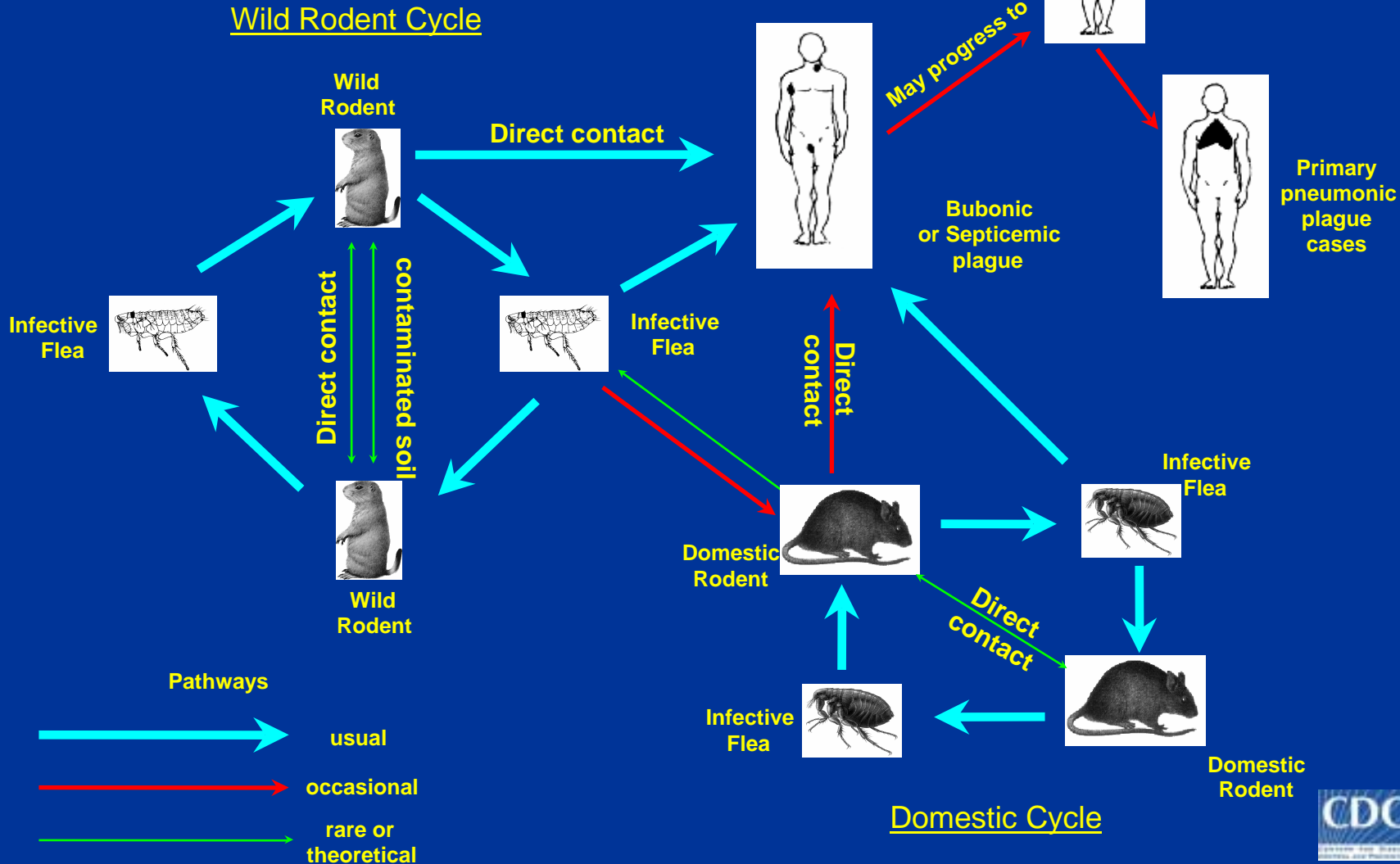


Plague – A National Update

Ken Gage

Bacterial Zoonoses Branch

DVBID/CDC



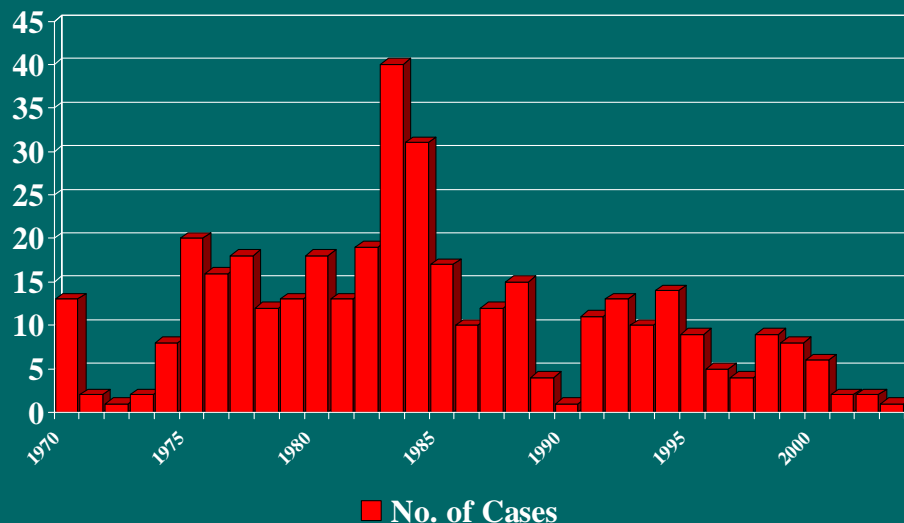
Is Plague a Submerging Infectious Disease?

- 3 human cases in last two years (All exposed in New Mexico)
- 2 NM cases diagnosed in New York City; high profile
- What explains the drop in cases? Control/prevention efforts?
- Very limited epizootic activity (Arizona, California, Colorado, and New Mexico all have low activity – sites of 90% of cases)
- Okay, but what caused the drop in epizootic activity?
- What does the future hold?
- Collaborative research over past decade suggests some likely answers to the above questions – Climatic factors?



Going, Going, Gone?

Do we need to find new journals for our papers?

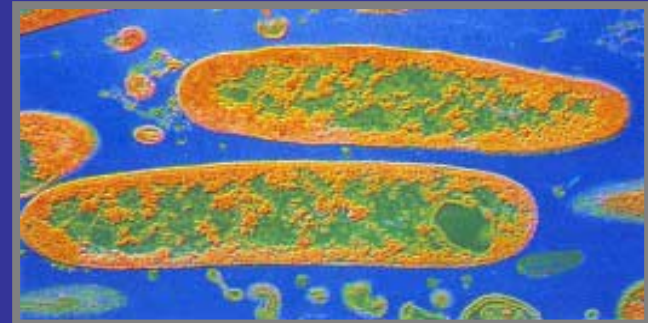


How Could Climate influence Plague Activity?

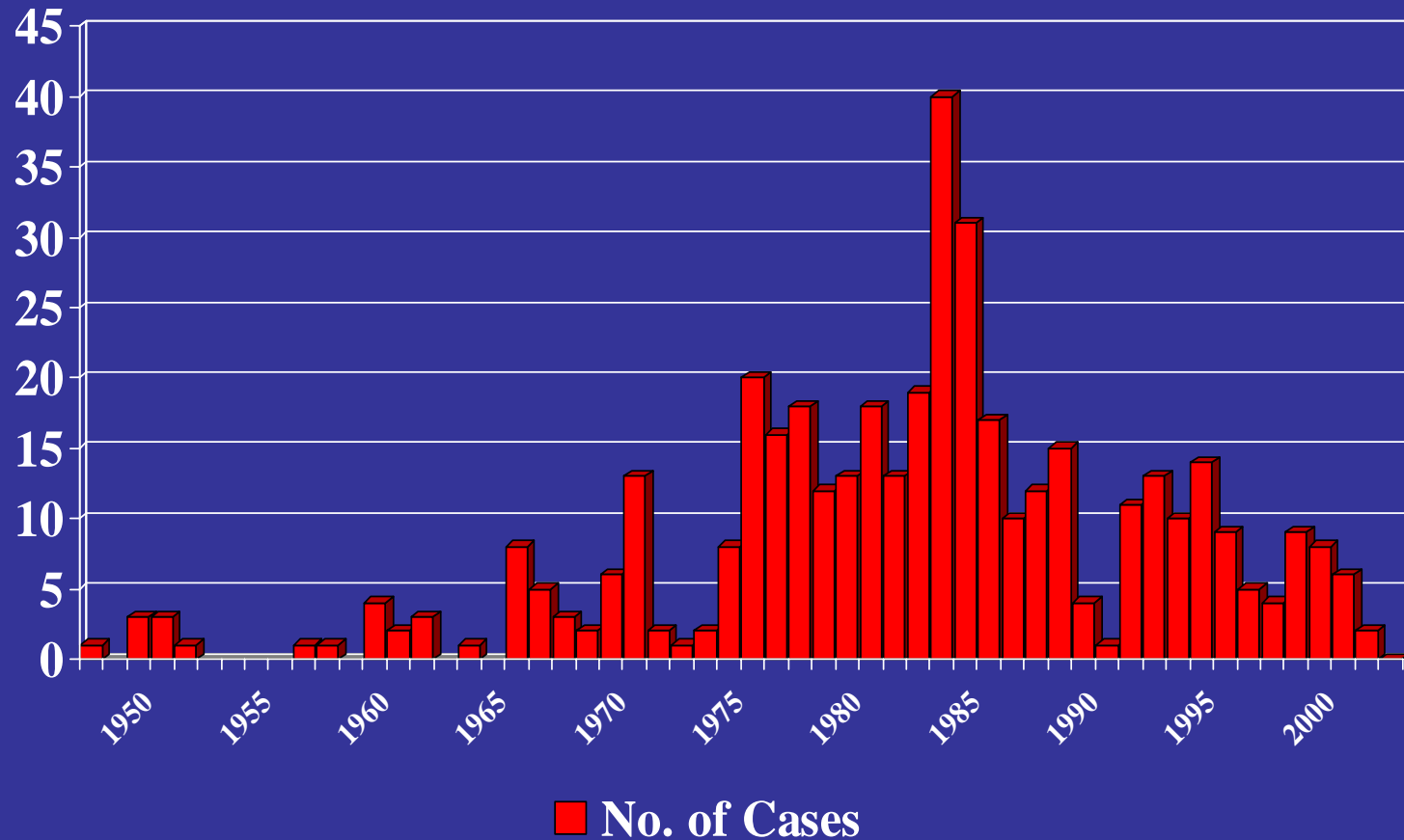
- **Seasonality of transmission**
- **Survival of fleas**
- **Ability of fleas to transmit and retain infection**
- **Rodent host and flea vector population dynamics**

Climate and Plague Transmission

- Major Pandemics (Justinian's Plague and the Black Death) were associated with major climatic fluctuations
- India – Temperature, humidity and rainfall effects (Greenwood 1911, Brooks 1915-1917, Rogers 1928)
- Vietnam – Decreased transmission at temperatures above 27° C (Cavanaugh and Marshall 1972)
- Southern Africa – Severe drought forces bush rodents into peridomestic environments (Isaacson 1983)
- Peru – Outbreaks after El Ninos
- Kazakhstan – Cases occurred 1-2 years after higher than normal rainfall years (Dubynsky et al. 1992)



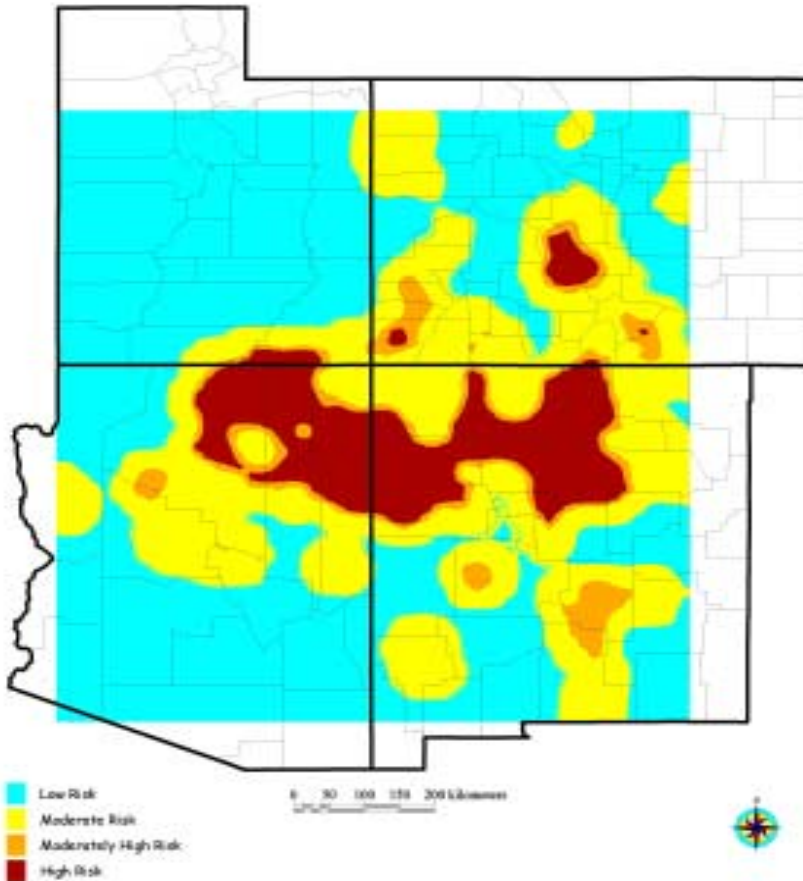
Reported Human Plague Cases By Year-U.S.A., 1947-2002



N = 420

Plague in the Southwest

Areas at risk for Plague



- Site of about 80% of U.S. cases
- High risk areas well-defined (pinon-juniper and nearby areas)
- Peridomestic exposures
- Rock squirrels, other ground squirrels, prairie dogs, wood rats, deer mice and their relatives
- Acquired via:
 - a. Flea bite (~ 80%)
 - b. Direct contact with animals (~ 20%)
 - c. Inhalation (rare – cats with pneumonic plague)

INCIDENCE OF PLAGUE ASSOCIATED WITH INCREASED WINTER-SPRING PRECIPITATION IN NEW MEXICO

ROBERT R. PARMENTER, EKTA PRATAP YADAV, CHERYL A. PARMENTER,
PAUL ETTESTAD, AND KENNETH L. GAGE

*Department of Biology, University of New Mexico, Albuquerque, New Mexico; Office of Epidemiology,
New Mexico State Department of Health, Santa Fe, New Mexico; Division of Vector-Borne Infectious Diseases,
National Center for Infectious Diseases, Center for Disease Control and Prevention, Fort Collins, Colorado*

Abstract. Plague occurs episodically in many parts of the world, and some outbreaks appear to be related to increased abundance of rodents and other mammals that serve as hosts for vector fleas. Climate dynamics may influence the abundance of both fleas and mammals, thereby having an indirect effect on human plague incidence. An understanding of the relationship between climate and plague could be useful in predicting periods of increased risk of plague transmission. In this study, we used correlation analyses of 215 human cases of plague in relation to

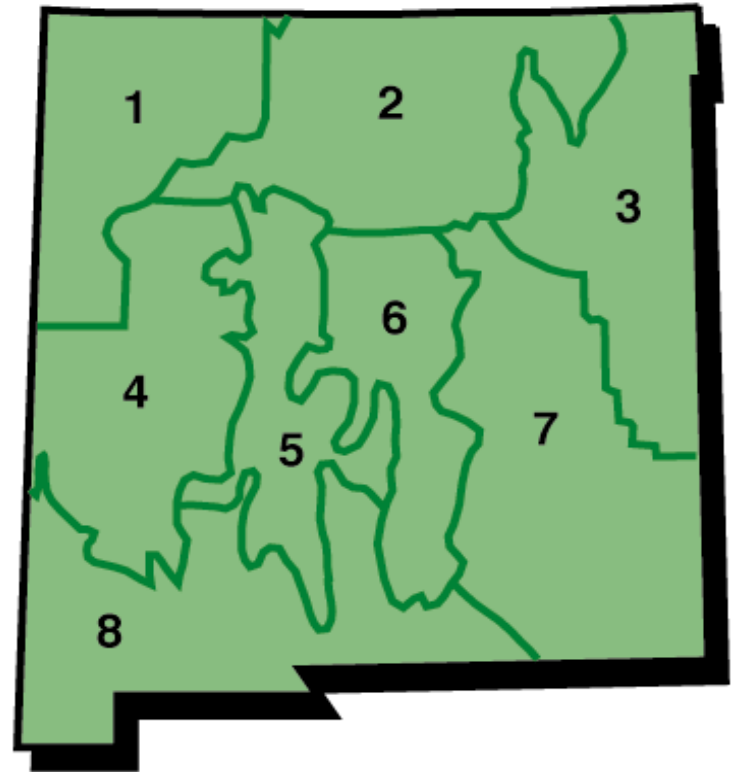
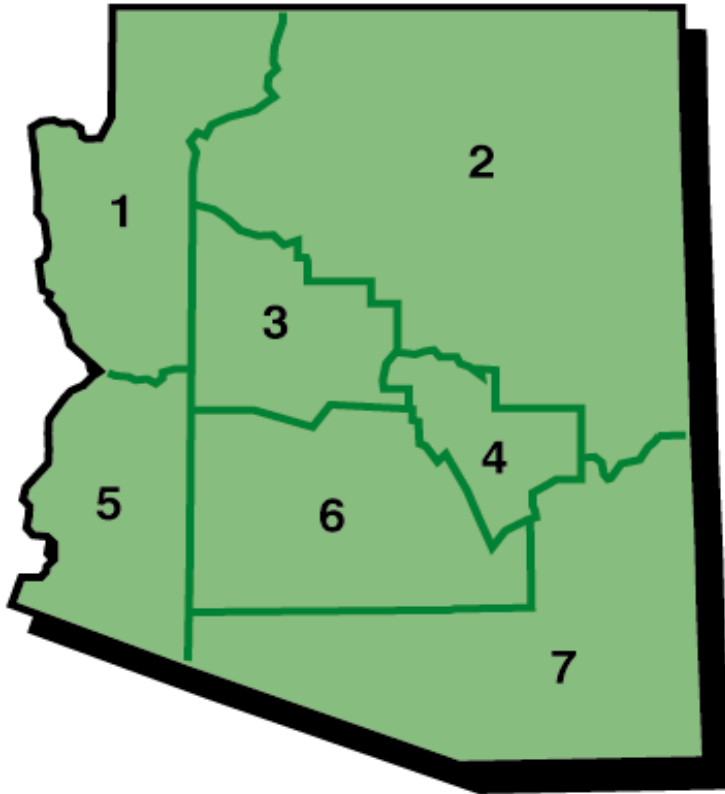
- Parmenter et al. (1999) – Positive correlation between winter-spring precipitation and occurrence of human cases (218 cases reported 1948-1996)
- Proposed trophic cascade model - Increased precipitation enhances small mammal food resource productivity, which leads to increases in host populations and plague risk.

MODELING RELATIONSHIPS BETWEEN CLIMATE AND THE FREQUENCY OF HUMAN PLAGUE CASES IN THE SOUTHWESTERN UNITED STATES, 1960–1997

RUSSELL E. ENSCORE, BRAD J. BIGGERSTAFF, TED L. BROWN, RALPH E. PULGHAM, PAMELA J. REYNOLDS, DAVID M. ENGELTHALER, CRAIG E. LEVY, ROBERT R. PARMENTER, JOHN A. MONTENIERI, JAMES E. CHEEK, RICHIE K. GRINNELL, PAUL J. ETTESTAD, AND KENNETH L. GAGE

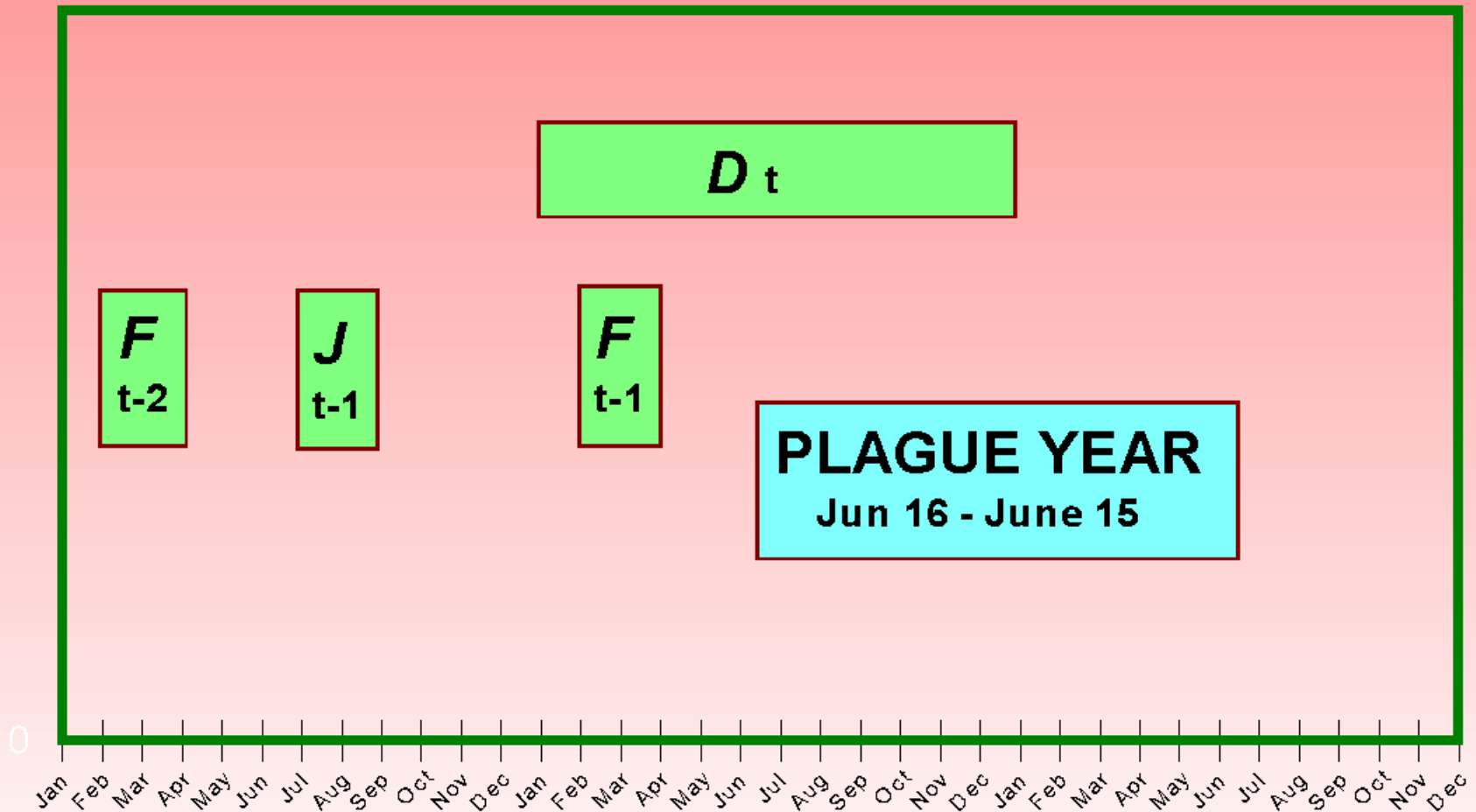
Division of Vector-Borne Infectious Diseases, National Center for Infectious Diseases, Centers for Disease Control and Prevention, Fort Collins, Colorado; Vector Control Program, New Mexico Environment Department, Santa Fe, New Mexico; Office of Environmental Health and Engineering, Navajo Area, Indian Health Service, Window Rock, Arizona; Vector-Borne and Zoonotic Disease Section, Arizona Department of Health Services, Phoenix, Arizona; Department of Biology, University of New Mexico, Albuquerque, New Mexico; Epidemiology Branch, Headquarters West, Indian Health Service, Albuquerque, New Mexico; Office of Environmental Health and Engineering, Albuquerque Area, Indian Health Service, Albuquerque, New Mexico; Office of Epidemiology, New Mexico Department of Health, Santa Fe, New Mexico

Abstract. The relationships between climatic variables and the frequency of human plague cases (1960–1997) were modeled by Poisson regression for two adjoining regions in northeastern Arizona and northwestern New Mexico. Model outputs closely agreed with the numbers of cases actually observed, suggesting that temporal variations in plague risk can be estimated by monitoring key climatic variables, most notably maximum daily summer temperature values and time-lagged (1 and 2 year) amounts of late winter (February–March) precipitation. Significant effects also were observed for time-lagged (1 year) summer precipitation in the Arizona model. Increased precipitation during specific periods resulted in increased numbers of expected cases in both regions, as did the number of days above certain lower thresholds for maximum daily summer temperatures (80°F in New Mexico and 85°F in Arizona). The number of days above certain high-threshold temperatures exerted a strongly negative influence on the numbers of expected cases in both the Arizona and New Mexico models (95°F and 90°F, respectively). The climatic variables found to be important in our models are those that would be expected to influence strongly the population dynamics of the rodent hosts and flea vectors of plague.



Palmer Regions for Arizona and New Mexico

Time Line for variables vs plague year



Arizona Model (Region 2)

$$\ln \lambda_t = \mu + \beta_1 (F_{t-1}) + \beta_2 (F_{t-2}) + \theta_1 (J_{t-1}) + \delta_{T1} (D_{t90}) + \delta_{T2} (D_{t95})$$

$\mu, \beta_1, \beta_2, \theta_1, \delta_{T1}, \delta_{T2}$: Estimated parameters

F_{t-1} and F_{t-2} : Feb-March precipitation

J_{t-1} : July-Aug precipitation (monsoon)

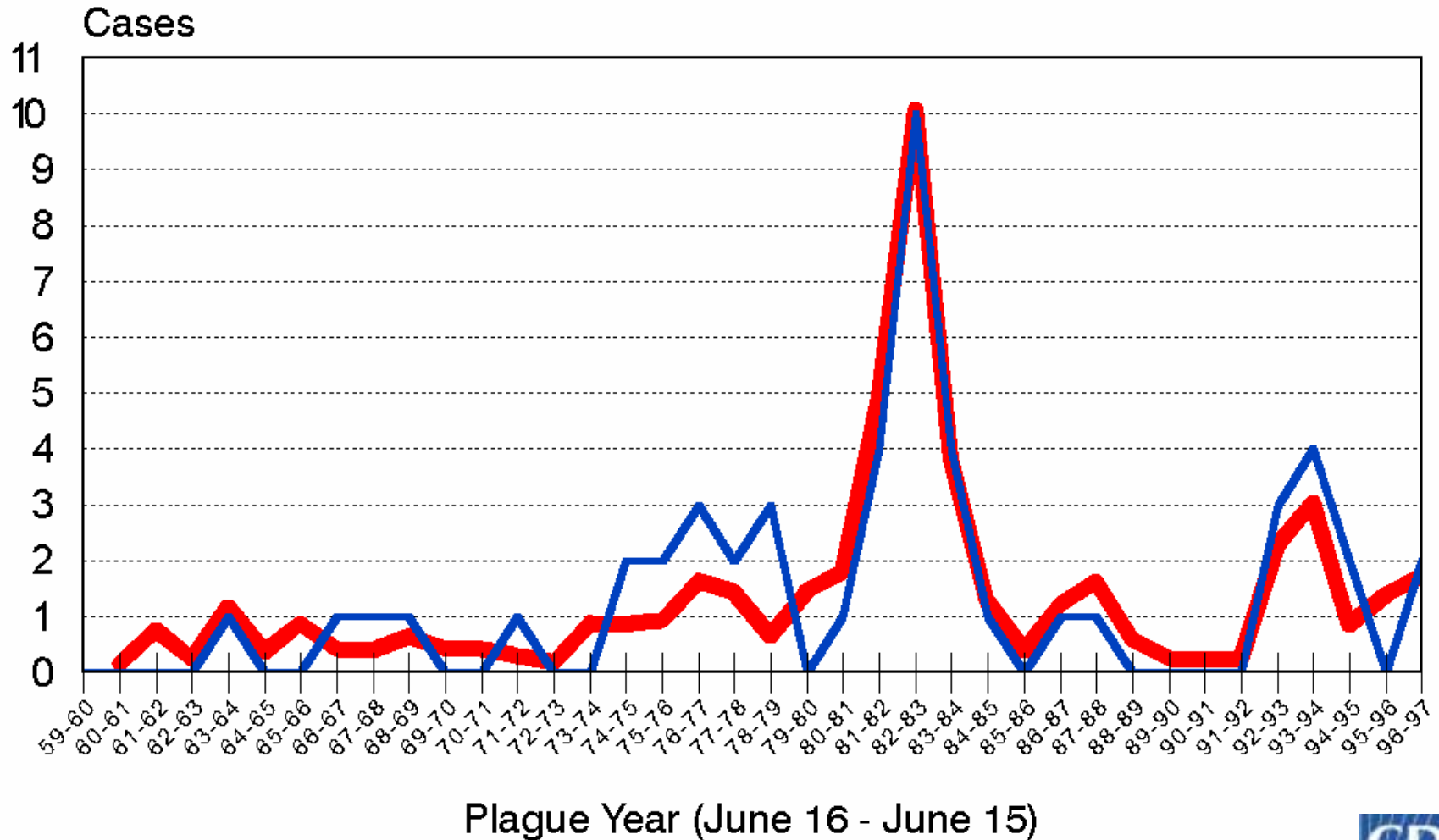
D_{t90} : Days above 90° F

D_{t95} : Days above 95° F

(Ensore et al. Am. J. Trop. Med. Hyg. 66:186-196, 2002)

Arizona Region 2

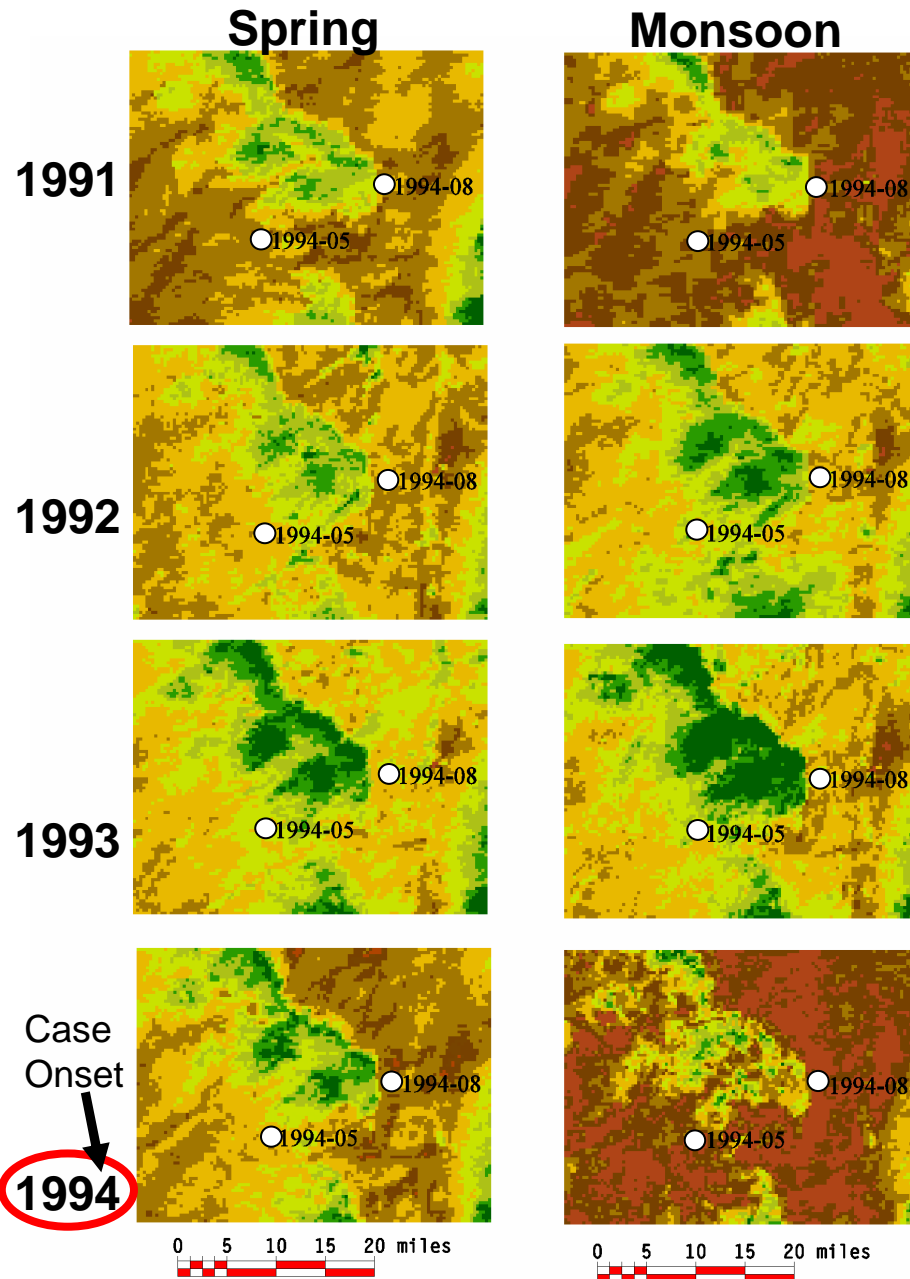
Observed vs Modeled

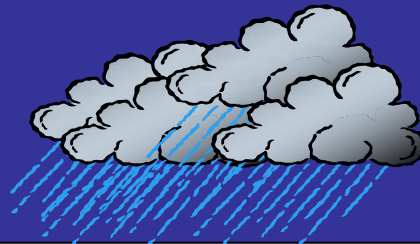


**NDVI Changes
Around Two Arizona
Case Sites
(Onset May 1994)**

Note:

- “Green” peak in Spring 1993 (1993 hantavirus outbreak?)
- Prolonged “greenness” in summer 1992 and 1993
- Prolonged favorable conditions for rodents and fleas
- “Brown-down” in summer 1994
- Hot, dry conditions bring epizootics to a halt (summer 1994)





Increased rodent food sources



Effects of Increased Precipitation
Feb. – March
(Major effect)
↓
July – Aug
(Minor effect)
↓
Feb. – March
(Minor effect)

Increased soil moisture and available hosts

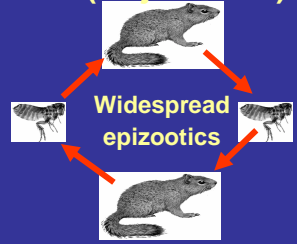


Increased rodent survival and reproduction

Increased flea survival and reproduction

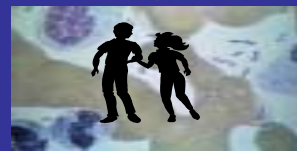
Cool summer
(15 – 18 months after first wet winter)

(Major effect)



High rodent densities favor epizootic spread

Cool temperatures favor survival of infected fleas



Increased human plague risks

Conclusions

- Small number of human cases and lack of widespread epizootic activity probably related to climatic factors (prolonged hot, dry conditions in the Southwest)
- Annual and seasonal variations in precipitation and threshold temperatures influence rodent and flea population dynamics
- High rodent and flea densities will increase the likelihood of epizootics
- Case numbers can be expected to increase following
 - Increased rainfall during critical periods (periods might vary from region to region)
 - Moderate to cool summers
- Human factors also greatly influence risk (about 80 percent of cases are peridomestic)

