Thinking epidemiologically (in Africa) about the determinants of non-communicable disease

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Study design

Cross-sectional studies

Risk factor and outcome prevalence surveys.

Measure diseases states and risk factors simultaneously.

E.g. SA Demographic and Health Survey: chronic bronchitis (cough and sputum) (Ehrlich et al. 2004)

Risk factors (descending strength):
- History of TB (odds ratio 5 to 6)
- Smoking; older; female;
- Smoky fuel (women); "Dusts/gases" occupation (men)

Strongly protective:
- Education (odds ratio 3.3 for >. 12 yrs)

Problem: Incidence/prevalence bias

Case – control studies

Recruit cases of disease – ideally new onset or diagnosis.
Recruit controls – difficult!
Examine risk factor differences
Problem of recall bias.

- E.g. INTERHEART study (Steyn et al. 2005) – Factors associated with acute heart attacks across a number of African countries and 3 "demographic groups" (descending OR) overall:

- Lipid markers (apolipoproteins)
- Diabetes history
- Hypertension history (highest OR in black African group)
- Abdominal obesity
- Smoking

→ Population attributable fraction = 89%!

Cohort studies

Recruited by exposure or via cohort entry, e.g. birth cohort.
Many exposures/risk factors can be examined.
Outcomes: different diseases, etc.

Allows for the passage of time and long lags between experience or risk factor and disease, and re-measurement.
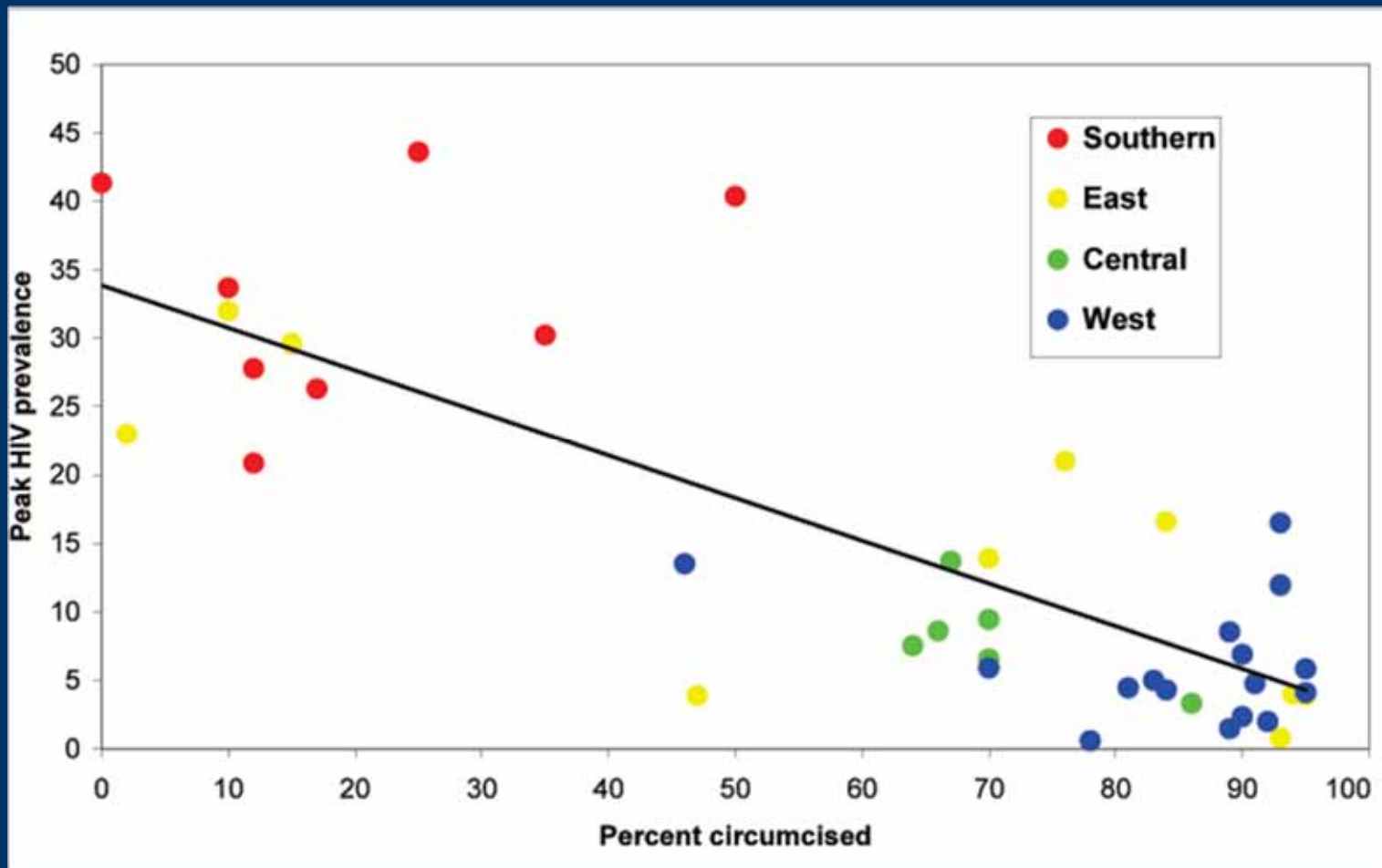
E.g. (South African) Birth to Ten Study (Levitt et al. 1999)

Systolic blood pressure at 5 years associated with:

• Weight at 5 years
• Height at 5 years
• *Low birthweight*

Ecologic studies:
HIV and circumcision in 4 regions of Sub Saharan Africa

Hargrove 2007

# Causes of cases vs causes of incidence

Rose (1985): Do the differences between cases and non-cases explain the differences between populations?

"Variation paradox" : To find a causal or predictive risk factor in a population, it has to *vary sufficiently within the population*.

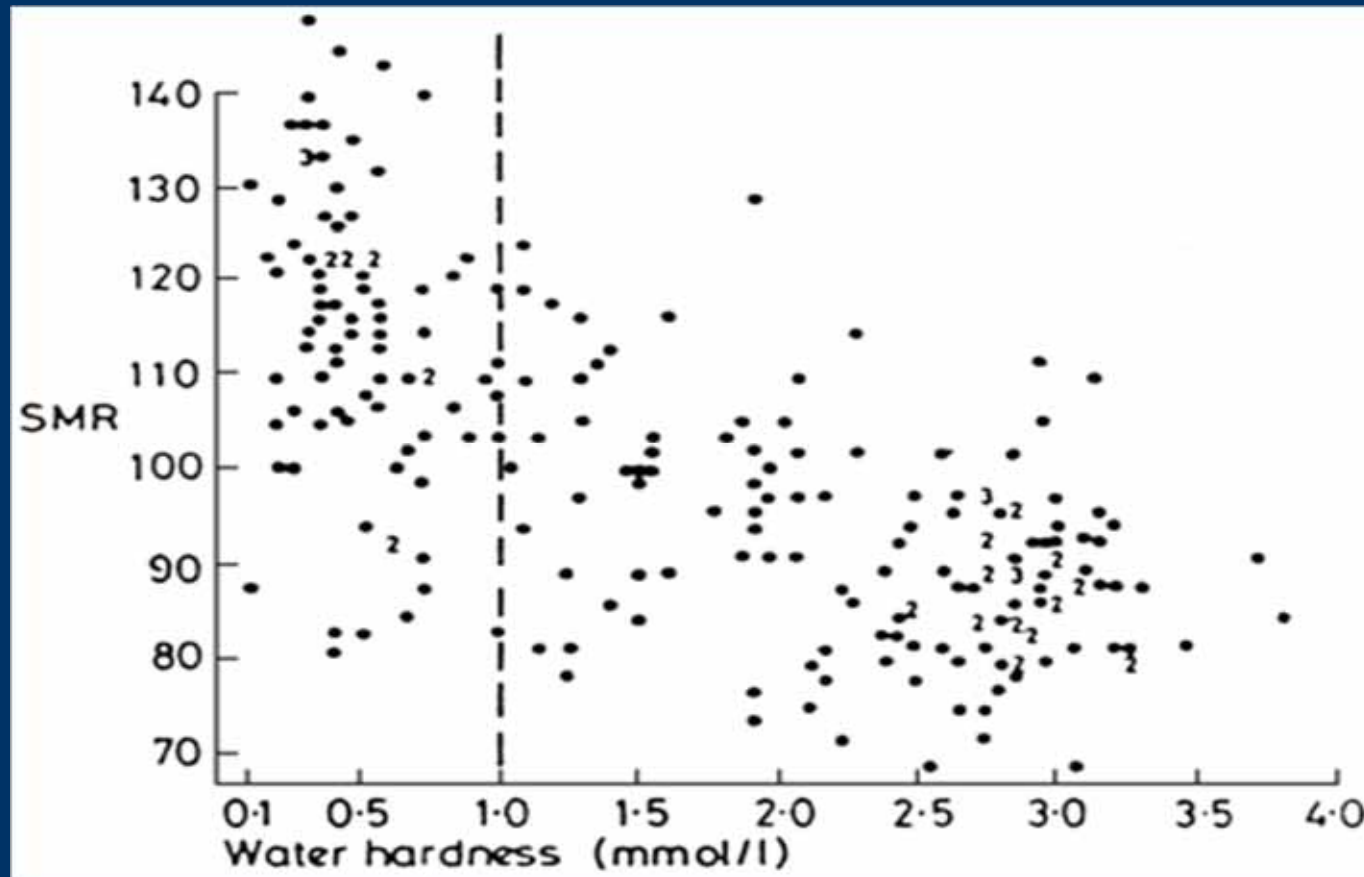If it doesn't vary sufficiently, you require a:

- comparison between populations, or

- comparison of same population over time if risk factor varies over time

Cross-population comparisons: systolic blood pressure

NCD Epidemiology for Africa

Rose 2001
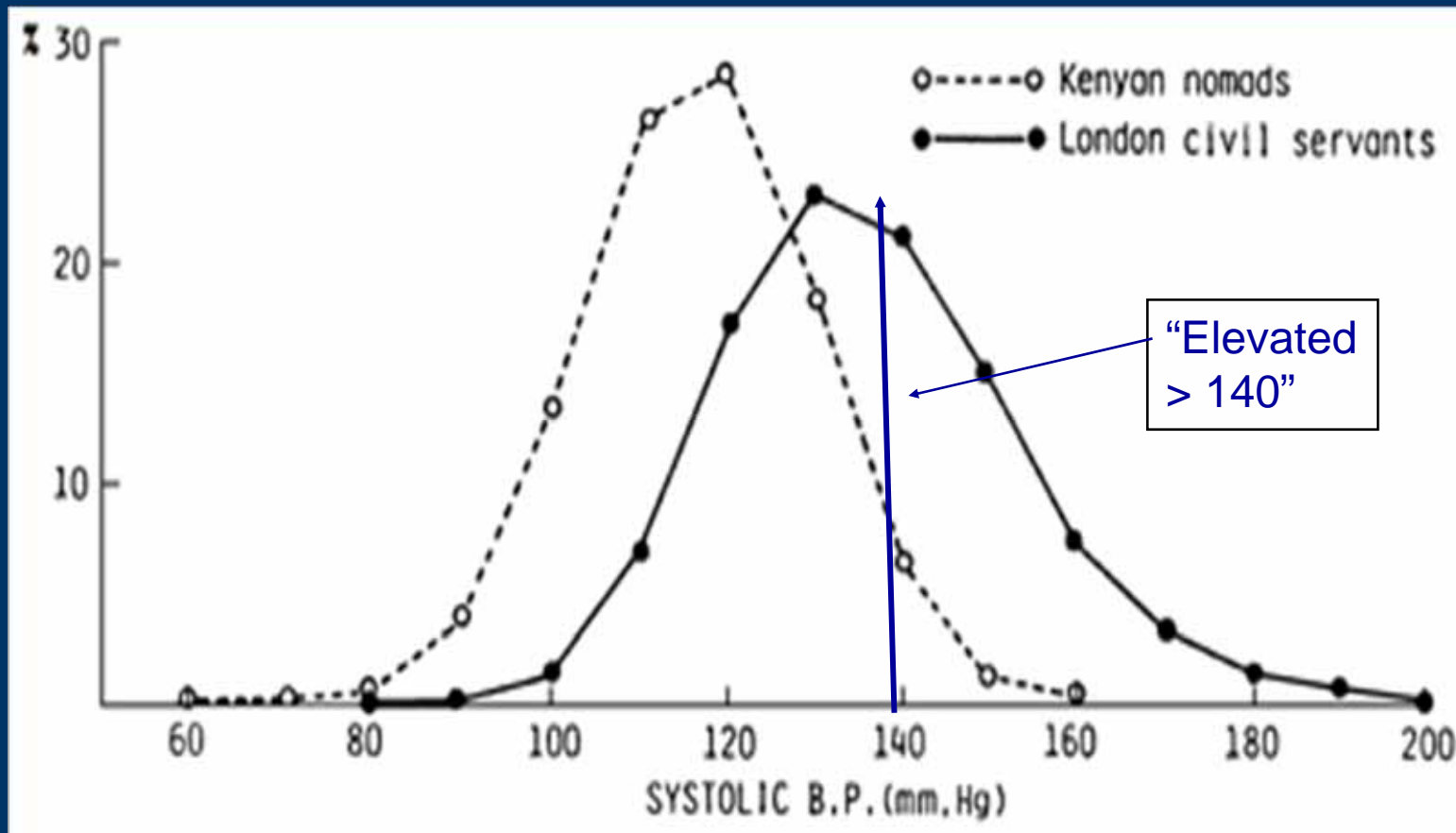
Ecologic study: death rate from coronary heart disease vs water hardness (Ca, Mg content)
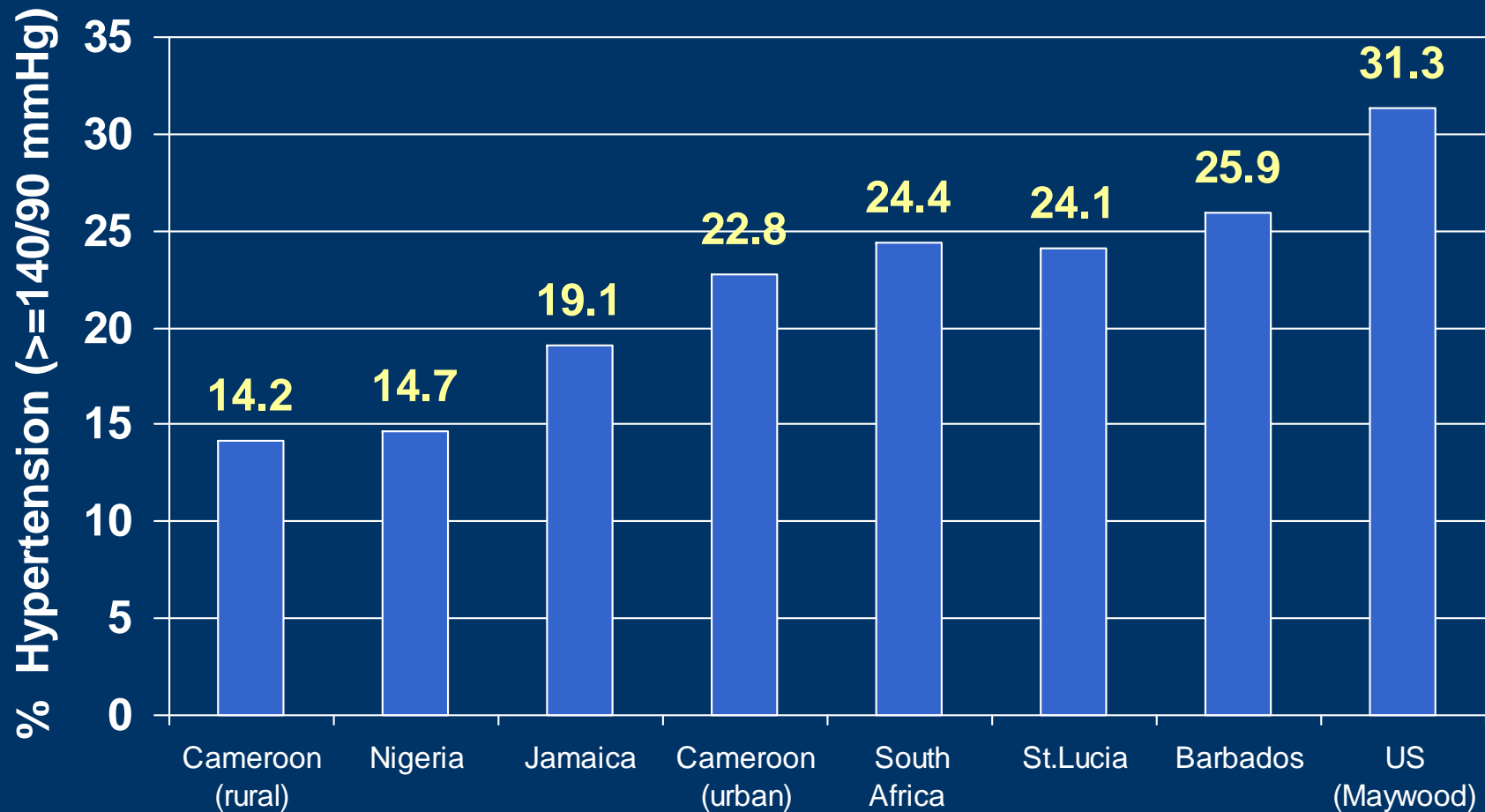

Rose 1985

Limited Gradient – no association

Wider gradient – inverse association apparent

Prevalence of hypertension in African-origin populations

Cooper et al., 1997; Steyn et al., 2001.

Slide courtesy Prof. K. Steyn

To summarise

Within population ("causes of cases"):

• Large variation in *susceptibility factors* (e.g. genetic)

• Large variation in some risk factors, e.g. smoking

• Little or no variation in some important risk factors (e.g. dietary fat, contextual factors)

Between populations ("causes of incidence")

• *Relatively* little variability in genetic factors;

• May be large variation in *population level* risk factors

What are these population level risk factors that "apply to everyone" ?

Policy or intervention factors (Visible)

- Medical care (e.g. comprehensive primary care)
- Availability of screening (e.g. cancer cervix)
- Control of alcohol sales
- Road traffic control

Socioeconomic or sociocultural (social determinants) (Invisible)

- Income inequality
- "Neighbourhood" e.g. crime, environmental degradation
- Food environment
- Linkages (social capital, cohesion, solidarity)

Levels of determinants

Socio-ecological model for factors shaping health behaviour and health outcomes
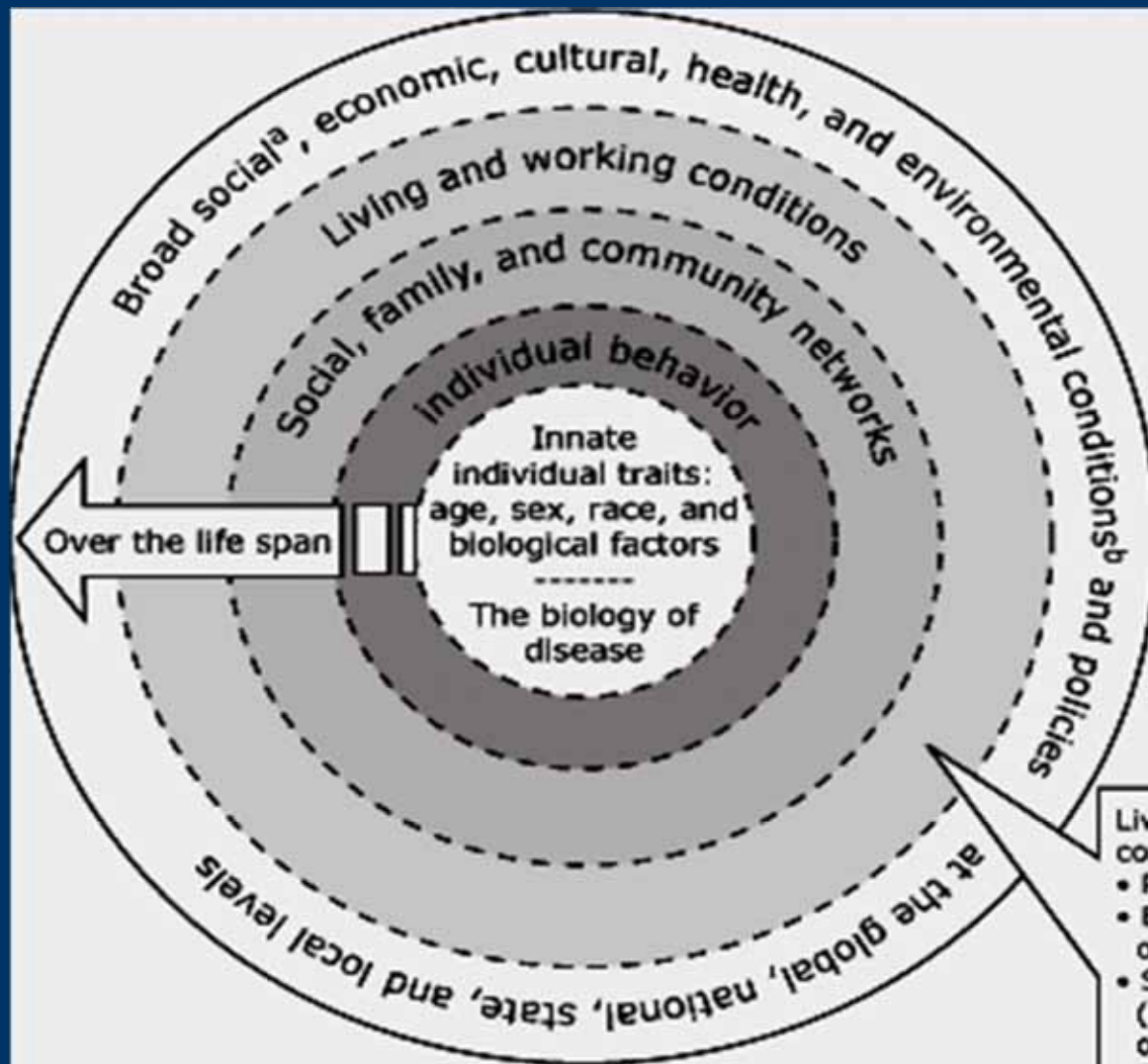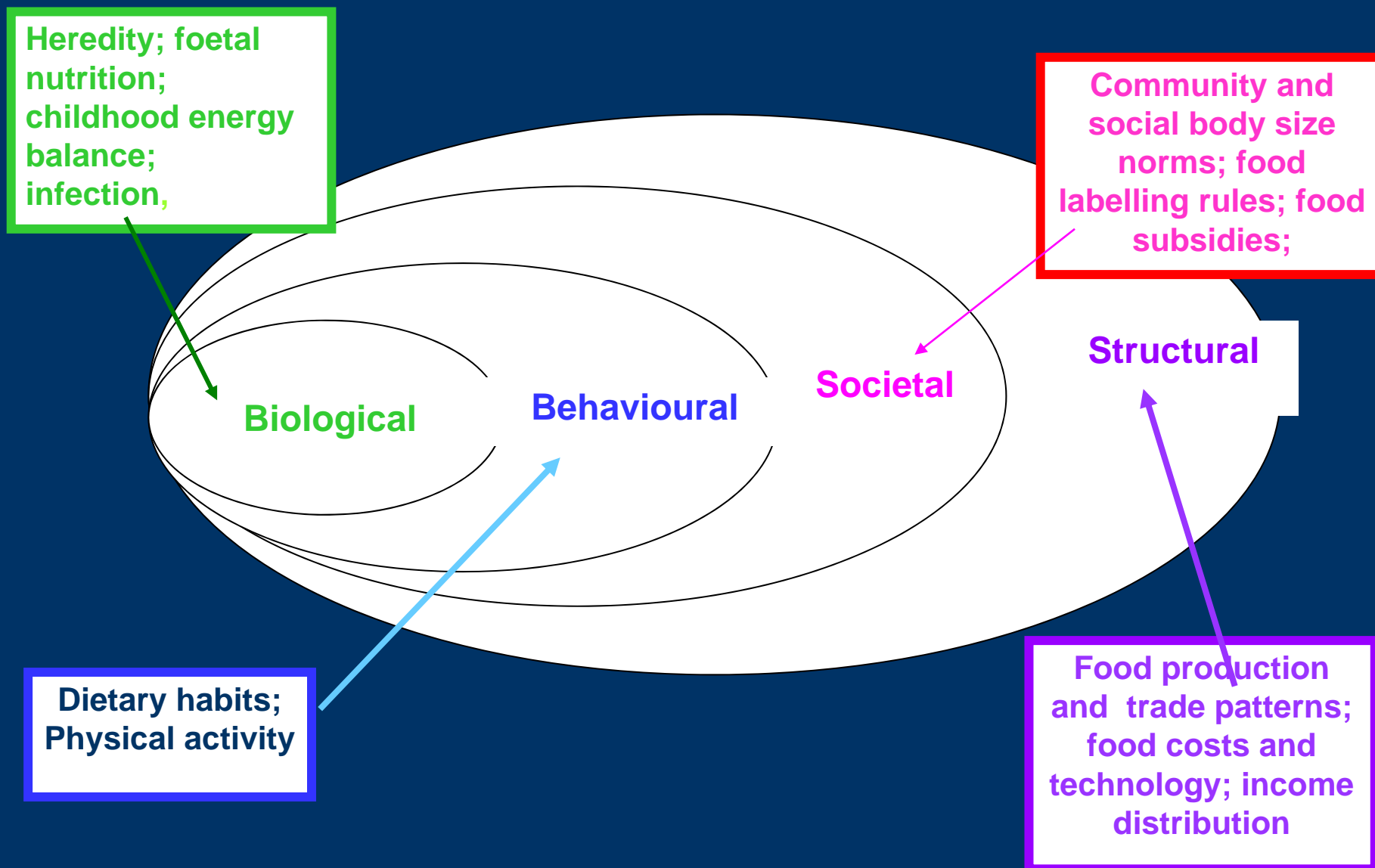

'**Social Ecology** - the study of the influence of the social context on behaviour, including institutional and cultural variables. (Sallis & Owen, 2002)

Broad social, economic, cultural, health, and environmental conditions and policies at the global, national, state, and local levels

Living and working conditions

Social, family, and community networks

individual behavior

Innate individual traits: age, sex, race, and biological factors
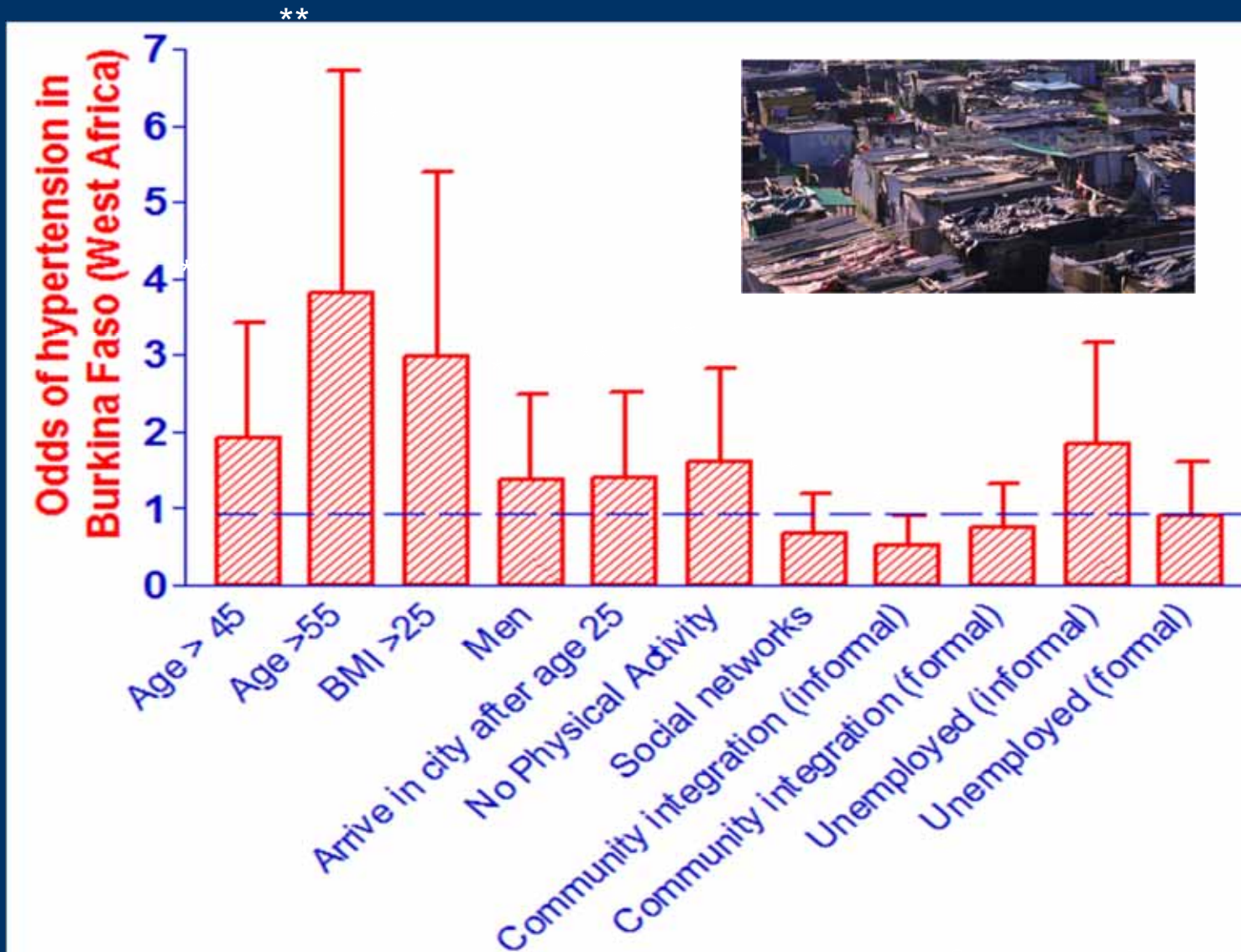
The biology of disease

Over the life span

Living and working conditions may include:
- Psychosocial factors
- Employment status and occupational factors
- Socioeconomic status (income, education, occupation)
- The natural and built environments
- Public health services
- Health care services

Russian doll framework: diabetes

Heredity; foetal nutrition; childhood energy balance; infection,

Community and social body size norms; food labelling rules; food subsidies;

Structural

Societal

Behavioural

Biological

Dietary habits; Physical activity

Food production and trade patterns; food costs and technology; income distribution

Odds of hypertension in urban West Africa: interaction with social disparities

(Niakara et al., *Trans Royal Soc Tropical Med Hygiene* 2007; 101: 1136-1142)

Slide courtesy Prof. V. Lambert

Built environment a contextual factor: physical activity (Can we extrapolate from Global North?)

London

Dar Es Salaam

Kampala

Copenhagen

Nairobi

(Forrest KY et al., Med Sci Sports Exerc. 2001 Sep;33(9):1598-604)

Where does "lifestyle" or "choice" fit in?

"I want to consume this product; should public health experts stop me?"

Anaesthetist Michael Keane says no

In Melbourne, a prominent billboard summarises: "Alcohol does not cause violence. Blame and punish the individual". Ironically, this simple message articulates a far more comprehensive understanding of the complete body of relevant knowledge than many public health academics who advocate reactionary, prohibition-like controls on the voluntary consumption of ever more products.

Public health traditionally focused on the health consequences of unwanted phenomena. Nobody wanted to drink faeces-flavoured water, but they did want convenient disposal of sewage.[1] In contrast, today's public health focuses increasingly on restricting the active and deliberate consumption of desired products and services, thus imposing government lifestyle mandates (GLMs) on the population.

Alcohol, fast food, cigarettes, shopping, soft drinks, gambling and other "vices" unquestionably bring utility as well as harm. What price for the enjoyment of, say, a night of alcohol intoxication? Only the individual knows the answer. The long-established principle of autonomy acknowledges that only the individual can apportion the appropriate weighting to each of the myriad factors in any harm–benefit calculation.

GLMs are health interventions, and, like any intervention, need to be consistent with contemporary medical ethics. Sensationalist studies of harm are inadequate to justify enforcing health interventions against peoples' will. Political scientist Eli Feiring summarises: "Given that respect for the autonomous choices of patients runs deep in modern healthcare, there are strong reasons to value the claim that competent and well-informed individuals are the best interpreters of their own interest and that they should be free to make choices others would regard as non-beneficial to them".[2]

Furthermore, it is meaningless to present the sum of harm resulting from a product without reference to fault. A fully established societal and legal principle is that harm caused to oneself is treated differently from harm caused to others. There is certainly no academic basis to nihilistically accept that people who are "glassed" in the face or killed by drunk drivers are merely victims of alcohol-related harm. The solution is not to apply prohibitionist measures to collectively punish everyone.

> The solution is not to apply prohibitionist measures to collectively punish everyone

government to run our lives. Admittedly, many public-health-inspired intrusions are minimal, such as the perennial example of seatbelts. But this example is then misused to justify extreme mission creep, up to and including prohibition-like measures. All behaviour can ultimately be coded as health-related in our system and, by reductio ad absurdum, a truly Orwellian state can be justified. Where does it end? Who decides? Yes, *Prime Minister*'s Sir Humphrey Appleby, or some other fictitious public servant?

A more potent concern with the nanny state is the propagation of the "disease" model, which promotes society-damaging, malignant lack of personal responsibility — "it's not my fault, it's my disease". Conceptualising the degree of responsibility for one's behaviour is at the intersection of neuroscience, ethics and, ultimately, philosophy within the burgeoning field of neuroethics.[5] If decision making is a function of the brain, should individuals really be held responsible for their decisions? With this neuroscientific and philosophical uncertainty, "addictions" should not be equated with other diseases in medicine. While the concept of addiction is complex and evolving,[6] the current tendency to regard even the most reckless, selfish and antisocial behaviour as the biologically bound phenomena of a particular product is essentially just an expression of opinion and ideology. Regarding autonomy, does an individual who is smoking, drinking, gambling, eating junk food and being indiscriminate about sexual partners ("sex addiction") really employ a sophisticated risk management equation and decide that the benefits outweigh the costs? Ultimately, public health advocates believe that such people are incapable of making the right decisions; we, the "elite" who know best, therefore have the right to decide what's good for them.

A further justification to usurp established ethical principles is based on the divisive and emotive argument that we have the right to control people because they cost us money in the form of health care expenditure. However, contradictory economic analyses on the real costs of alcohol, cigarettes and fatty foods abound. History and the human condition teach us that it would be dangerously naive to enforce interventions against peoples' will on the basis of conflicting, often ideologically inspired economic analyses. Crucially, again, government overreach would then make it legitimate to forcibly treat individuals against their will, if it meant a reduction in government expenditure.

Let me opt out.

Competing interests: No relevant disclosures.

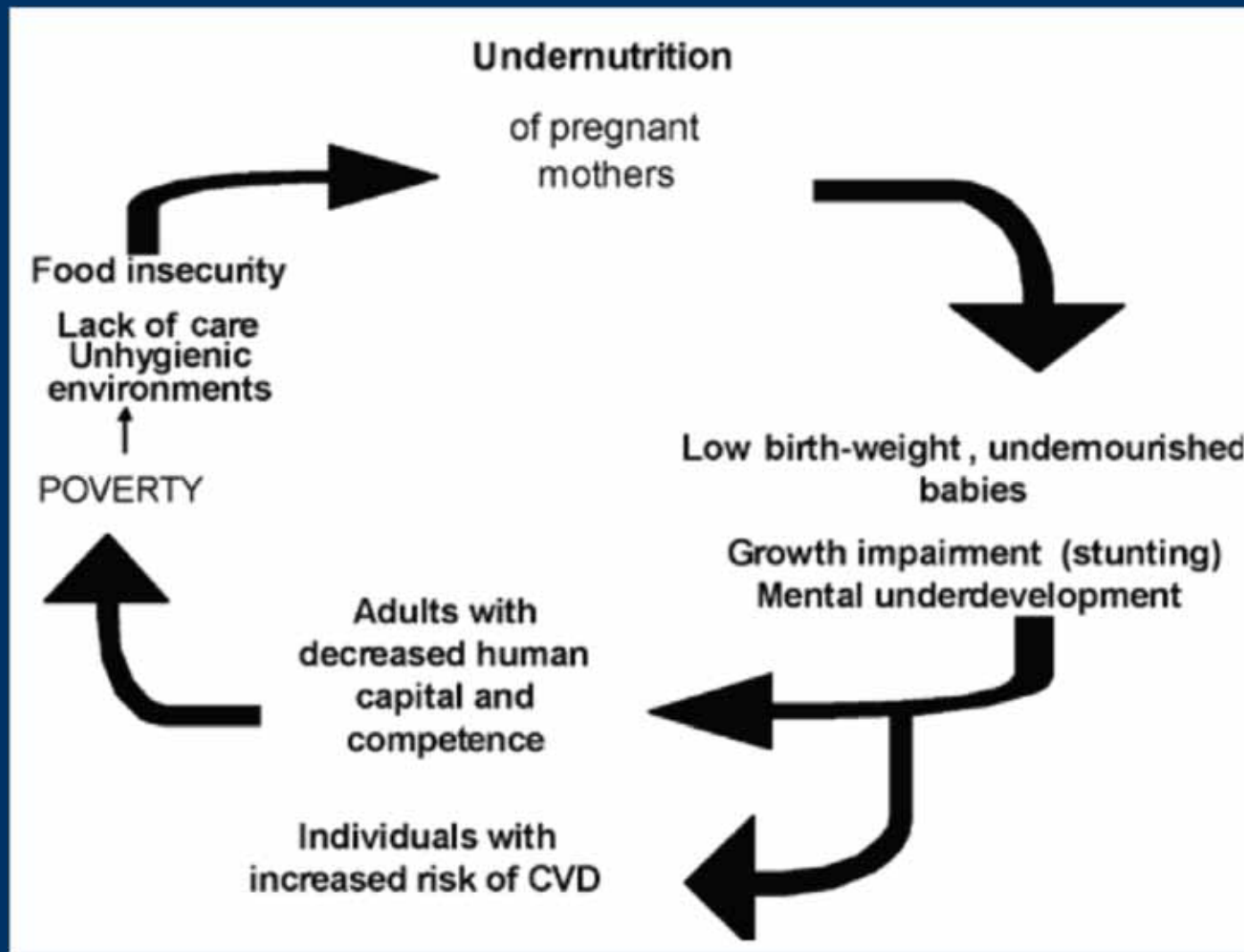Provenance: Commissioned; not externally peer reviewed.

1 Keane M. Public health interventions need to meet the same standards of

Developmental vs degenerative processes

Life course epidemiology

Intergenerational circle of undernutrition and poverty: risk of cardiovascular disease (CVD)

"Accumulation of risk" models

1.   With *independent and uncorrelated* insults or risk factors
        E.g. genetic predisposition plus smoking

2.   With *correlated insults*:

- 'Risk clustering' :
    E.g. high fat diet + smoking + lack of exercise + lower SES
      → adult cardiovascular disease
    - "Standard model"
    - Confounding a problem in epidemiological studies

- 'Chains of risk' with additive or trigger effects
    E.g. paternal alcoholism → family dysfunction →
    childhood abuse → adult depression
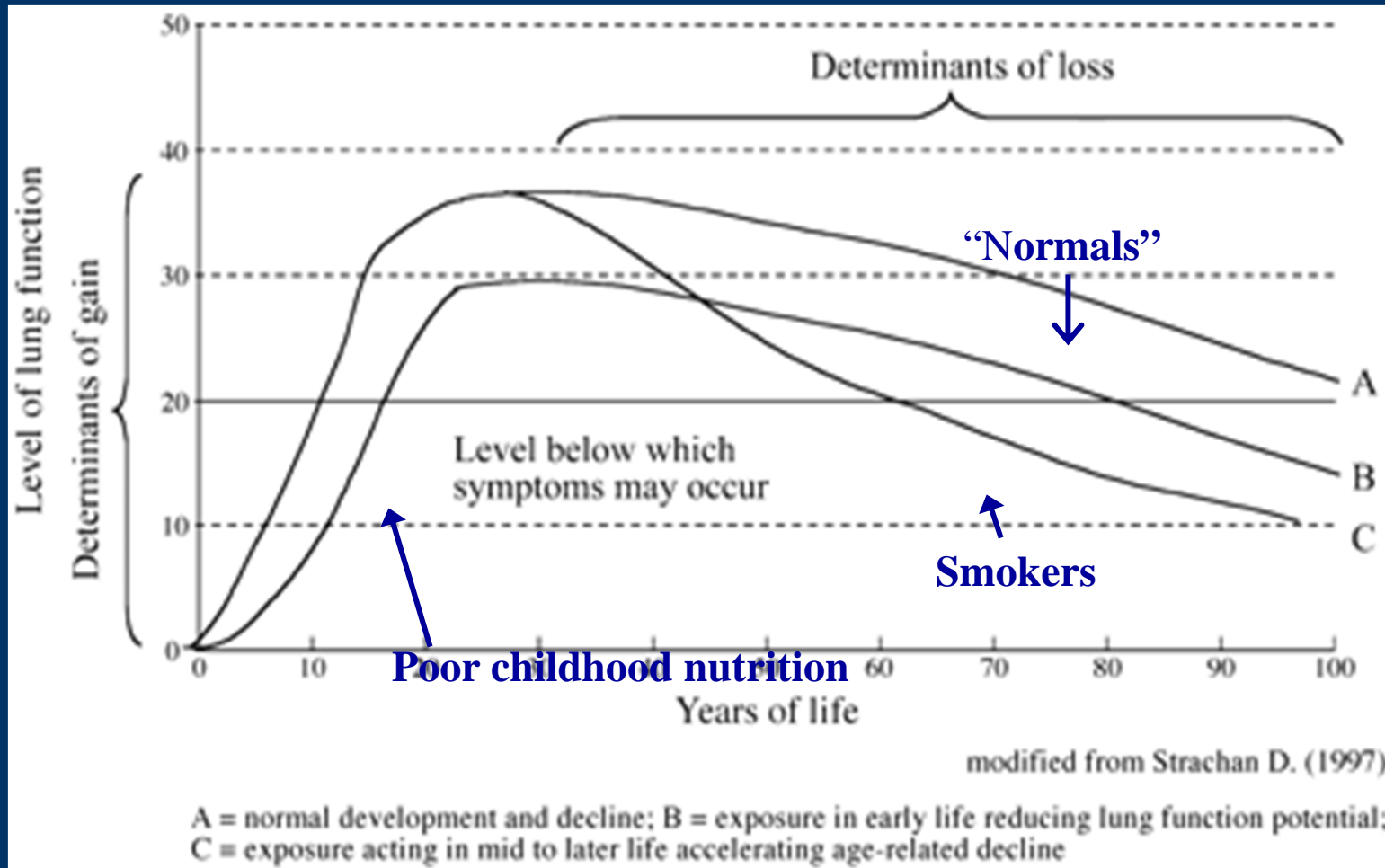
Ben Shlomo and Kuh

"Critical period" models

1. With or without added later life risk factors:

   E.g. *decreased* infective exposure → childhood asthma

2. With later life *effect modifiers:*

   E.g. *low* birth weight + *accelerated* weight gain in childhood and later life → increased risk of CVD, diabetes and hypertension

Developmental vs degenerative model: lung function

A = normal development and decline; B = exposure in early life reducing lung function potential; C = exposure acting in mid to later life accelerating age-related decline

NCD Epidemiology for Africa

Disease control/prevention

Approaches to cardiovascular *primary* prevention (i.e. avoid development of new cases)

« **Population or low risk strategy** »

- Public health approach
- Targets entire population
- Seeks small changes in highly prevalent risk factor
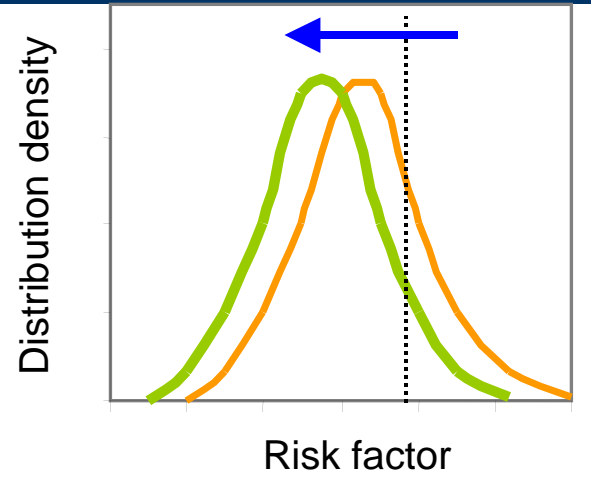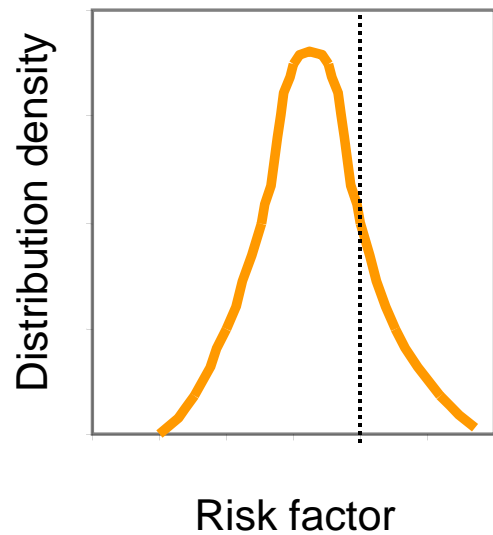- **Radical attempt** to deal with underlying causes

« **Individual or high-risk strategy** »

- Screening & treatment of risk factor
- Targets selected individuals
- Seeks to identify people with high risk and treat them
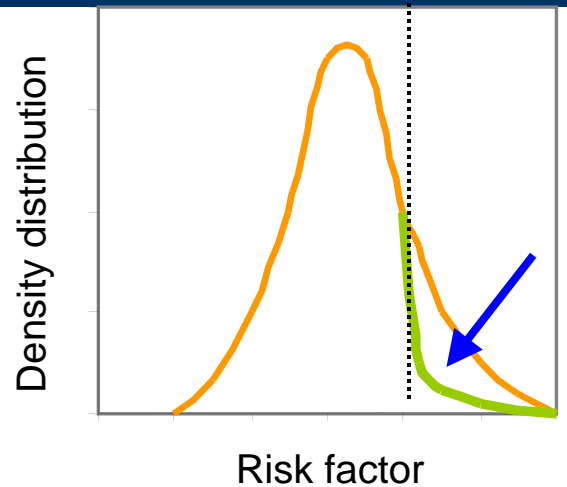- Rescue operation (delays consequences)

Bovet, P., Building Capacity

"Population" strategy vs high-risk strategy

Population (low risk) approach

Original distribution

Combined strategies

High risk approach

Bovet P., IUHPE/CDC Seminar Series: Building Capacity for CVD Health Promotion and Chronic Disease Prevention and Control in Africa, Entebbe, Uganda July 2009
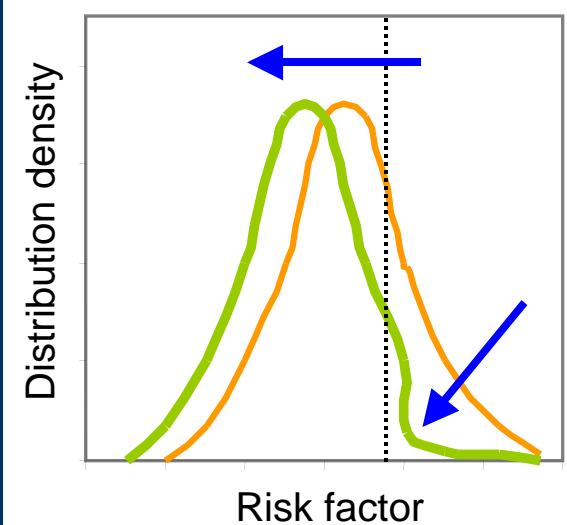
High risk strategy, e.g. *screen and treat*

Advantages

- Intervention appropriate to individual

- Subject motivation higher (own health)

- Physician motivation higher (own patient's health)

- Benefit: risk ratio viewed as favourable (if there is any risk, balanced by observable benefit)

Disadvantages

- Difficulties and high costs of *screening* individuals;

- Palliative and temporary—not radical. Have to repeat in each   generation;
- Limited potential for (a) individual
- (b) population;

- Predictive power of risk factors low; relative risk may be high, but attributable fraction may be low;

- Behaviourally "inappropriate" (individual has to deviate from norm).

Rose 1985

Low risk strategy (e.g. *regulate)*

Advantages

- Large potential benefit for entire community

- Can be sustained when life-style considered 'the norm'

- Does not require cooperation from individuals

- Can target unaware groups

Disadvantages

- Small benefit for individuals ('prevention paradox')

- Need for mass change is hard to communicate. (Poor motivation of individuals – most people regard themselves as low risk)

- Resisted by vested interests

- Doctor motivation weak

- Benefit: risk ratio worrisome - if *any* significant costs or risks - because so widespread (e.g. universal vaccination).

- Evidence of 'macro' measures hard to demonstrate

Rose 1985