Epidemiology in Action

Edgar P. Simard PhD MPH
Lenox Hill Hospital
April 24th 2015

Agenda

- Definitions, brief history
- Example studies
- Internet, Big Data, and modern information management
- Questions

What is epidemiology?

- Who
- What
- Where
- When
- How

My definition: “The study of the distribution and determinants of disease in different populations”

Typically “population-based” vs. “hospital-based”

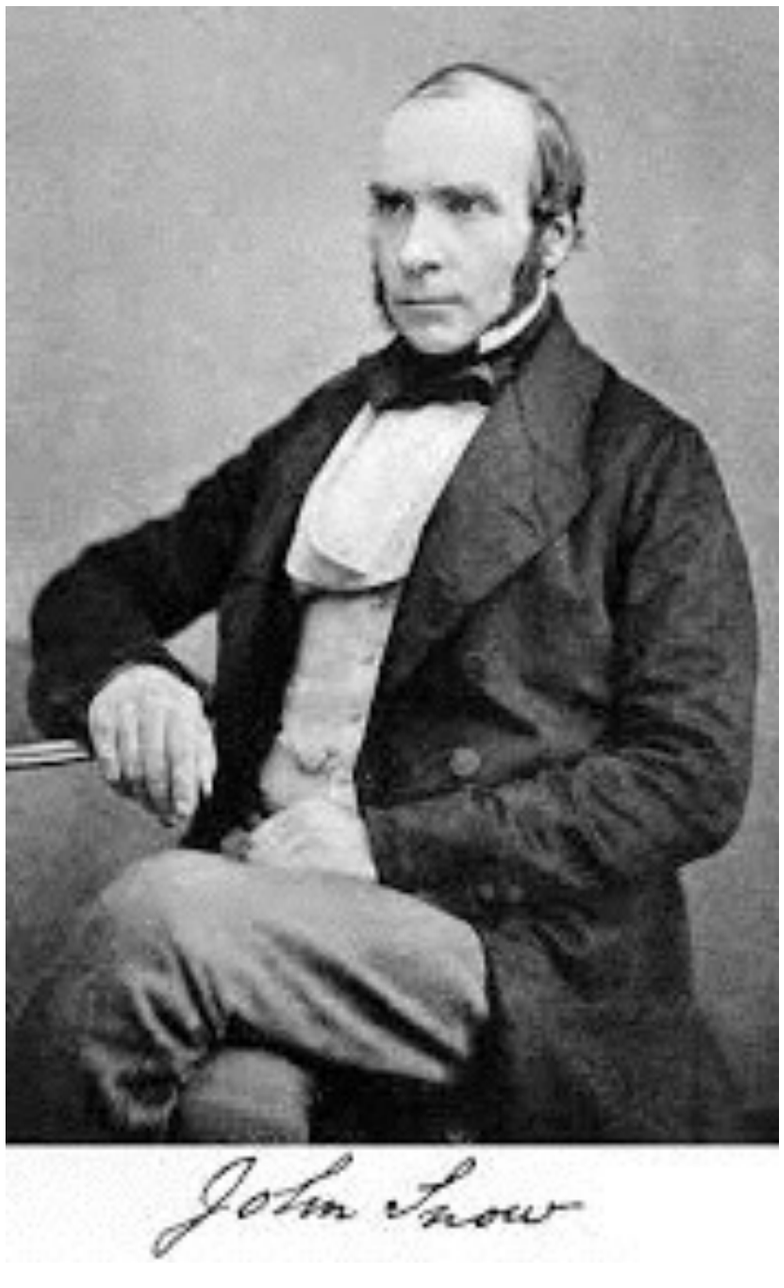
Unit of inference is the “population”

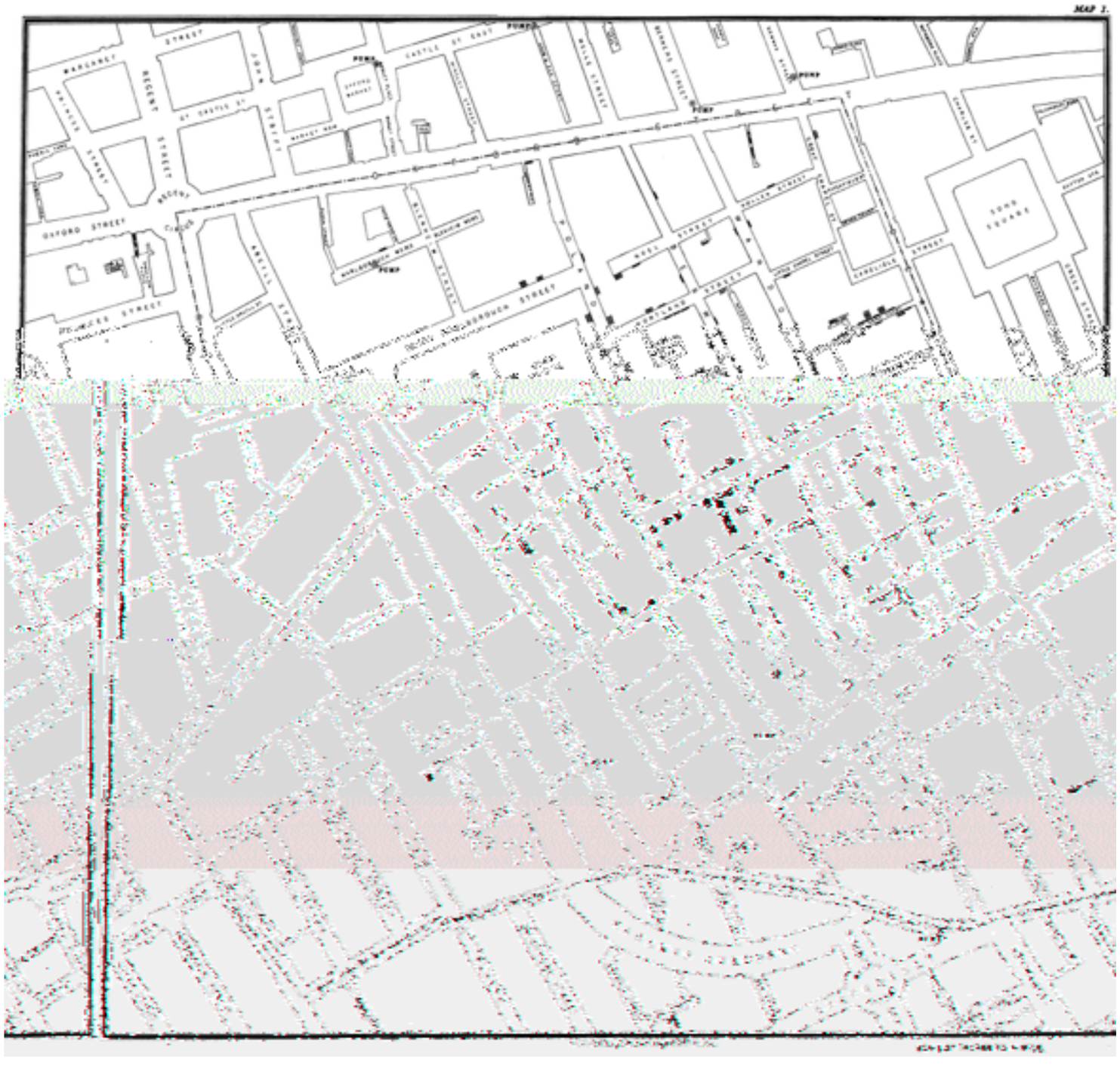
Epidemiology

Epidemiology is the science that studies the patterns, causes, and effects of health and disease conditions in defined populations. It is the cornerstone of public health, and informs policy decisions and evidence-based practice by identifying risk factors for disease and targets for preventive healthcare. Epidemiologists help with study design, collection, and statistical analysis of data, and interpretation and dissemination of results (including peer review and occasional systematic review). Epidemiology has helped develop methodology used in clinical research, public health studies, and, to a lesser extent, basic research in the biological sciences

Wikipedia

Historical Milestones





Modern Epidemiology

Molecular Mechanisms	Physical Activity	Population Health
Developmental Origins	Dietary Intake	Solution-oriented Research
Genetics/ Epigenetics	Drug Safety	Policy/ Practice

Etiology → **Prevention**

Canonical Themes: Bradford Hill Criteria

Strength: A small association does not mean that there is not a causal effect, though the larger the association, the more likely that it is causal.

Consistency: Consistent findings observed by different persons in different places with different samples strengthens the likelihood of an effect.

Specificity: Causation is likely if a very specific population at a specific site and disease with no other likely explanation. The more specific an association between a factor and an effect is, the bigger the probability of a causal relationship.

Temporality: The effect has to occur after the cause (and if there is an expected delay between the cause and expected effect, then the effect must occur after that delay).

Biological gradient: Greater exposure should generally lead to greater incidence of the effect. However, in some cases, the mere presence of the factor can trigger the effect. In other cases, an inverse proportion is observed: greater exposure leads to lower incidence.

Plausibility: A plausible mechanism between cause and effect is helpful (but Hill noted that knowledge of the mechanism is limited by current knowledge).

Coherence: Coherence between epidemiological and laboratory findings increases the likelihood of an effect. However, Hill noted that "... lack of such [laboratory] evidence cannot nullify the epidemiological effect on associations".

Experiment: "Occasionally it is possible to appeal to experimental evidence".

Analogy: The effect of similar factors may be considered.

Canonical Themes:

- Types of studies:
  Interventional (RCTs) versus Observational

- Outcomes:
  Rates of disease, death, disability, QoL, etc.

- Methods complications: confounding, interaction, bias

- Places where epidemiology is conducted:
  Everywhere: government (federal, state, local), NGO, academia, private sector (consulting, pharma), international, etc.

- Types of work:
  Field studies, interventions, surveillance, data analyses, policy implications (cost effectiveness, comparative effectiveness), Highlight work works, what doesn't, who is left out, ways to advance public health

Leadership Centers

CDC

NIH

National Institutes of Health

Columbia University

American Cancer Society

Field Studies and Policy Implications

Hepatitis A Vaccine

ORIGINAL CONTRIBUTION

Control of Hepatitis A Through Routine Vaccination of Children

Francisco Averhoff, MD, MPH
Craig N. Shapiro, MD
Beth P. Bell, MD, MPH
Insu Hyams, BSRN
Leslie Burd, BA
Adeline Deladisma, MPH
Edgar P. Simard, BS
David Nalin, MD
Barbara Kuter, PhD
Chester Ward, MD
Mark Lundberg, MD, MPH
Natalie Smith, MD, MPH
Harold S. Margolis, MD

Context The impact of routine hepatitis A vaccination of children living in large communities with elevated disease rates has not been evaluated.

Objective To determine the effect of routine vaccination of children on disease incidence in a community with recurrent hepatitis A epidemics.

Design, Setting, and Participants Community-based demonstration project conducted from January 12, 1995, through December 31, 2000, in Butte County, California, among children aged 2 to 17 years.

Intervention In 1995, vaccination was offered to children aged 2 to 12 years during vaccination clinics conducted on 2 occasions 6 to 12 months apart at most schools in the county. In 1996-2000, vaccine was distributed to community health care clinicians, who vaccinated eligible children without charge. Vaccine was also available at health department clinics, selected child care centers, and other sites.

Main Outcome Measures Hepatitis A vaccination coverage, hepatitis A incidence, and vaccine effectiveness.

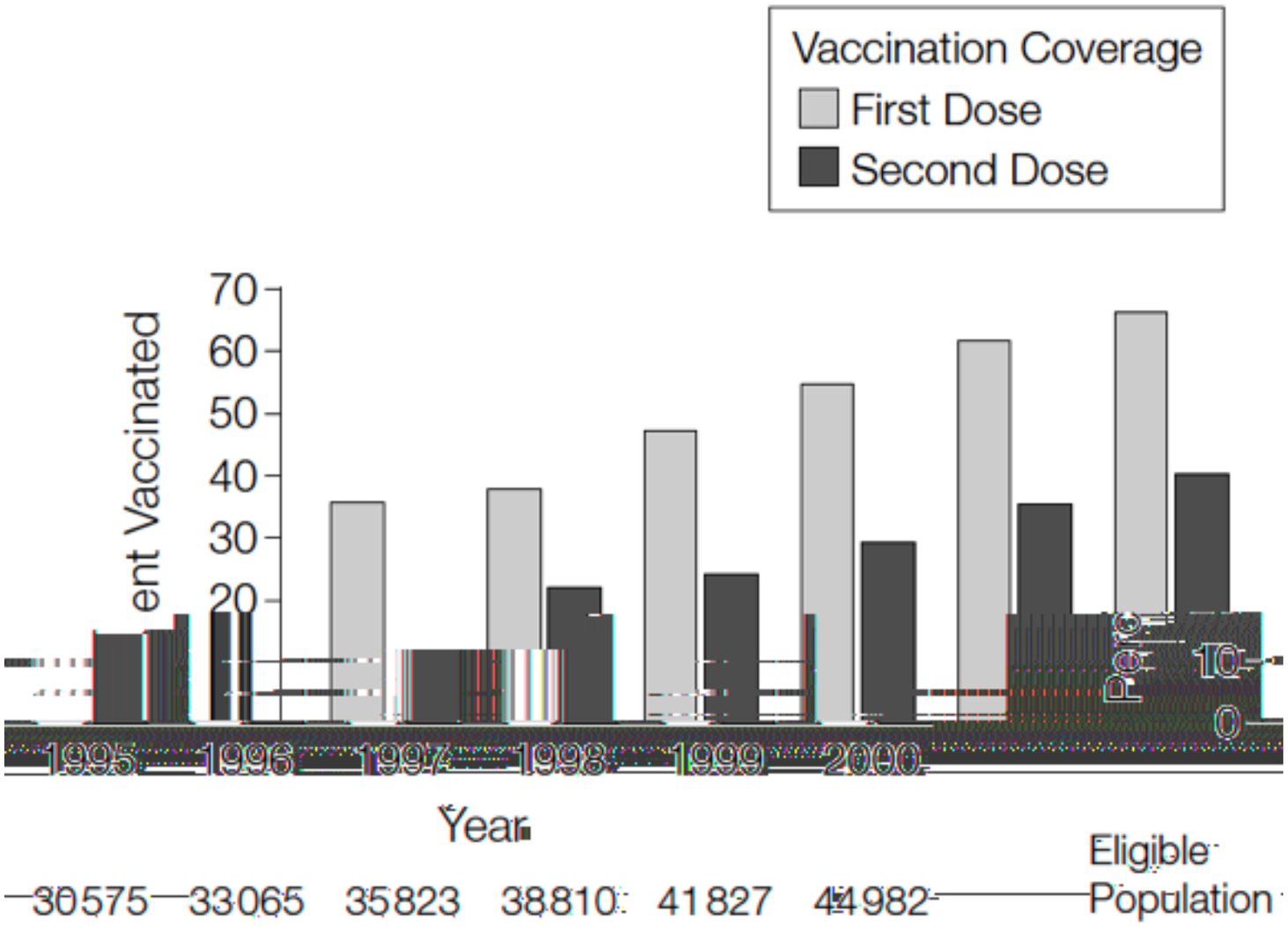
Results During the study period, 29 789 (66.2%) of an estimated 44 982 eligible children received at least 1 vaccine dose; 17 681 (39.3%) received a second dose. The number of hepatitis A cases among the entire county population declined 93.5% during the study period, from 57 cases in 1995 to 4 in 2000; the lowest number of cases reported in the county since hepatitis A surveillance began in 1966. The 2000 incidence rate of 1.9 per 100 000 population was the lowest of any county in the state. Of the 245 cases reported during the 6-year period, 40 (16.3%) occurred among children 17 years of age or younger, of which 16 (40%) occurred in 1995 and only 1 in 2000. One of the 27 case patients eligible for vaccination had been vaccinated, having received the first dose 3 days before symptom onset. The estimated protective vaccine efficacy was 98% (95%-confidence-interval, 86%-100%).

Conclusions In this population, hepatitis A vaccine was highly effective in preventing disease among recipients. Childhood vaccination appears to have decreased hepatitis A incidence among children and adults and controlled the disease in a community with recurrent epidemics.

JAMA. 2001;286:2968-2973 www.jama.com

HEPATITIS A CONTINUES TO BE one of the most frequently reported vaccine-preventable diseases in the United States. Hepatitis A incidence displays a cyclic pattern, and most disease occurs in the context of community-wide outbreaks during which a large proportion of patients do not have a recognized risk factor.[1-6] Available data suggest that young children, frequently asymptomatic when infected, play an important role in hepatitis A virus (HAV) transmission.[7-12] Until recently, immune globulin and im-

Figure 1. Hepatitis A Vaccination Coverage Among Children by Year in Butte County, California, 1995-2000



ıps varied by year: 1995 includes 2- to 12-
; 1996 includes 2- to 13-year-olds; 1997 in-
to 14-year-olds; 1998 includes 2- to 15-
; 1999 includes 2- to 16-year-olds; and 2000
2- to 17-year-olds.

Age grou
year-olds
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Figure 2. Reported Hepatitis A Cases in Butte County, California, 1980-2000 (n = 1558)

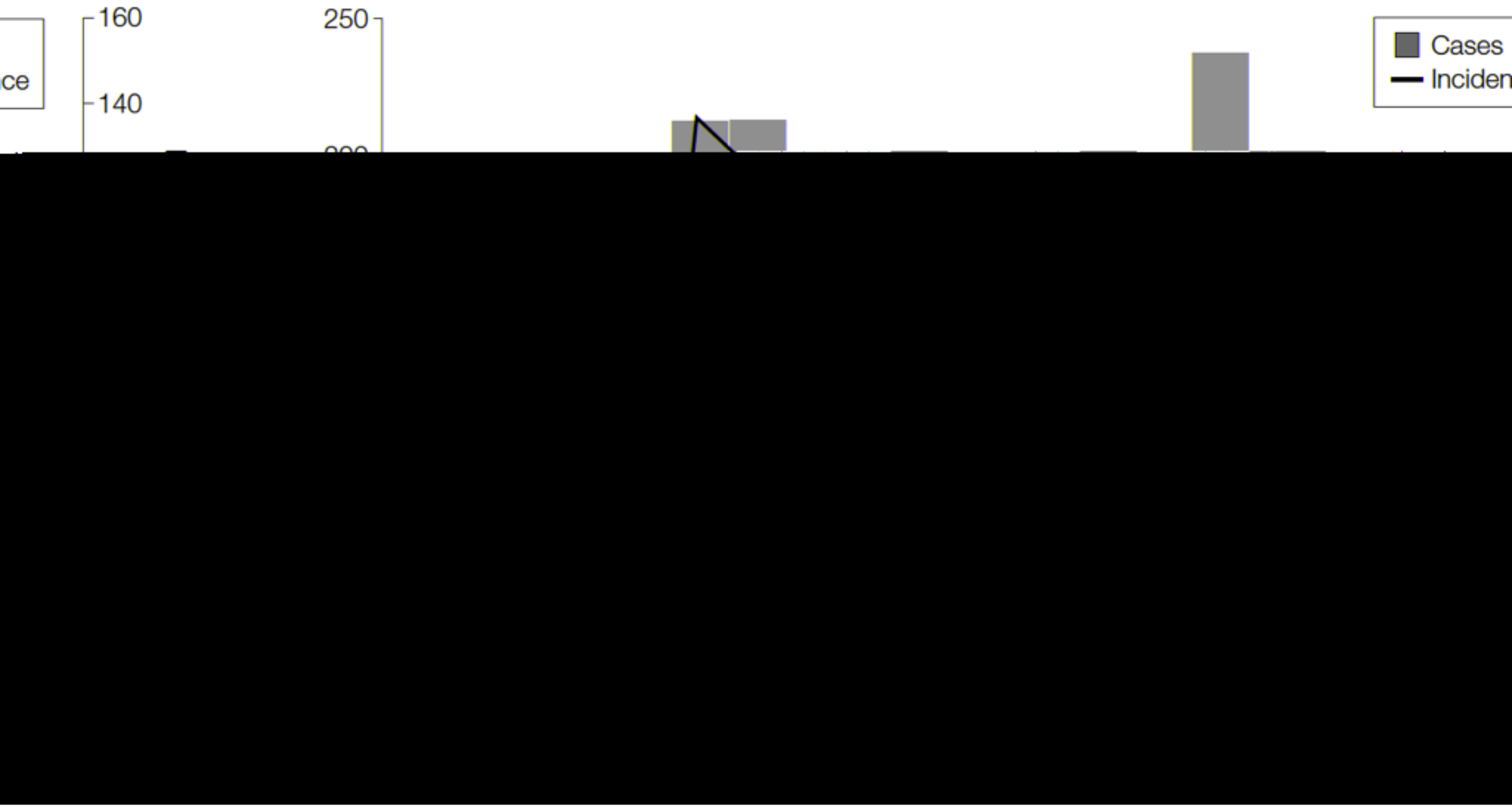


Figure 3. Average Annual Age-Specific Hepatitis A Incidence in Butte County, California, 1990-1994 and 1995-2000

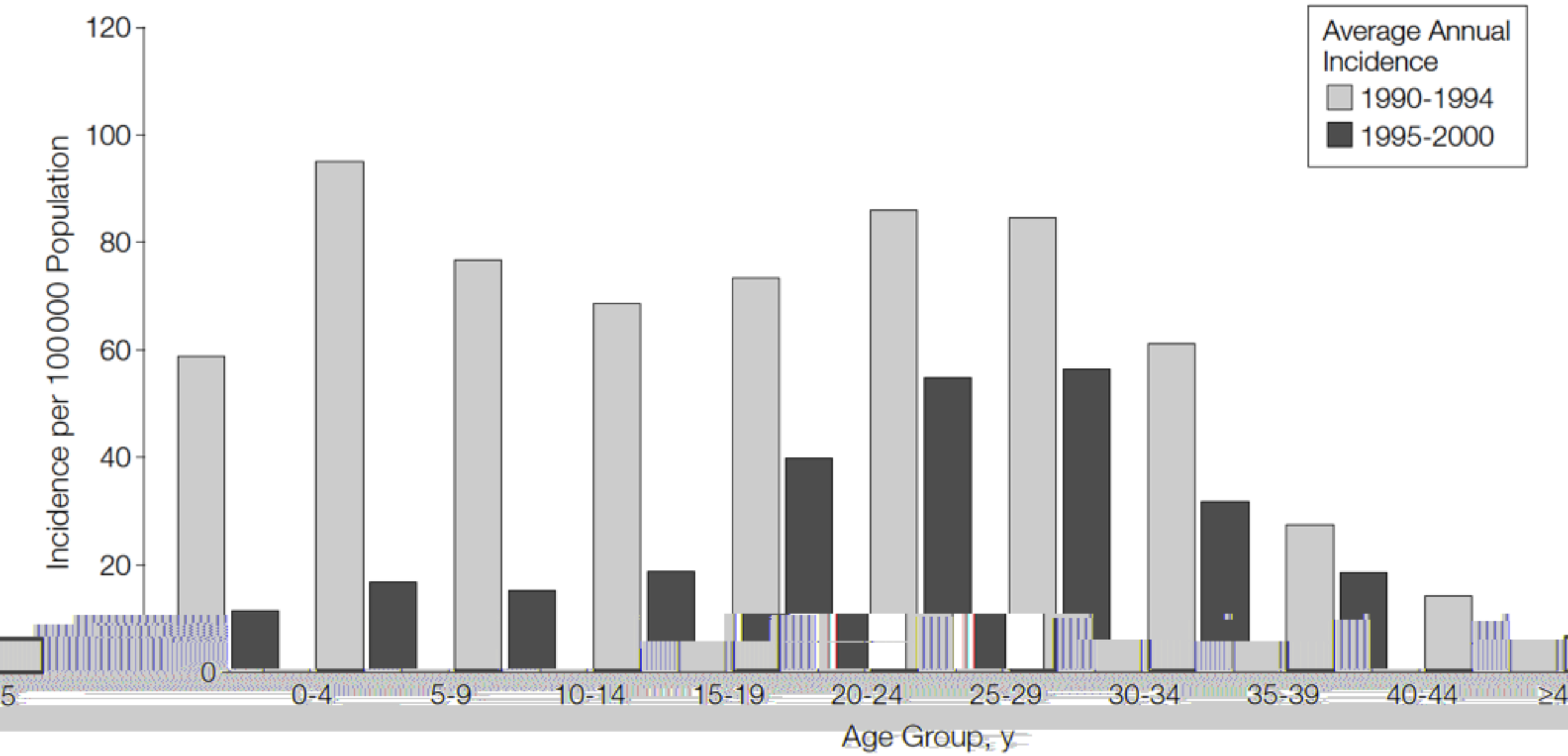


Figure 4. Hepatitis A Annual Incidence in Butte County, California, and All of California, 1990-2000

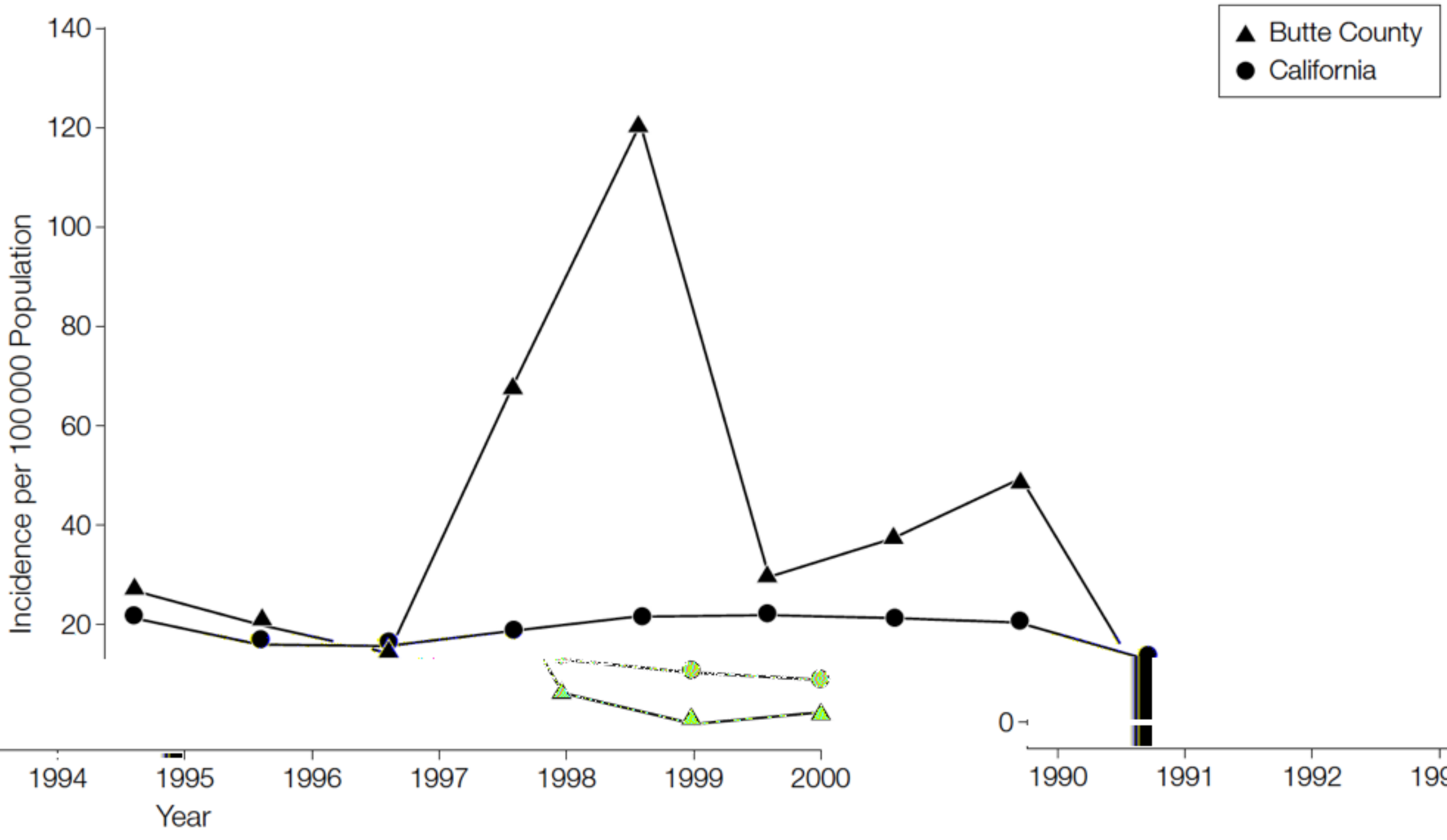
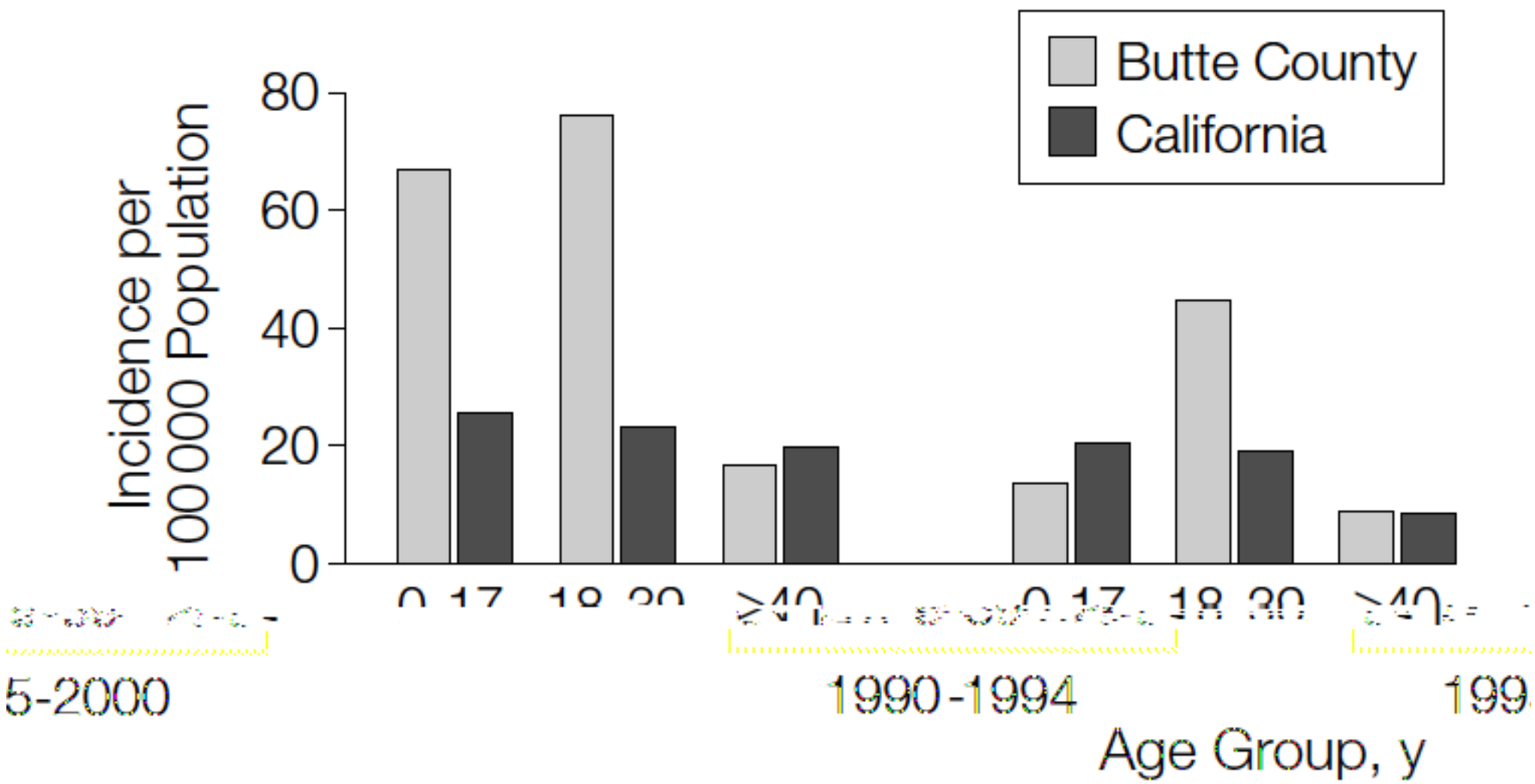


Figure 5. Average Annual Hepatitis A Incidence by Age Group for Butte County, California, and All of California, 1990-1994 and 1995-2000



CDC

MMWR™

Morbidity and Mortality Weekly Report

Recommendations and Reports May 19, 2006 / Vol. 55 / No. RR-7

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Prevention of Hepatitis A Thro or Passive Immunizati

Committee ACIP)

Recommendations of the Advisory on Immunization Practices (

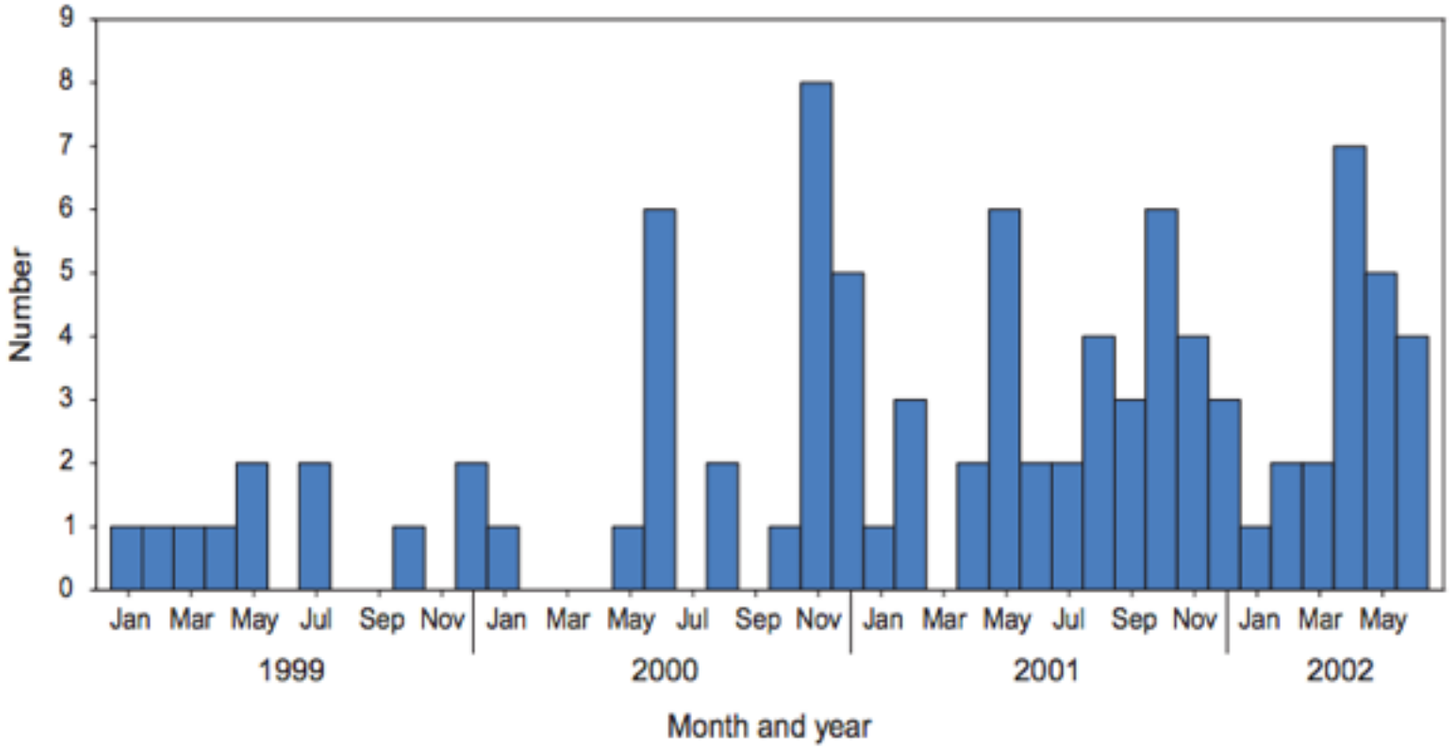
CDC
MMWR
Morbidity and Mortality Weekly Report
Weekly August 6, 2004 / V

Transmission of Hepatitis B Virus in Correctional Facilities — Georgia, January 1999–June 2002

Incarcerated persons have a disproportionate burden of infectious diseases (*1*), including hepatitis B virus (HBV) infection. Among U.S. adult prison inmates, the overall prevalence of current or previous HBV infection ranges from 13% to 47%. The prevalence of chronic HBV infection among inmates is approximately 1.0%–3.7%, two to six times the prevalence among adults in the general U.S. population (*1*).

persons can acquire HBV infection in the com-
correctional settings (*1*). This report summa-
lts of 1) an analysis of hepatitis B cases among
ates reported to the Georgia Department of
urces, Division of Public Health (DPH) during
–June 2002, including a retrospective investiga-
eported during January 2001–June 2002; and 2)
urvey conducted in prison intake centers during
ırch 2003. These efforts identified cases of acute
multiple Georgia prisons and documented evi-
oing transmission of HBV in the state correc-
The findings underscore the need for hepatitis B
rograms in correctional facilities.
ia correctional system houses approximately
es in 68 correctional facilities; approximately
nmates are admitted each year and processed
of five intake centers. The correctional system
nely screen inmates for HBV infection, and
ing is left to the judgment of individual physi-
cians. In August 2000, in response to two hepatitis B

FIGURE. Number* of cases of acute hepatitis B reported in correctional facilities, by month and year — Georgia, January 1999–June 2002



* N = 92.

Annals of Internal Medicine

ARTICLE

The Prevalence of Hepatitis C Virus Infection in the United States, 1999 through 2002

Gregory L. Armstrong, MD; Annemarie Wasley, ScD; Edgar P. Simard, MPH; Geraldine M. McQuillan, PhD; Wendi L. Kuhnert, PhD; and Miriam J. Alter, PhD

Background: Defining the primary characteristics of persons infected with hepatitis C virus (HCV) enables physicians to more easily identify persons who are most likely to benefit from testing for the disease.

Objective: To describe the HCV-infected population in the United States.

Design: Nationally representative household survey.

Setting: U.S. civilian, noninstitutionalized population.

Participants: 15 079 participants in the National Health and Nutrition Examination Survey between 1999 and 2002.

Measurements: All participants provided medical histories, and those who were 20 to 59 years of age provided histories of drug use and sexual practices. Participants were tested for antibodies to HCV (anti-HCV) and HCV RNA, and their serum alanine aminotransferase (ALT) levels were measured.

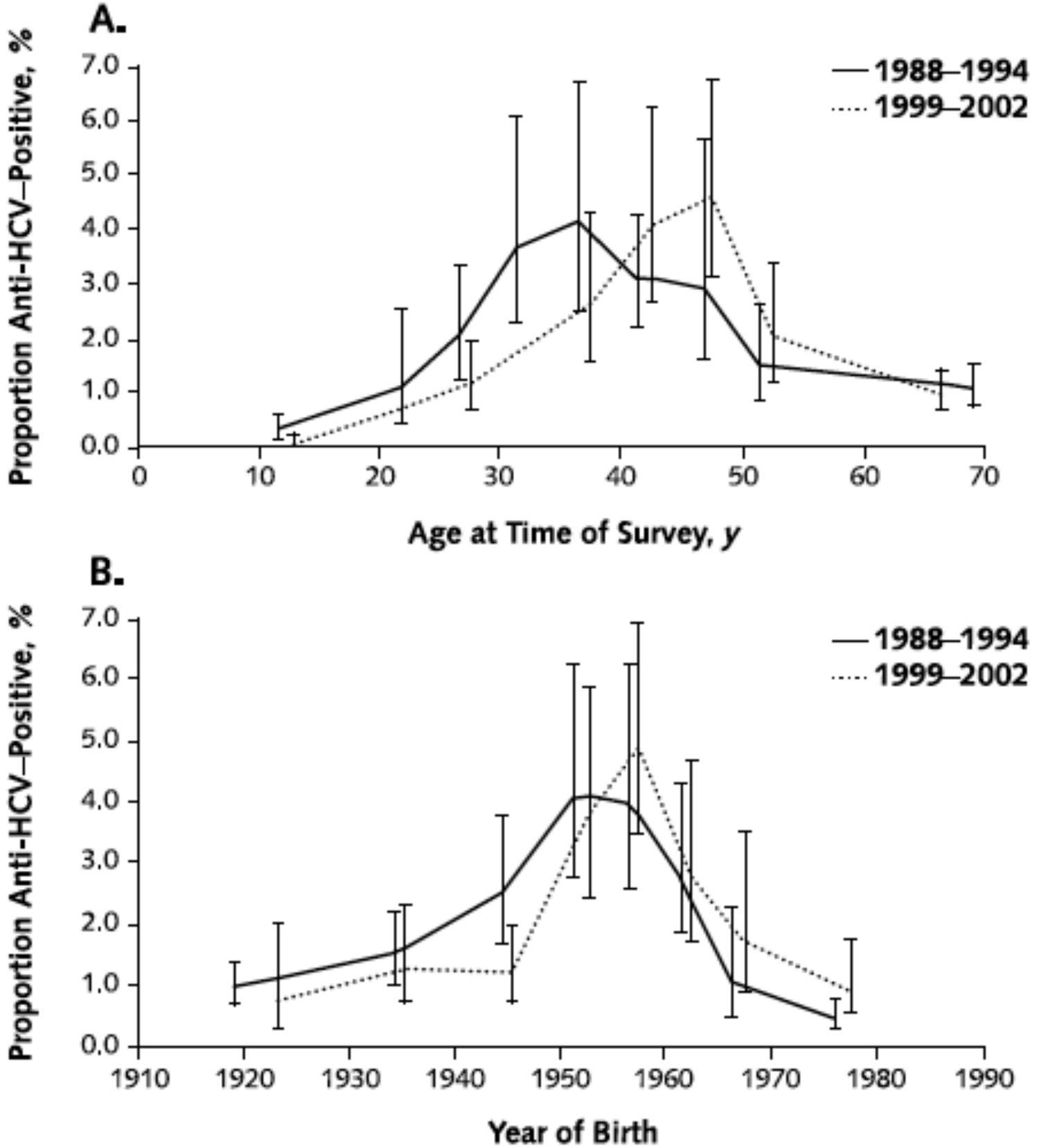
Results: The prevalence of anti-HCV in the United States was 1.6% (95% CI, 1.3% to 1.9%), equating to an estimated 4.1 million (CI, 3.4 million to 4.9 million) anti-HCV–positive persons nationwide; 1.3% or 3.2 million (CI, 2.7 million to 3.9 million) persons had chronic HCV infection. Peak prevalence of anti-HCV (4.3%) was observed among persons 40 to 49 years of age. A total of 48.4% of anti-HCV–positive persons between 20 and 59 years of age reported a history of injection drug use, the strongest risk factor for HCV infection. Of all persons reporting such a history, 83.3% had not used injection drugs for at least 1 year before the survey. Other significant risk factors included 20 or more lifetime sex partners and blood transfusion before 1992. Abnormal serum ALT levels were found in 58.7% of HCV RNA–positive persons. Three characteristics (abnormal serum ALT level, any history of injection drug use, and history of blood transfusion before 1992) identified 85.1% of HCV RNA–positive participants between 20 and 59 years of age.

Limitations: Incarcerated and homeless persons were not included in the survey.

Conclusions: Many Americans are infected with HCV. Most were born between 1945 and 1964 and can be identified with current screening criteria. History of injection drug use is the strongest risk factor for infection.

Ann Intern Med. 2006;144:705-714. www.annals.org

For author affiliations, see end of text.


A.
Proportion Anti-HCV–Positive, %
7.0
6.0
5.0
4.0
3.0
2.0
1.0
0.0
0
10
20
30
40
50
60
70
Age at Time of Survey, y
1988–1994
1999–2002
B.
Proportion Anti-HCV–Positive, %
7.0
6.0
5.0
4.0
3.0
2.0
1.0
0.0
1910
1920
1930
1940
1950
1960
1970
1980
1990
Year of Birth
1988–1994
1999–2002

Analyses of Existing Data and Policy Implications

ORIGINAL INVESTIGATION

Spectrum of Cancer Risk Late After AIDS Onset in the United States

Edgar P. Simard, PhD, MPH; Ruth M. Pfeiffer, PhD; Eric A. Engels, MD, MPH

Background: Persons living with AIDS today remain at elevated cancer risk. Highly active antiretroviral therapy (HAART), widely available since 1996, prolongs life, but immune function is not fully restored. We conducted this study to assess long-term cancer risk among persons with AIDS relative to the general population and the impact of HAART on cancer incidence.

Methods: Records of 263 254 adults and adolescents with AIDS (1980-2004) from 15 US regions were matched to cancer registries to capture incident cancers during years 1 through 5 and 6 through 10 after AIDS onset. Standardized incidence ratios (SIRs) were used to assess risks relative to the general population. Poisson regression models were used to compare cancer incidence before and after 1996 and assess the impact of availability of HAART.

Results: Risk was elevated for the 2 major AIDS-defining cancers: Kaposi sarcoma (SIRs, 5321 and 1347 in years 3-5 and 6-10, respectively) and non-Hodgkin lymphoma (SIRs, 32 and 15). Incidence of both malignancies declined in the HAART era (1996-2006). Risk was elevated for all non–AIDS-defining cancers combined (SIRs, 1.7 and 1.6 in years 3-5 and 6-10, respectively) and for the following specific non–AIDS-defining cancers: Hodgkin lymphoma and cancers of the oral cavity and/or pharynx, tongue, anus, liver, larynx, lung and/or bronchus, and penis. Anal cancer incidence increased between 1980-1995 and 1996-2006 (RR, 2.9; 95% confidence interval [CI], 2.1-4.0), as did that of Hodgkin lymphoma (RR, 2.0; 95% CI, 1.3-2.9).

Conclusion: Among people who survived for several years after AIDS diagnosis, we observed large risks of AIDS-defining cancers and increasing incidence of anal cancer and Hodgkin lymphoma.

Arch Intern Med. 2010;170(15):1337-1345

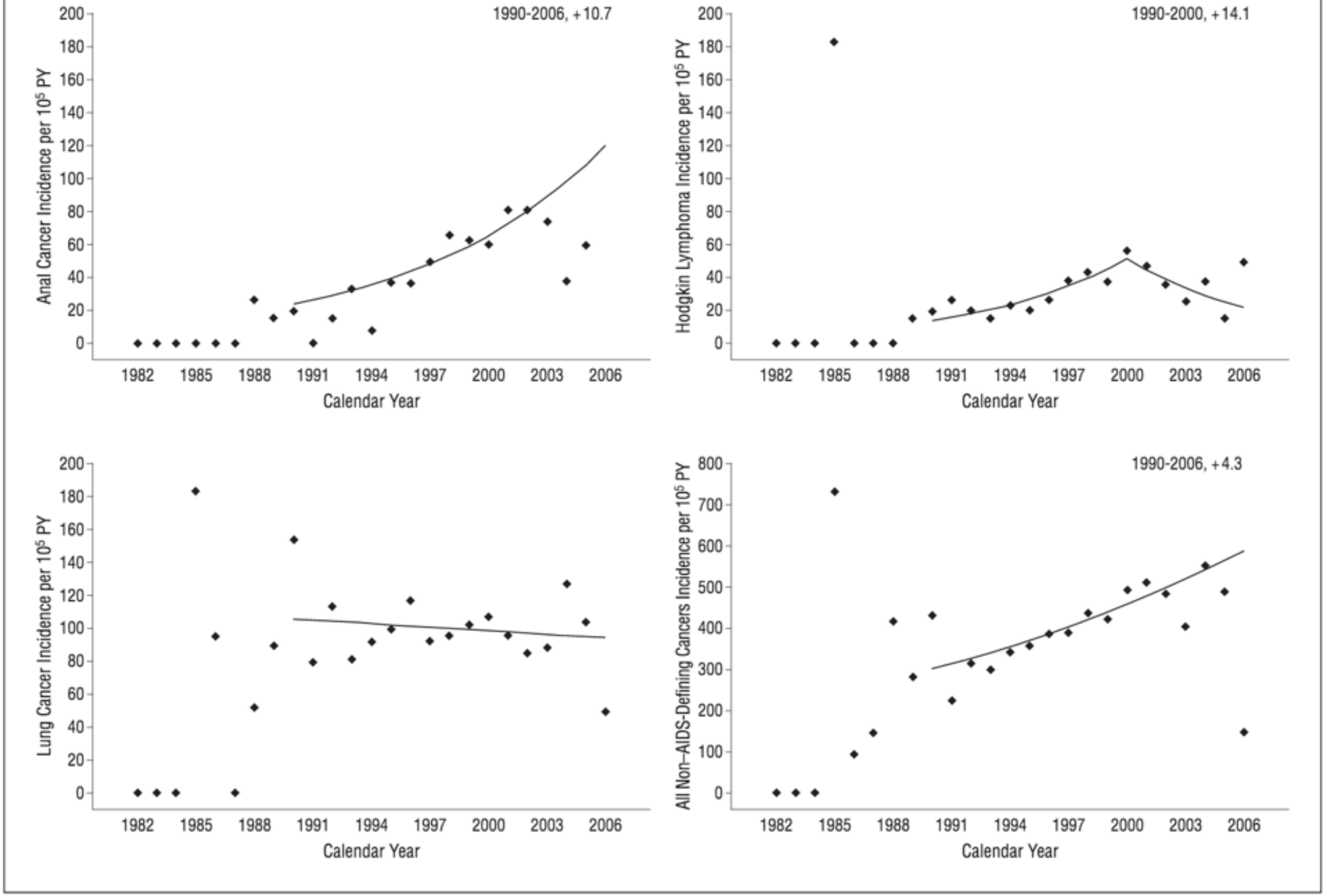


Figure 2. Incidence of selected non–AIDS-defining malignancies as a function of calendar year. The panels show cancer incidence during the period 3 to 10 years after AIDS onset as a function of attained calendar year. The points correspond to the individual year estimates, while the lines correspond to results from the joinpoint regression. Annual percentage change is indicated for calendar years where the change was significantly different from 0 ($P < .05$). PY indicates person-years.

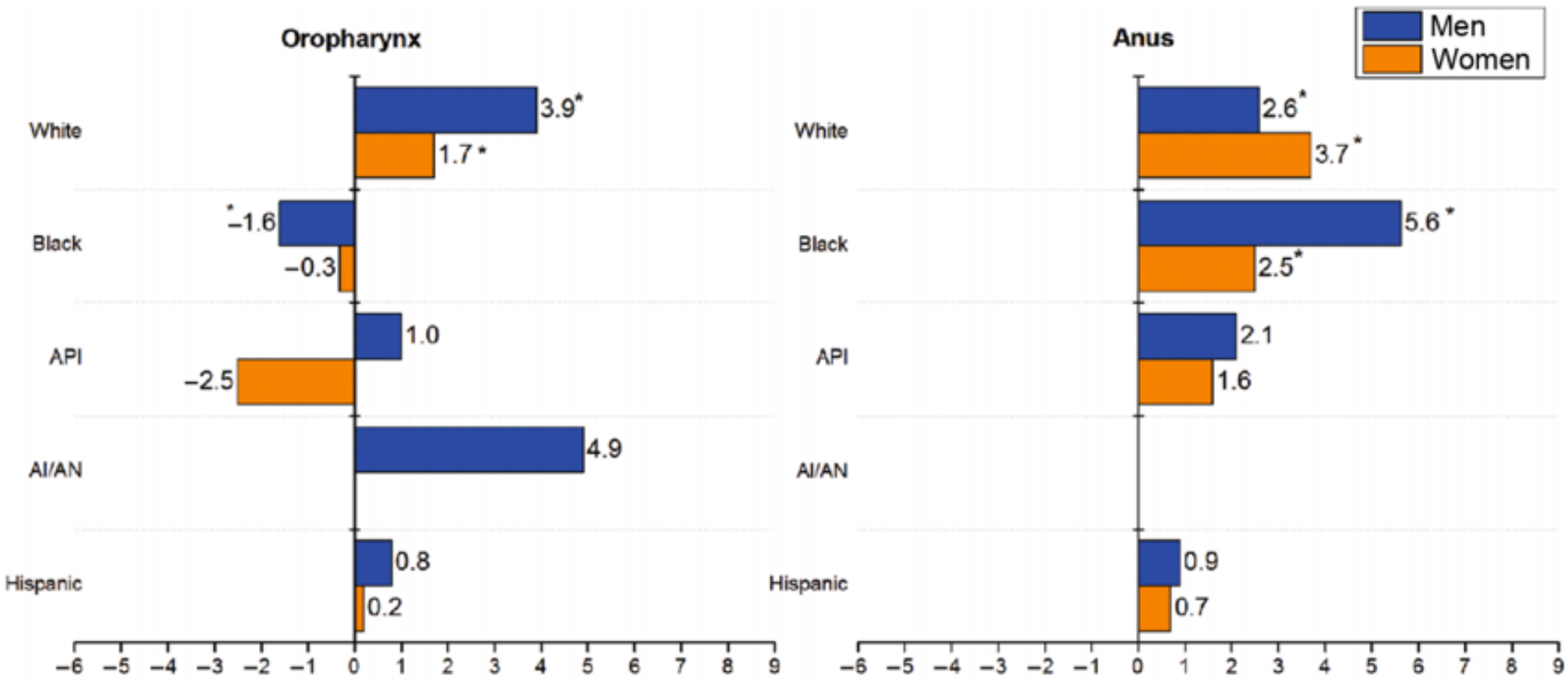
ARTICLE

Annual Report to the Nation on the Status of Cancer, 1975–2009, Featuring the Burden and Trends in Human Papillomavirus (HPV)–Associated Cancers and HPV Vaccination Coverage Levels

Ahmedin Jemal, Edgar P. Simard, Christina Dorell, Anne-Michelle Noone, Lauri E. Markowitz, Betsy Kohler, Christie Eheman, Mona Saraiya, Priti Bandi, Debbie Saslow, Kathleen A. Cronin, Meg Watson, Mark Schiffman, S. Jane Henley, Maria J. Schymura, Robert N. Anderson, David Yankey, Brenda K. Edwards

Manuscript received August 15, 2012; revised October 18, 2012; accepted October 19, 2012.

Correspondence to: *Ahmedin Jemal, DVM, PhD, Surveillance Research Program, American Cancer Society, 250 Williams St NW, Atlanta, GA 30303* (e-mail: ajemal@cancer.org).


Oropharynx
White
3.9*
1.7*
Black
*−1.6
−0.3
API
1.0
−2.5
AI/AN
4.9
Hispanic
0.8
0.2
−6 −5 −4 −3 −2 −1 0 1 2 3 4 5 6 7 8 9
Anus
Men
Women
White
2.6*
3.7*
Black
5.6*
2.5*
API
2.1
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Hispanic
0.9
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Centers for Disease Control and Prevention
MMWR
Morbidity and Mortality Weekly Report
Recommendations and Reports / Vol. 63 / No. 5 August 29, 2014 Re

Human Papillomavirus Vaccination

Recommendations of the Advisory Committee on Immunization Practices (ACIP)

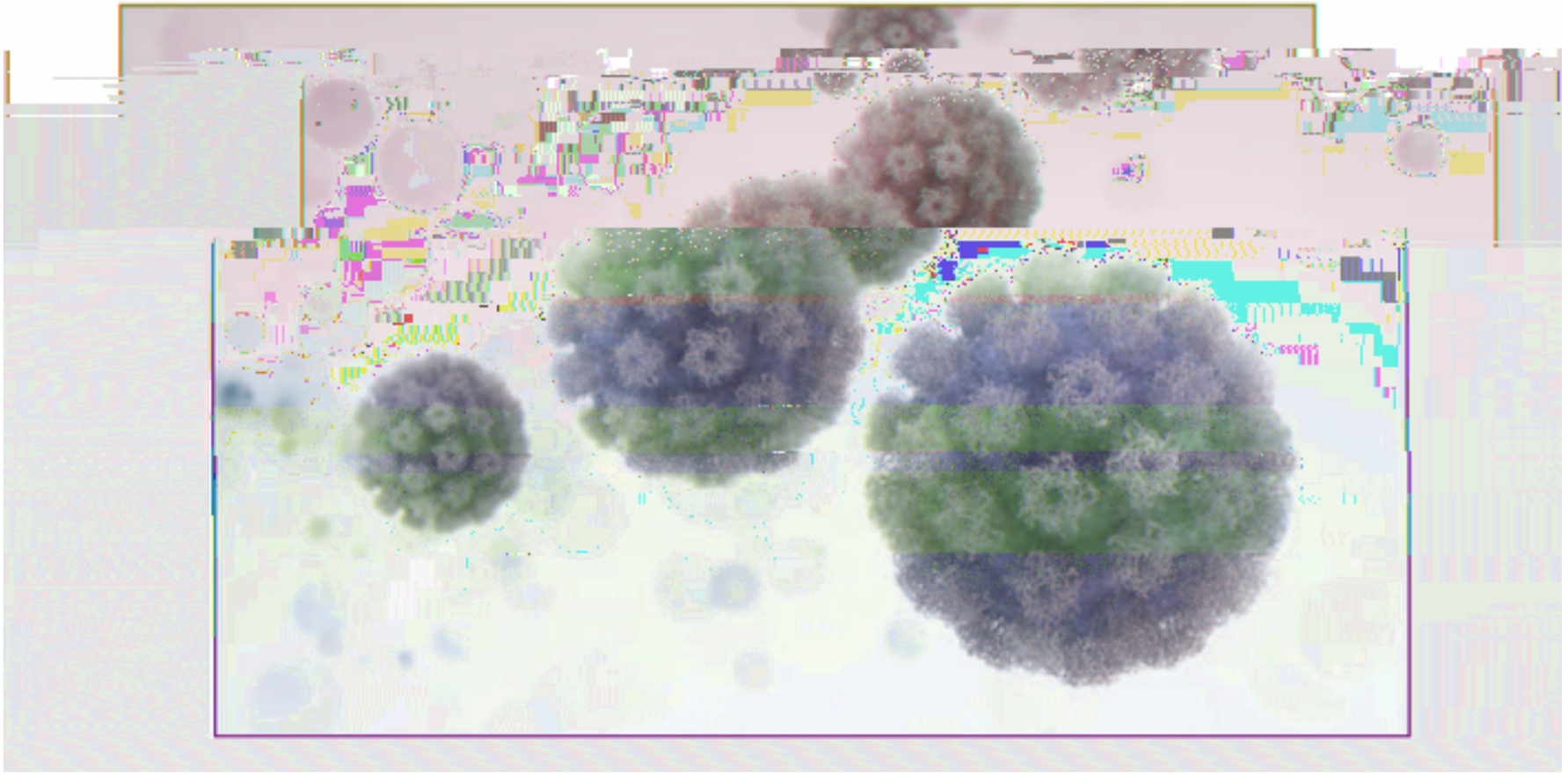


TABLE 1. Human papillomavirus vaccines licensed in the United States and ACIP recommendations for vaccination, 2006–2014

Characteristic	Quadrivalent HPV vaccine (HPV4)	Bivalent HPV vaccine (HPV2)
Manufacturer	Merck and Co, Inc.	GlaxoSmithKline
HPV types	HPV 6, 11, 16, 18	HPV 16, 18
Year of licensure (age range)	Females: 2006 (9–26 years)	Females: 2009 (9–25 years)
	Males: 2009 (9–26 years)	Not licensed for use in males
ACIP recommendations, 2006*	Females: routine vaccination with 3-dose series at age 11 or 12 years[†,§] and through age 26 years if not vaccinated previously	
ACIP recommendations, 2009[¶]	Females: either vaccine for routine vaccination with 3-dose series at age 11 or 12 years[†,§] and through age 26 years if not vaccinated previously	
	Males aged 9–26 years may be vaccinated, but vaccination not routinely recommended for males	
ACIP recommendations, 2011**	Females: either vaccine for routine vaccination with 3-dose series at age 11 or 12 years[†,§] and through age 26 years if not vaccinated previously	
	Males: routine vaccination with 3-dose series at age 11 or 12 years[†,§] and through age 21 years if not vaccinated previously[††] Vaccination recommended through age 26 years for men who have sex with men and men who are immunocompromised (including those with HIV infection)	

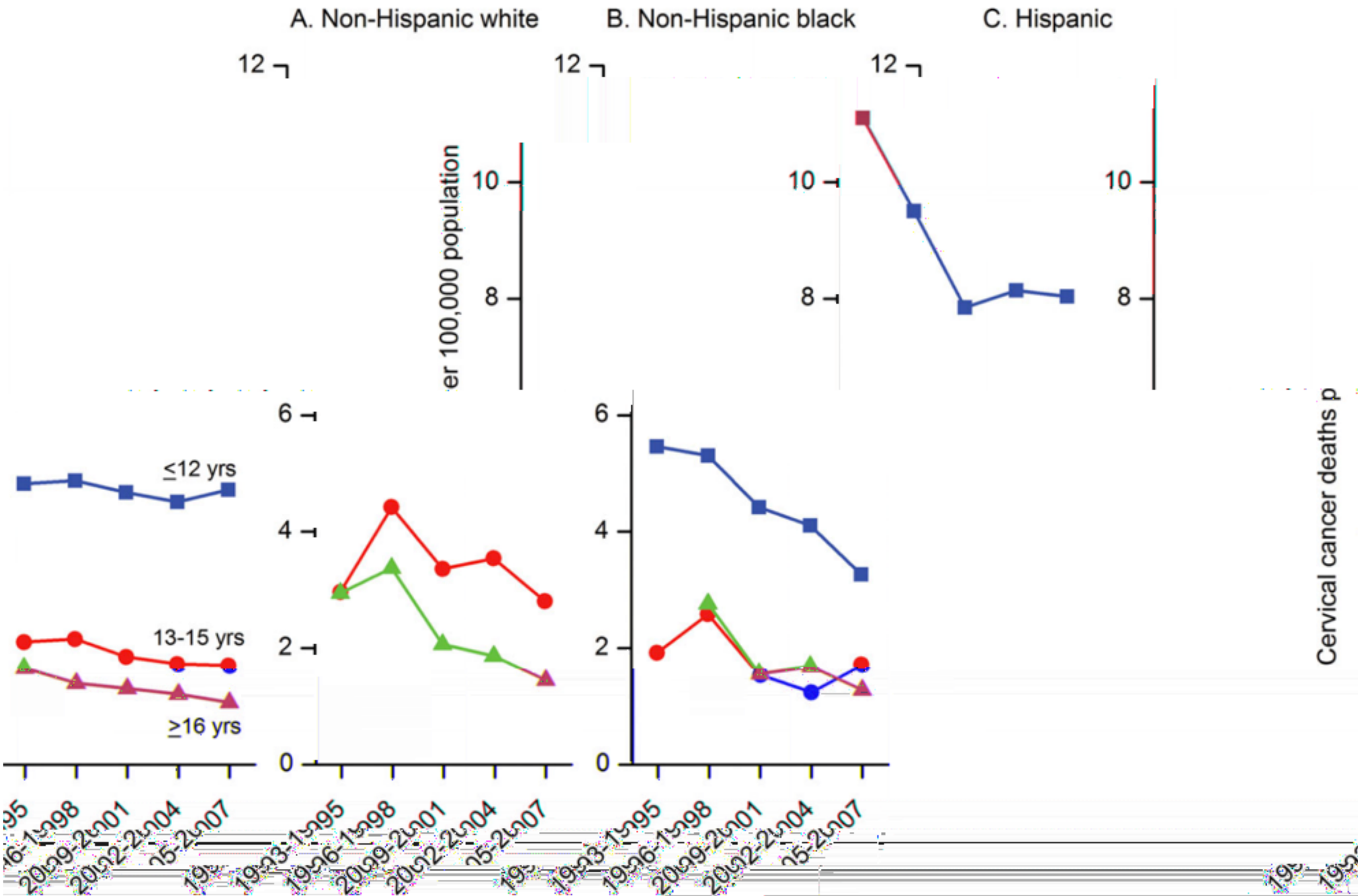
Original Article

Widening Socioeconomic Disparities in Cervical Cancer Mortality Among Women in 26 States, 1993-2007

Edgar P. Simard, PhD, MPH[1]; Stacey Fedewa, MPH[2]; Jiemen Ma, PhD, MHS[1]; Rebecca Siegel, MPH[1]; and Ahmedin Jemal, DVM, PhD[1]

BACKGROUND: Despite substantial declines in cervical cancer mortality because of widespread screening, socioeconomic status (SES) disparities persist. The authors examined trends in cervical cancer mortality rates and the risk of late-stage diagnoses by SES.

METHODS: Using data from the National Vital Statistics System, trends in age-standardized mortality rates among women ages 25 to 64 years (1993-2007) by education level (<12 years, 13-15 years, and ≥16 years) and race/ethnicity for non-Hispanic white (NHW)



are illustrated in age-adjusted cervical cancer death rates among women ages 25 to 64 years in
nicity and educational attainment (1993-2007).

Figure 1. (A-C) Temporal tren
26 states according to race/e

ONLINE FIRST

The Influence of Sex, Race/Ethnicity, and Educational Attainment on Human Immunodeficiency Virus Death Rates Among Adults, 1993-2007

Edgar P. Simard, PhD, MPH; Maglin Fransua, MD; Deepa Naishadham, MA, MS; Ahmedin Jemal, DVM, PhD

Background: Overall declines in human immunodeficiency virus (HIV) mortality may mask patterns for subgroups, and prior studies of disparities in mortality have used area-level vs individual-level socioeconomic measures. The aim of this study was to examine temporal trends in HIV mortality by race/ethnicity and level of education (as a proxy for socioeconomic status).

Methods: We examined HIV deaths among non-Hispanic white, non-Hispanic black, and Hispanic men and women aged 25 to 64 years in 26 states (1993-2007; N=913 [illegible]) reported to the National Vital Statistics System. The main outcome measures were age-standardized death rates, rate differences, and rate ratios by educational attainment and between the least- and the most-educated (≤12 vs ≥16 years) individuals.

Results: Between 1993-1995 and 2005-2007, mortality declined for most men and women by race/ethnicity and educational levels, with the greatest absolute decreases among whites owing to their higher baseline rates. Among men with the most education, rates per 100 000 population decreased from 117.89 (95% CI, 101.08-134.70) to 15.35 (12.08-18.62) in blacks vs from 26.42 (24.93-27.92) to 1.79 (1.50-2.08) in whites. Rates were unchanged for the least-educated black women (26.76; 95% CI, 24.30-29.23; during 2005-2007) and remained high [illegible]. Relative declines were greater with increasing levels of education ($P < .001$), resulting in widening disparities. Among men, the disparity rate ratio (comparing the least- and the most-educated) increased from 1.04 (95% CI, 0.89-1.21) during 1993-1995 to 3.43 (2.74-4.30) during 2005-2007 for blacks and from 0.98 (0.91-1.05) to 2.82 (2.34-3.40) for whites.

Conclusions: Although absolute declines in HIV mortality were greatest for nonwhites, rates remain high among blacks, especially in the lowest educated groups, underscoring the need for additional interventions.

Arch Intern Med
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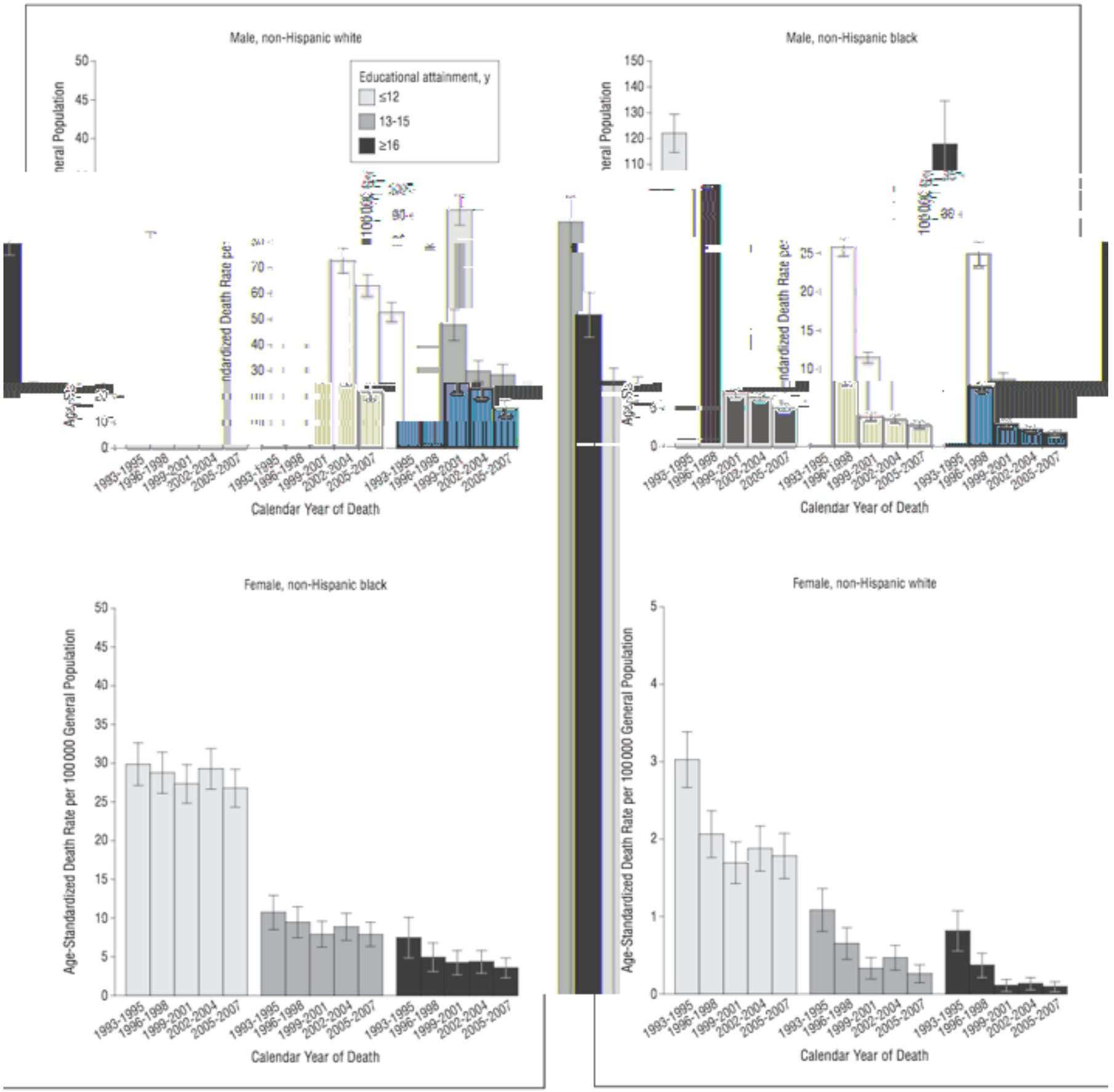


n-Hispanic whites and non-Hispanic blacks by sex, educational attainment, and range of the y-axis differs across groups. Rates are presented only for those

Figure 1. Trends in human immunodeficiency virus infection death rates for no calendar period in 26 states, 1993-2007. Error bars represent 95% CIs, and the individuals with a specified level of education recorded on death certificates.

Table 2. Changes in Age-Standardized HIV Infection Death Rates by Sex, Race/Ethnicity, and Educational Attainment Among Individuals Aged 25 to 64 Years in 26 States, 1993-2007

Race/Ethnicity, Educational Attainment	Death Rates (95% CI) per 100 000 General Population[a]		Absolute and Relative Changes in Death Rates	
	1993-1995	2005-2007	RD (95% CI)[b]	RR (95% CI)[c]
Male				
Non-Hispanic white, y				
All education	26.54 (25.75 to 27.34)	3.64 (3.40 to 3.88)	22.91 (22.08 to 23.74)	0.14 (0.13 to 0.15)
≤12	25.77 (24.62 to 26.92)	5.04 (4.59 to 5.50)	20.72 (19.49 to 21.96)	0.20 (0.18 to 0.22)
13-15	24.93 (23.39 to 26.46)	2.82 (2.39 to 3.25)	22.10 (20.51 to 23.70)	0.11 (0.10 to 0.13)
≥16	26.42 (24.94 to 27.90)	1.79 (1.50 to 2.08)	24.63 (23.11 to 26.16)	0.07 (0.06 to 0.08)[d]
RD (95% CI) for ≤12 vs ≥16 y	−0.65 (−2.54 to 1.23)	3.26 (2.72 to 3.79)		
RR (95% CI) for ≤12 vs ≥16 y	0.98 (0.91 to 1.05)	2.82 (2.34 to 3.40)		
Non-Hispanic black, y				
All education	119.65 (113.88 to 125.42)	40.64 (38.61 to 42.68)	79.00 (72.89 to 85.12)	0.34 (0.32 to 0.36)
≤12	122.02 (114.67 to 129.38)	44.71 (41.96 to 47.45)	77.31 (69.95 to 84.67)	0.37 (0.34 to 0.40)
13-15	97.53 (77.92 to 97.34)	21.81 (13.56 to 25.16)	55.72 (55.45 to 75.96)	0.28 (0.21 to 0.36)
≥16	117.89 (101.03 to 134.70)	15.35 (12.08 to 18.62)	102.54 (85.42 to 119.66)	0.13 (0.10 to 0.17)[d]
RD (95% CI) for ≤12 vs ≥16 y	4.13 (−14.21 to 22.45)	37.36 (32.39 to 42.33)		
RR (95% CI) for ≤12 vs ≥16 y	1.04 (0.89 to 1.21)	3.43 (2.74 to 4.30)		
Hispanic, y				
All education	58.67 (52.21 to 65.14)	8.09 (6.85 to 9.34)	50.58 (44.00 to 57.17)	0.14 (0.11 to 0.17)
≤12	61.60 (53.46 to 69.75)	9.01 (7.36 to 10.66)	52.59 (44.28 to 60.91)	0.15 (0.12 to 0.18)
13-15	40.09 (28.43 to 51.75)	4.96 (2.56 to 7.36)	35.13 (23.22 to 47.03)	0.12 (0.07 to 0.22)
≥16	49.84 (33.26 to 66.42)	3.13 (0.91 to 5.35)	46.71 (29.98 to 63.44)	0.06 (0.03 to 0.14)[d]
RD (95% CI) for ≤12 vs ≥16 y	11.76 (−6.71 to 30.24)	5.88 (3.11 to 8.65)		
RR (95% CI) for ≤12 vs ≥16 y	1.24 (0.86 to 1.77)	2.88 (1.38 to 5.99)		

How do we move forward?

- Preventive healthcare
    - Primary prevention
    - Secondary prevention
- Access to care and insurance
    - Affordable care act
- Health equality vs. Health equity
- Increase quality of care
- Critically evaluate old and new interventions for cost effectiveness
- Technology

Beyond
Traditional Research and PubMed

- Data analytics (Twitter feeds, Google Searches)
- Real-time disease reporting
- At-home diagnostics
  - 23&ME
  - Theranos
- Activity trackers

theranos

fitbit.

Opportunities and Challenges

- Consumer products (lifestyle) vs. medical devices (diagnostic + treatment)

- Leveraging big data for meaningful insights

- Accuracy of products, predictions

- Gaining uptake and acceptance in a crowded space (value proposition)

- Dissemination to the people who need it most (public health)

Bench to Bedside: Translational Research & mHealth

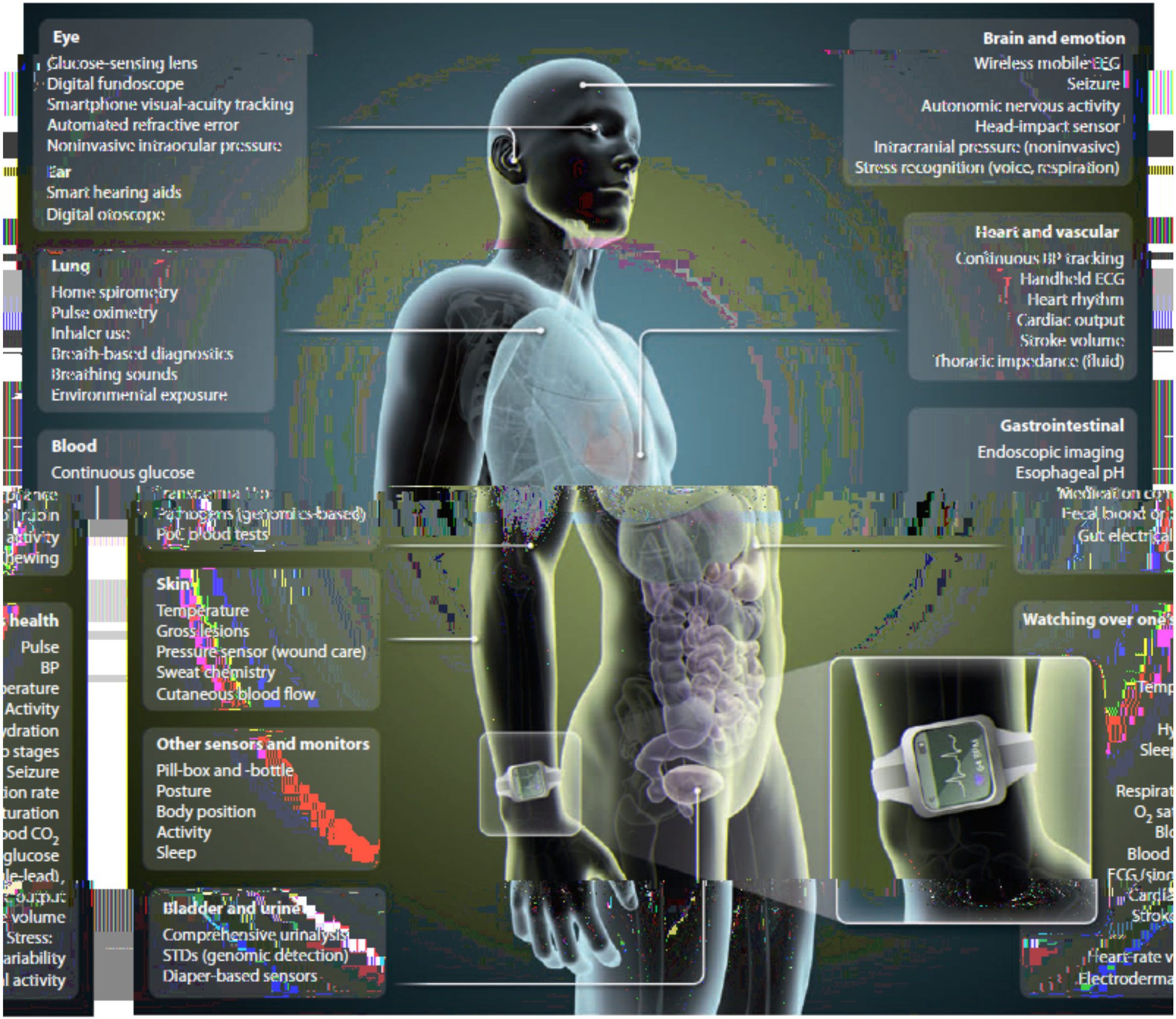
REVIEW

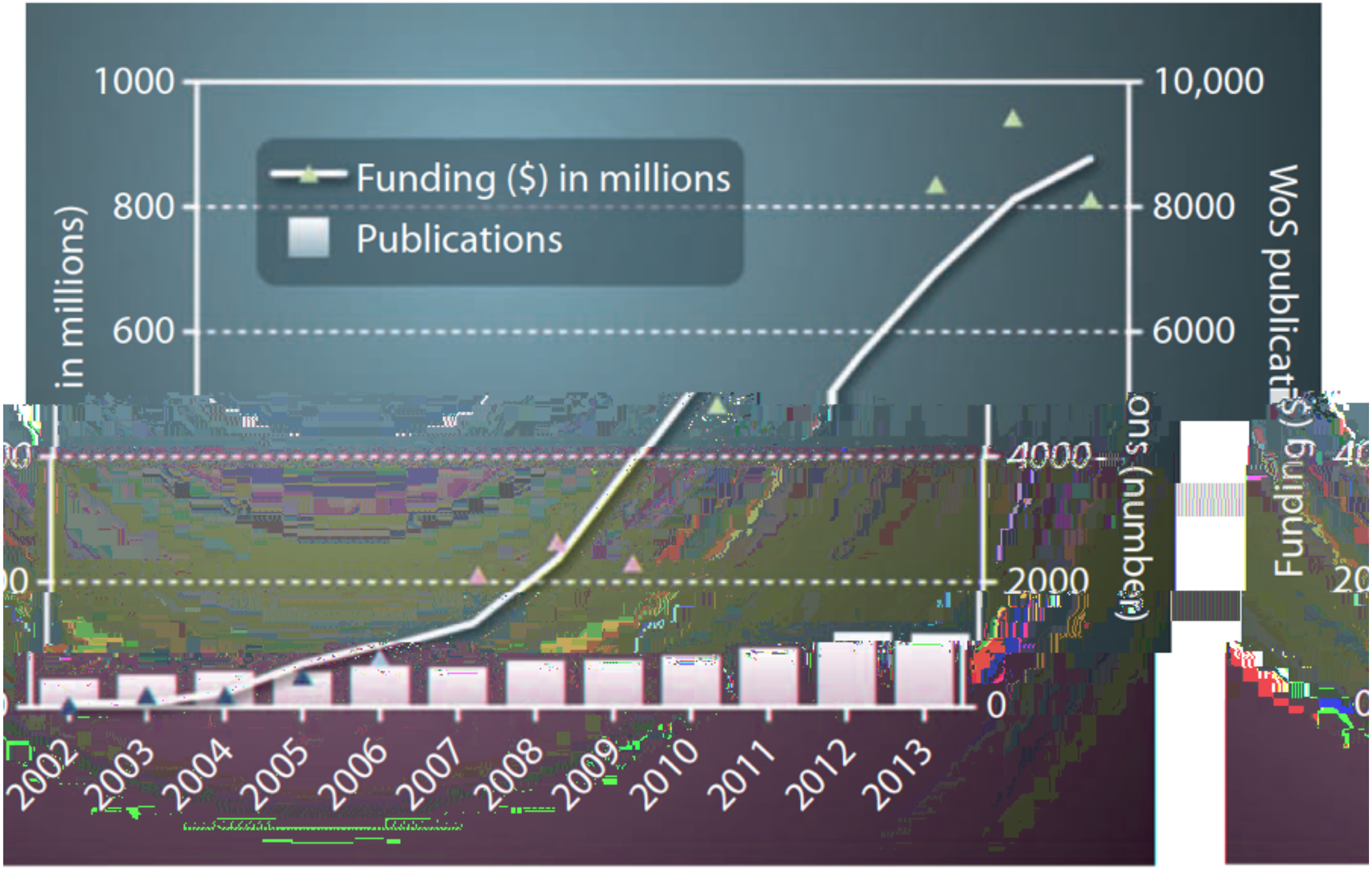
HEALTH CARE

The emerging field of mobile health

Steven R. Steinhubl,* Evan D. Muse, Eric J. Topol

The surge in computing power and mobile connectivity have fashioned a foundation for mobile health (mHealth) technologies that can transform the mode and quality of clinical research and health care on a global scale. Unimpeded by geographical boundaries, smartphone-linked wearable sensors, point-of-need diagnostic devices, and medical-grade imaging, all built around real-time data streams and supported by automated clinical decision support tools, will enable care and enhance our understanding of physiological variability. However, the path to mHealth incorporation into clinical care is fraught with challenges. We currently lack high-quality evidence that supports the adoption of many new technologies and have financial, regulatory, and security hurdles to overcome. Fortunately, sweeping efforts are under way to establish the true capabilities and value of the evolving mHealth field.


Eye
Glucose-sensing lens
Digital fundoscope
Smartphone visual-acuity tracking
Automated refractive error
Noninvasive intraocular pressure
Ear
Smart hearing aids
Digital otoscope
Lung
Home spirometry
Pulse oximetry
Inhaler use
Breath-based diagnostics
Breathing sounds
Environmental exposure
Blood
Continuous glucose
Pathogens (genomics-based)
PoC blood tests
Skin
Temperature
Gross lesions
Pressure sensor (wound care)
Sweat chemistry
Cutaneous blood flow
Other sensors and monitors
Pill-box and -bottle
Posture
Body position
Activity
Sleep
Bladder and urine
Comprehensive urinalysis
STDs (genomic detection)
Diaper-based sensors
Brain and emotion
Wireless mobile EEG
Seizure
Autonomic nervous activity
Head-impact sensor
Intracranial pressure (noninvasive)
Stress recognition (voice, respiration)
Heart and vascular
Continuous BP tracking
Handheld ECG
Heart rhythm
Cardiac output
Stroke volume
Thoracic impedance (fluid)
Gastrointestinal
Endoscopic imaging
Esophageal pH
Watching over one's



n Health is taking center stage. Measures are funding and number of related publications. Fig. 2. H
re the annual total funding for patient-facing mHealth companies and the annual num- Shown a
lated publications [identified with Web of Science (WoS) using search terms "telemedi- ber of re
d "mhealth*" and "digital health" and "digital medicine"]. Funding data provided by cine" and
and A. Pai of MobiHealthNews. B. Dolan

mHealth: Challenging next steps

- Expanding the evidence base
- Financial obstacles and public + private partnerships
- Privacy and security concerns
- Avoiding data overload, “worried well”
- Staying patient-centered and outcome-focused

BIG DATA

Google Flu Trends: The Limits of Big Data

By STEVE LOHR MARCH 28, 2014 7:00 AM 14 Comments

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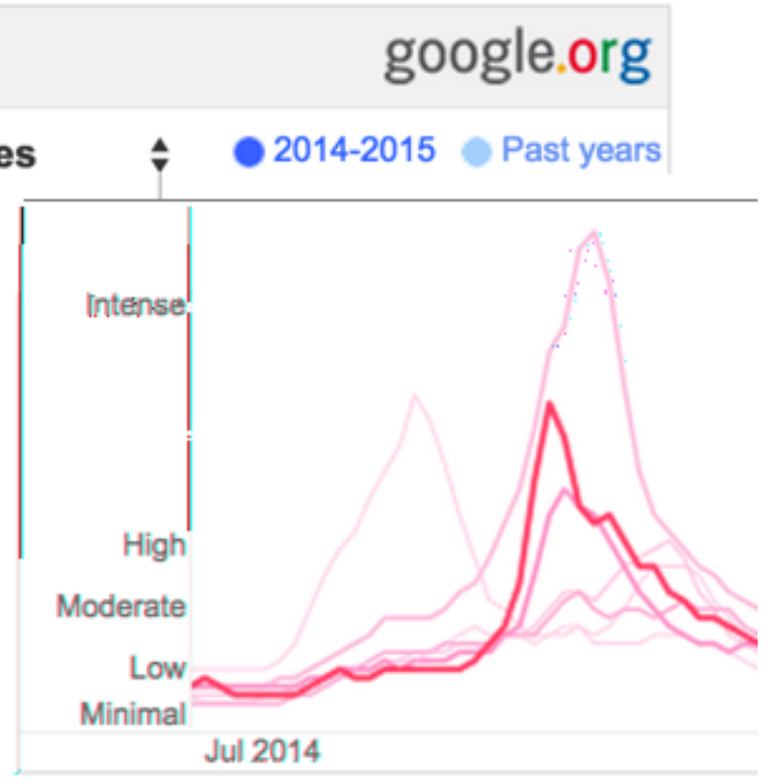

Google Flu Trends, once a poster child for the power of big-data analysis, seems to be under attack.

This month, in a Science magazine article, four quantitatively adept social scientists reported that Google's flu-tracking service not only wildly overestimated the number of flu cases in the United States in the 2012-13 flu season — a well-known mi has also consistently overshot in the last four years. Google Trends' estimate for the 2011-12 flu season was m percent higher than the cases reported by the Cen Control and Prevention. And, they wrote, for a pe two years ending in September 2013, the Google high in 100 out of 108 weeks.

ss — but

Flu

ore than 50

nters for Disease

riod of more than

estimates were

Questions?

Thank you!

epsimard@gmail.com
@asymptoticstat