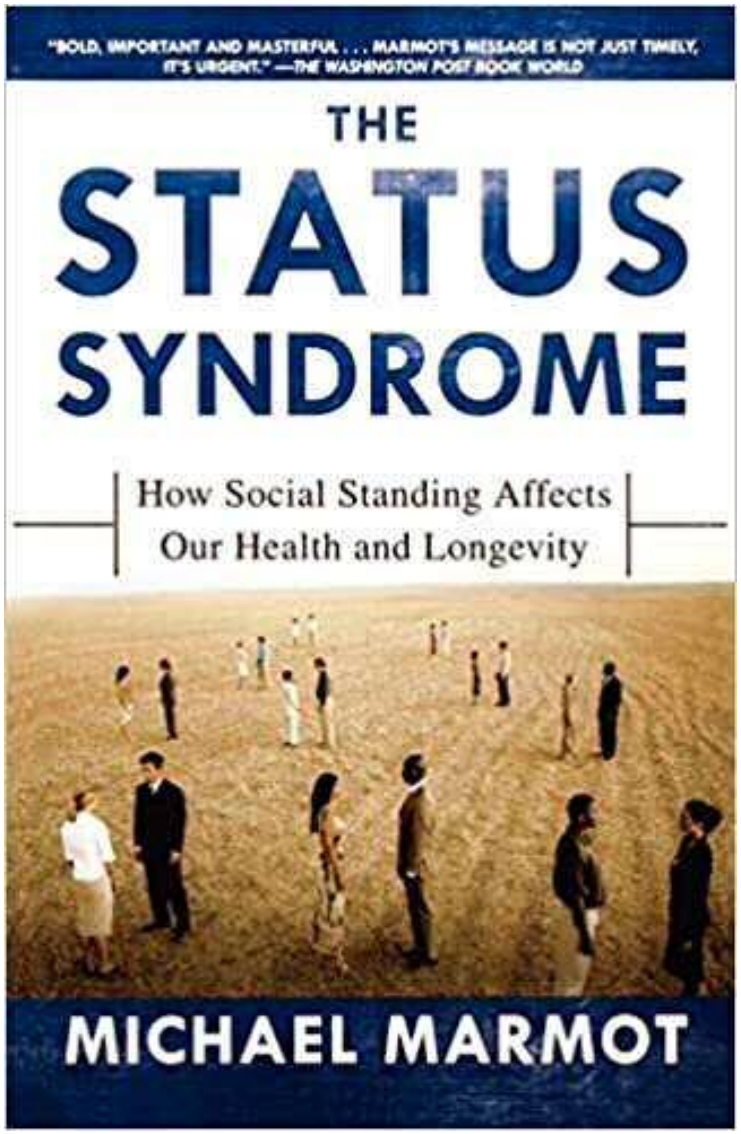
Higher ranks in the British civil service are associated with lower risk of death, even though all had reasonably high incomes

Oscar winners *(United States)*

Oscars winners live, on average, 4 years longer than other successful non-Oscar actors and actresses

Education levels *(Sweden)*

PhDs live longer than Master's degrees, who live longer than Bachelor's degrees, and so on down the educational chain



Marmot, Michael. *Status Syndrome – How Social Standing Affects Our Health and Longevity.* New York, NY: Henry Holt and Company, 2004.

Status syndrome

Health and longevity are intimately related to position in the social hierarchy. The lower the status, the higher risk of illness and death, and consequently the shorter the life expectancy. In his book of the same name, **Michael Marmot** calls this social gradient in health the "Status Syndrome". So what exactly is the cause of this gradient?

As described in *Status Syndrome*[1], the gap in life expectancy between the top and bottom of the hierarchy is big. This can be illustrated in the USA by a ride on the Washington DC metro. Travel from the south east of downtown Washington to Montgomery County Maryland. For each mile travelled life expectancy rises about a year and a half. There is a 20-year gap between poor blacks at one end of the journey and rich whites at the other. Men in Japan have the longest life expectancy in the world at 77; men in Kazakhstan in the former Soviet Union are way down at 57. Within Washington and its environs, we see differences as big[1].

Poverty?

This headline figure of a gap of 20 years between the top and bottom of the hierarchy could be read as implying that the poor have poor health and the non-poor have reasonable health. They do, but this is to miss the challenging point, which is that health follows a gradient: the higher the social position, the better the health. Absolute poverty won't do as an explanation. This can be illustrated by three examples.

First is the one that has occupied me for the last 28 years: the Whitehall studies of British civil servants. The original Whitehall study showed that, among men aged 40–64, the higher the rank in the civil service, the lower the risk of death over successive periods of follow-up. None of these civil servants could be described as materially deprived in the sense of England during the 1930s or earlier, for example, yet position in the

"Position in the hierarchy is

chances

Singh showed that actors who win an Academy award live an astonishing 4 years longer than other actors[5]. 4 years is enormous. Removing coronary heart disease—the number one cause of death, statistically—would add about 3.6 years to the nation's life expectancy. Can winning an Oscar be causal and responsible for such a profound improvement in health? Or is it the other way round: the longer an actor lives, the more chance he or she has of winning one eventually? Redelmeier and Singh deal with this by taking two comparison groups: actors who were nominated and did not win, and actors who were in the film in which the winner appeared. Adjusting for age, they found that the Oscar winners lived about 4 decades after winning their award; the also-rans about 4 years less. The longer life expectancy was unlikely to be the result of simply having more money. The control group made an average of 47.4 movies in their careers.

The idea that the boost in status that goes with winning an Oscar is responsible for the longer life may, at first blush, seem to be contradicted by the fact that screen writers who win an Oscar don't have longer life expectancy than other screen writers[6]. But does winning an Oscar increase a screen writer's status? Can you name a screen writer who won an Oscar?

The third example of the gradient is Eriksson's study of the whole Swedish population, linking census data to mortality follow-up[7]. People with a PhD have a longer life expectancy than those with a Mas-

tion,

Marmot M. Status syndrome. *Significance* 2004; 1(4):150-4 (Dec).

a.k.a. Social gradient / social stress

Related to 2 fundamental human needs:

1. *To have control over your own life; and*
2. *To be a full social participant.*

Arises within the human brain:

"… stress arising from

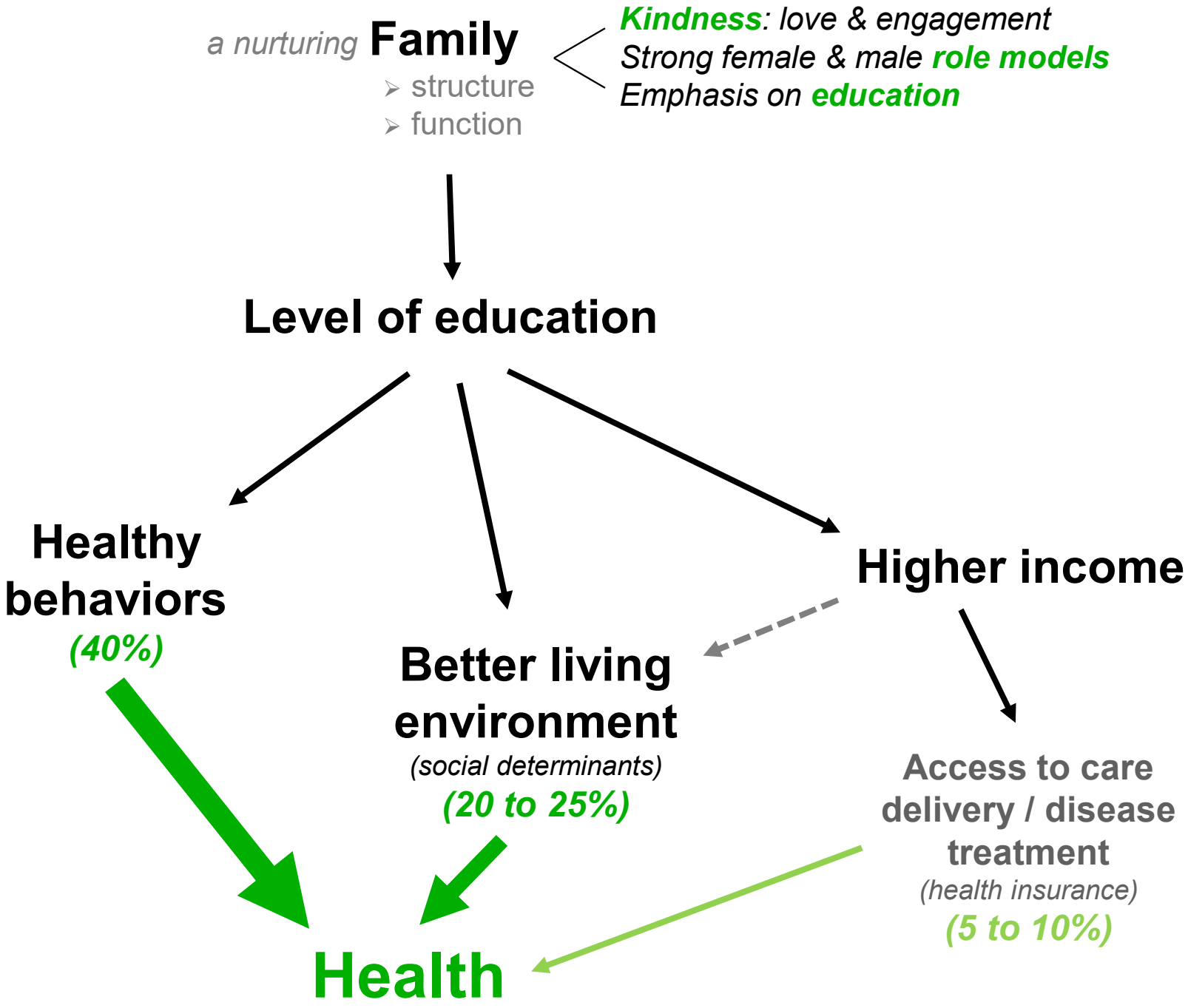
– an inability to control our lives,

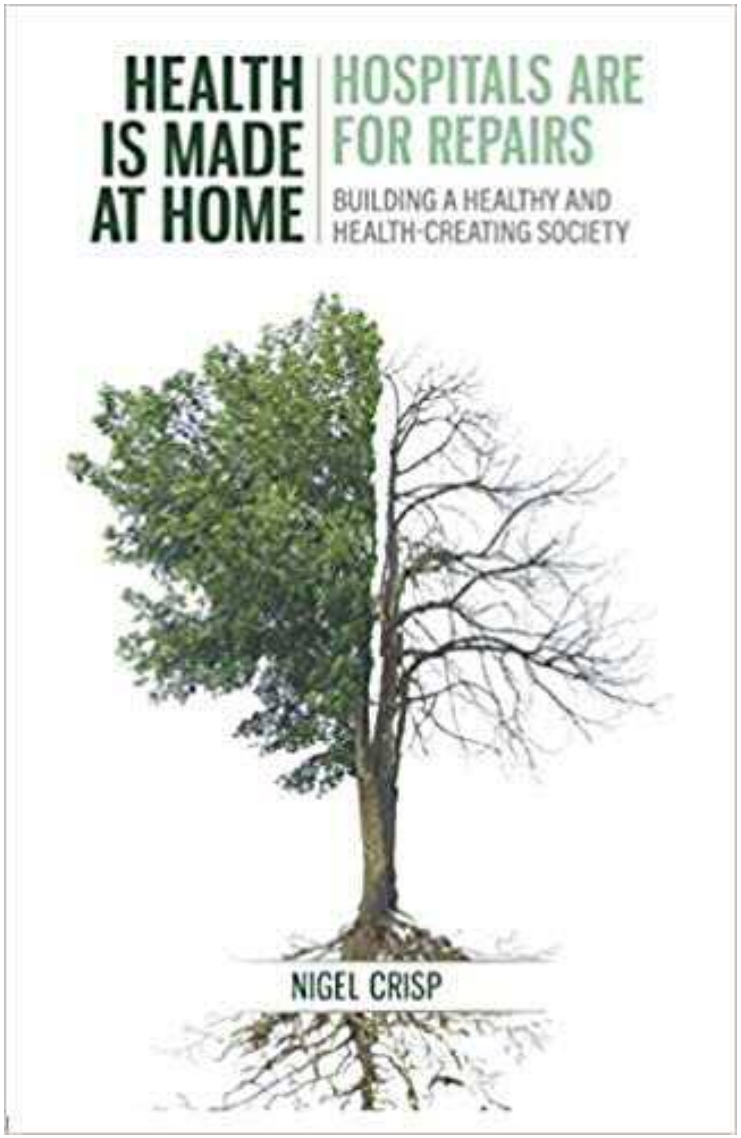
– to turn to others when we lose control, or

– to participate fully in all that society has to offer."

Links directly to health *(disease rates, life expectancy)*

The steepness of the social gradient is not constant, either across countries or within a country at different time periods.


a nurturing Family
structure
function
Kindness: love & engagement
Strong female & male role models
Emphasis on education
Level of education
Healthy behaviors
(40%)
Better living environment
(social determinants)
(20 to 25%)
Higher income
Access to care delivery / disease treatment
(health insurance)
(5 to 10%)
Health



Crisp, Nigel. *Health Is Made At Home Hospitals Are For Repairs – Building a Healthy and Health-Creating Society.* Billericay, Essex, United Kingdom: SALUS Global Knowledge Exchange, 2020.

Implication

After a person hits some minimum income threshold,

income is not the key driver that determines life expectancy.

Income does provide access to current health care delivery services, but has relatively low leverage – something on the order of about 10%.

Health-related behaviors and social determinants are far more important – together, they contribute about 60 to 65%.

(Money is strongly statistically associated with life expectancy, because both income and life expectancy are driven by the same factor: education level)

Implication

***Life expectancy** is **not** a good way to assess **health care delivery system performance** …*

At least, if we accept the current functional definition of the primary aim of health care delivery to be disease treatment: caring (always), curing (when possible), and rescue.

What if we shift our primary aim

to a deeper level of understanding of true "customer" need:

Population health

- *Healthy behaviors (40%)*
- *Environment (social determinants of health – 20 to 25%)*
  - *Physical environment – food insecurity, housing insecurity*
  - *Social environment – families, social networks, Marmot's Status Syndrome*
  - *Public health – control of epidemic infectious disease via immunization and sanitation*

Disease treatment (5 to 15%) –

easy access to care delivery that helps with health problems and conditions:

safe, timely, effective, efficient, and patient-centered/driven