Social Determinants of Infectious Disease

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Outline

• What is social epidemiology?

• Social patterning of infection
  – Examples

• Implications for prevention
Social Epidemiology

Emerged during the chronic disease era

• Term traced back to a 1950 article by Alfred Yankauer
  – The relationship of fetal and infant mortality to residential segregation: an inquiry into social epidemiology- *American Sociological Review*

• Term increased in the literature after 1969

Social Determinants

• Social Epidemiology
  – Focus on social conditions that promote or harm health rather than on specific outcomes

• Requires researchers to move from proximate to distal factors

Social Epidemiology Became a Study of Chronic Diseases

“field of inquiry that regards the role of social and psychological factors in the etiology of chronic diseases”
– Reynaud M. Rev Epidemiol Sante Publique. 1987;35:3-19

“a term which has recently come into favor to describe research concerned with social factors in the etiology of chronic disease”
The “Gradient” in chronic disease

- Wealthier, more educated people live longer, healthier lives (on average)

- Access to care and traditional behavioral factors do not entirely explain the gradient
Every meter in height of the grave, adjusted for year of death:

- 1.93 years (95% CI 1.06-2.80) later age at death for men
- 2.92 years (95% CI 1.76-4.08) later age at death for women

Died in 1800’s

Years of Education and Mortality

Age-adjusted Relative Risk of Mortality from Cox Models, National Longitudinal Mortality Study (NLMS), ~8 years follow-up time (males)
Socioeconomic and psychosocial gradients

- Cardiovascular disease
- Dementia and cognitive impairment
- Physical functioning
- Some cancers
Potential Pathway: Stress?

Lower socioeconomic position (SEP) associated with greater exposure to stressors such as:

– perceived financial strain
– job insecurity
– low job control
– negative life events
– unsafe residential environments
– discriminatory experiences
Hypothesized Pathways

STRESSORS

Central Nervous System

Endocrine hormones
Pituitary hormones
Endocrine hormones
Neuropeptides
Immunotransmitters

Endocrine System

Immune System

Figure from D.N. Khansar et al. (1990) *Immunol. Today* 11:170
What is the evidence for a relationship between social factors and infection in the US?
Today

• Social, political, behavioral and environmental factors are widely accepted as forces shaping emergence and reemergence of pathogens
Childhood Socioeconomics and Colds

Ownership from ages 1-9 was a more important marker than adolescence

Critical period hypothesis?

Figure from: Cohen et al. (2004) Childhood socioeconomic status and host resistance to infectious illness in adulthood. *Psychosomatic Med* 66:553-8
Hepatitis B Virus (HBV)

Chronic hepatitis

Hepatocellular carcinoma

Post-transplant accelerated atherosclerosis

Cytomegalovirus (CMV)

Helicobacter pylori

Peptic Ulcer Disease, Gastric lymphoma
Human Papilloma Virus (HPV)  
Varicella zoster  
Streptococcus pyogenes  

Cervical Carcinoma  
Shingles, neuropathy  
Rheumatic heart disease
Implicated Pathogens
Crohn’s disease

Clostridium

C. jejuni

Mycobacterium paratuber

HTLV-1, EBV

EBV

Chronic Fatigue Syndrome
Cytomegalovirus (CMV), Herpes Simplex Virus (HSV), *Chlamydia pneumoniae*, *Helicobacter pylori*, periodontal bacteria, hepatitis A virus (HAV)

*Toxoplasma gondii*, HSV-2, CMV, prenatal exposure to viruses/influenza

Cardiovascular disease

Schizophrenia, Dementia
Social Patterning of *H. pylori*


Social Patterning of *H. pylori*: importance of childhood

Figure from: Malaty, HM & Graham, DY. 1994. Gut. 35;742-745
Prevalence of HPV among US Women (N=1,921) ages 14-59

Data from: Kahn, JA et al.(2007) Obstetrics and Gynecology. 110(1):87-95
Age Adjusted Prevalence of Infection by Education, NHANES

CMV immune response by Age and Education, NHANES

*Adjusted for race/ethnicity, income, and sex. Censored regression of education in years: slope=-0.05 (0.02) p <0.01

CMV immune response by Age and Income, NHANES

*Adjusted for race/ethnicity, education, and sex. Censored regression of log family income: slope=-0.25 (0.07), p <0.01

Figure From: Epstein, SE. (2002) The multiple mechanism by which infection may contribute to atherosclerosis development and course. *Circulation Research*. 90:204
Pathogen Burden by Race and Education, NHANES

Disparities in A H1N1

• Race/ethnic disparities in infection
  – Boston Public Health

• Race/ethnic disparities in medical treatment
  – Chicago and Boston

• Prevention measures? Vaccines, antivirals, NPIs?
Dynamics of infection

- My disease status affects your disease status
  - Not independent units

  - Reproductive number
Reproductive number $R_0$

• “Are zero” or “are-naught”
  – From general population theory

• $R_0 = \text{expected number of secondary infectious cases that one infectious host will produce during his or her infectious period in a large population that is completely susceptible}$
Reproductive number $R_0$

Formula

$$R_0 = \text{Number of contacts per unit time} \times \text{Probability of transmission per contact} \times \text{Mean duration of infectiousness}$$

$c \times p \times d$
Public Health Standpoint and $R_0$

- For an epidemic to occur in a susceptible population $R_0 > 1$

- If $R_0 < 1$ an average case will not reproduce itself

- Caveat: since it is an average it is possible to have an $R_0 < 1$ with a case that causes more than one infective case.
  - A sustaining outbreak is very unlikely
## CMV in ages 12-49 in US

<table>
<thead>
<tr>
<th>Variable</th>
<th>Reproductive Ratio</th>
<th>Average Age at Infection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Entire US</td>
<td>1.7</td>
<td>28.6</td>
</tr>
<tr>
<td>Male</td>
<td>1.7</td>
<td>28.0</td>
</tr>
<tr>
<td>Female</td>
<td>1.6</td>
<td>29.1</td>
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<tr>
<td>NH Black</td>
<td>4.1</td>
<td>16.3</td>
</tr>
<tr>
<td>Mexican American</td>
<td>3.7</td>
<td>17.5</td>
</tr>
<tr>
<td>NH White</td>
<td>1.6</td>
<td>29.3</td>
</tr>
<tr>
<td>Income Low</td>
<td>2.7</td>
<td>21.9</td>
</tr>
<tr>
<td>Income Mid</td>
<td>1.9</td>
<td>26.7</td>
</tr>
<tr>
<td>Income High</td>
<td>1.6</td>
<td>28.9</td>
</tr>
</tbody>
</table>

*Primary CMV infections per 100 seronegative persons per year*

Table from: Colugnati FA, Staras SA, Dollard SC, Cannon MJ. BMC Infect.Dis. 2007. 7:71.
Immunization and $R_0$

• Immunization can be used to reduce the number of susceptible individuals

• What fraction do we need to vaccinate so that we produce enough immune whereby infective people are no longer able on average to infect one other person?
Immunization and R0

• Assume R0 = 2 for CMV in US population
  – Fraction (f) that needs to be immunized before the age of first infection is
    • f = 1 - (1/ R0)
    • 1 - (1/2) = 50%

• A higher R0 requires immunization of a higher fraction to eliminate transmission
  • Implications for SEP and race/ethnic groups
Conclusions

Strong SEP differentials in many infections in the US

- Affect wages, schooling, education attainment, occupational attainment

• Disparities affect disease dynamics leading to persistence in disparities over time and space in susceptible groups

Graphic: Want to help stop the spread of H1N1 influenza? Yes, you can! Courtesy of Ben Heine
Conclusions Cont.

• Some evidence to suggest a role for psychosocial factors in immune response

• Targeting interventions at social and biological level
  – Vaccinations?
  – Reducing poverty, improving living standards, nutrition, and lowering stress
"If two susceptible subjects are exposed to equal doses of the same germ, and one develops infection while the other does not, the factor governing the development of the infection clearly lies outside the germ."

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