

Chronic Obstructive Pulmonary Disease (ICD-9 Codes 490-492, 494, 496)

Chronic obstructive pulmonary disease (COPD) includes chronic bronchitis (ICD-9 codes 490-491), emphysema (ICD-9 code 492), bronchiectasis (ICD-9 code 494), and chronic airway obstruction (ICD-9 code 496). These diseases are commonly characterized by irreversible airflow limitation. Some authorities include asthma (ICD-9 code 493) and hypersensitivity pneumonitis (ICD-9 code 495) in the COPD rubric, but these infrequent causes of death (see Table 1) are categorized separately in this atlas.

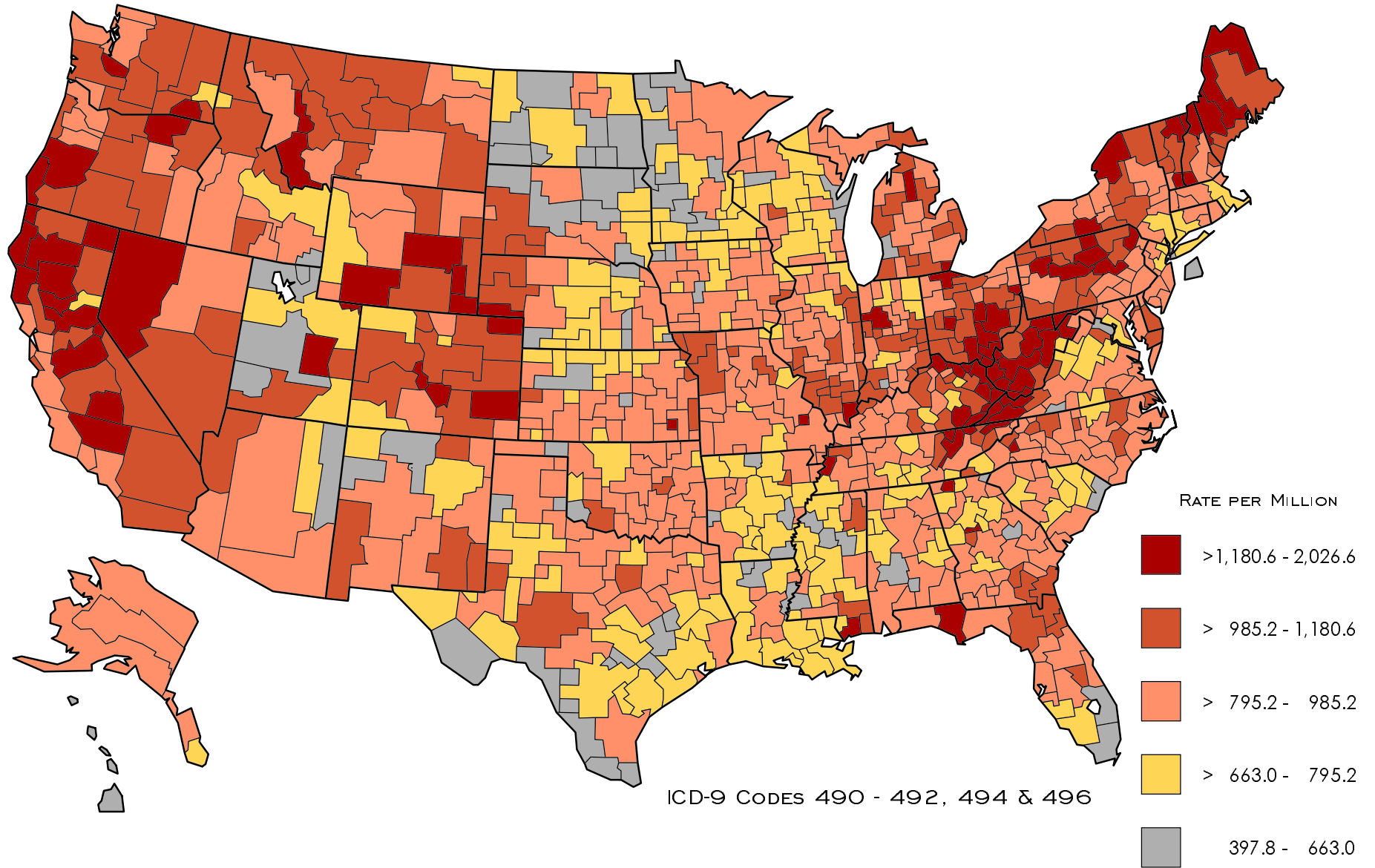
COPD has become a major public health concern. In 1995, it ranked as the fourth leading cause of death in the United States, accounting for over 100,000 deaths--nearly twice as many as in 1980 [NCHS 1997]. In 1994, an estimated 16 million individuals in the United States had the diagnosis of COPD, representing a 60 percent increase since 1982 [ALA 1996].

Although 80 to 90 percent of COPD cases are attributable to cigarette smoking [USDHHS 1984], the etiology of COPD is multifactorial. Additional risk factors include occupational exposures, air pollution, respiratory infection, and genetic factors. The relationship of COPD to workplace exposure is well documented for several occupational agents (e.g., coal mine dust, cotton dust, grain dust, etc.). More generally, estimates of the proportion of COPD attributable to occupational exposure based on community studies conducted in the United States range as high as 14 percent and 28 percent [Becklake 1994]. Investigations of the association of COPD with exposure to particulates in the general environment suggest that nonoccupational exposure to airborne particulate is also an important cause of preventable morbidity and mortality [Dockery et al. 1993; Sunyer et al. 1993].

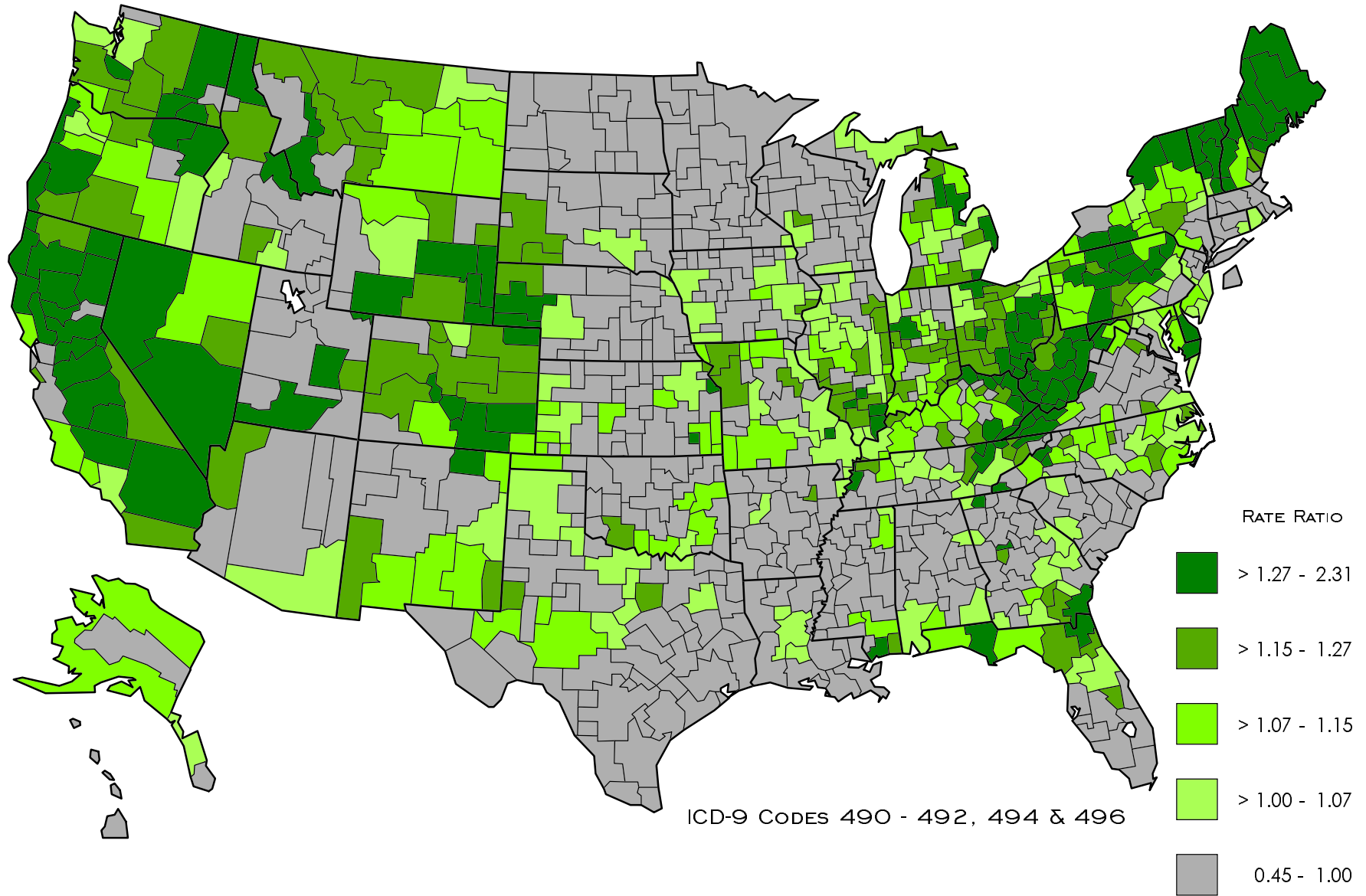
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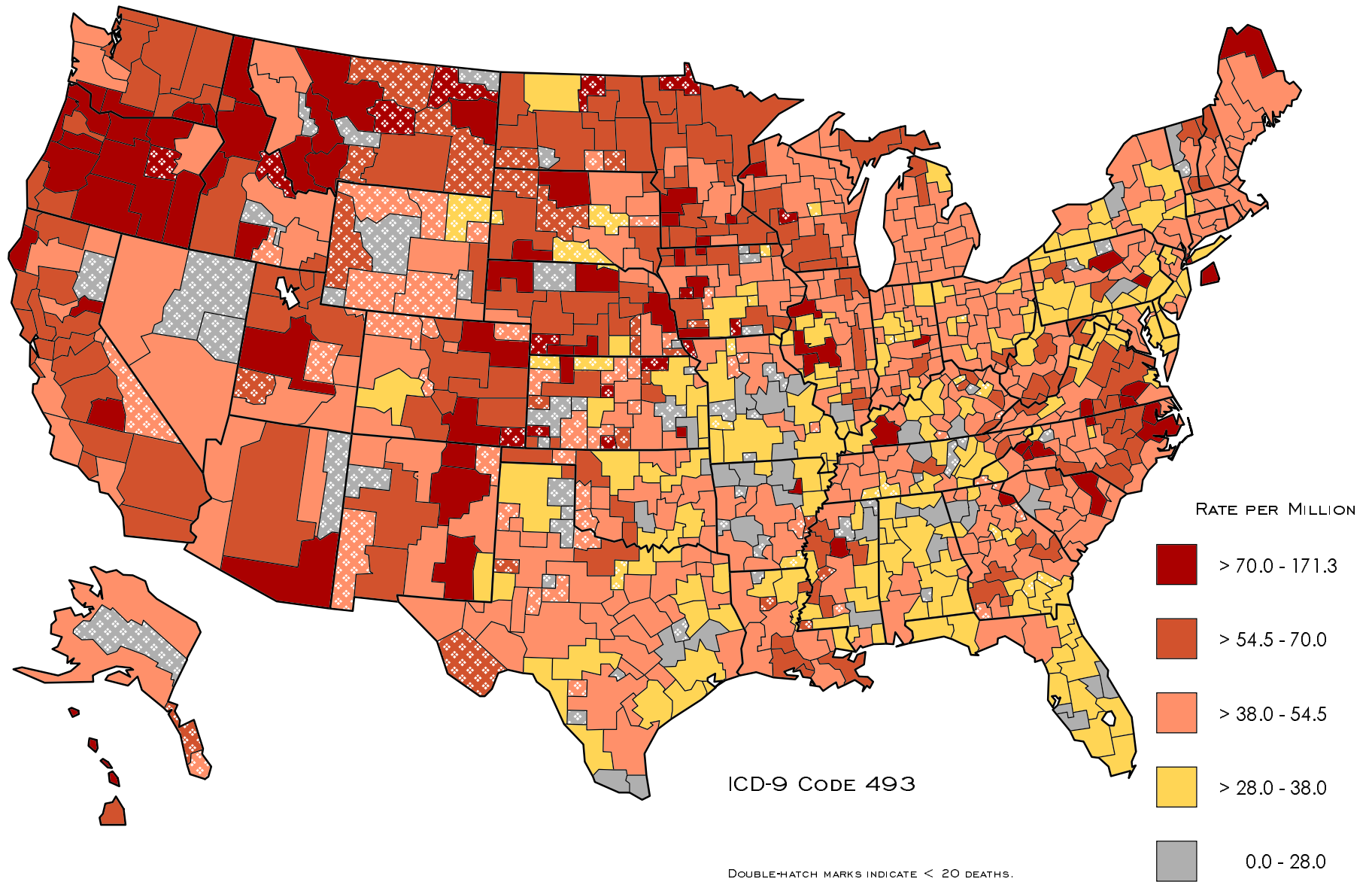
CHRONIC OBSTRUCTIVE PULMONARY DISEASE
AGE-ADJUSTED DEATH RATES BY HSA
U.S. RESIDENTS 15 YEARS OF AGE AND OLDER, 1982-1993



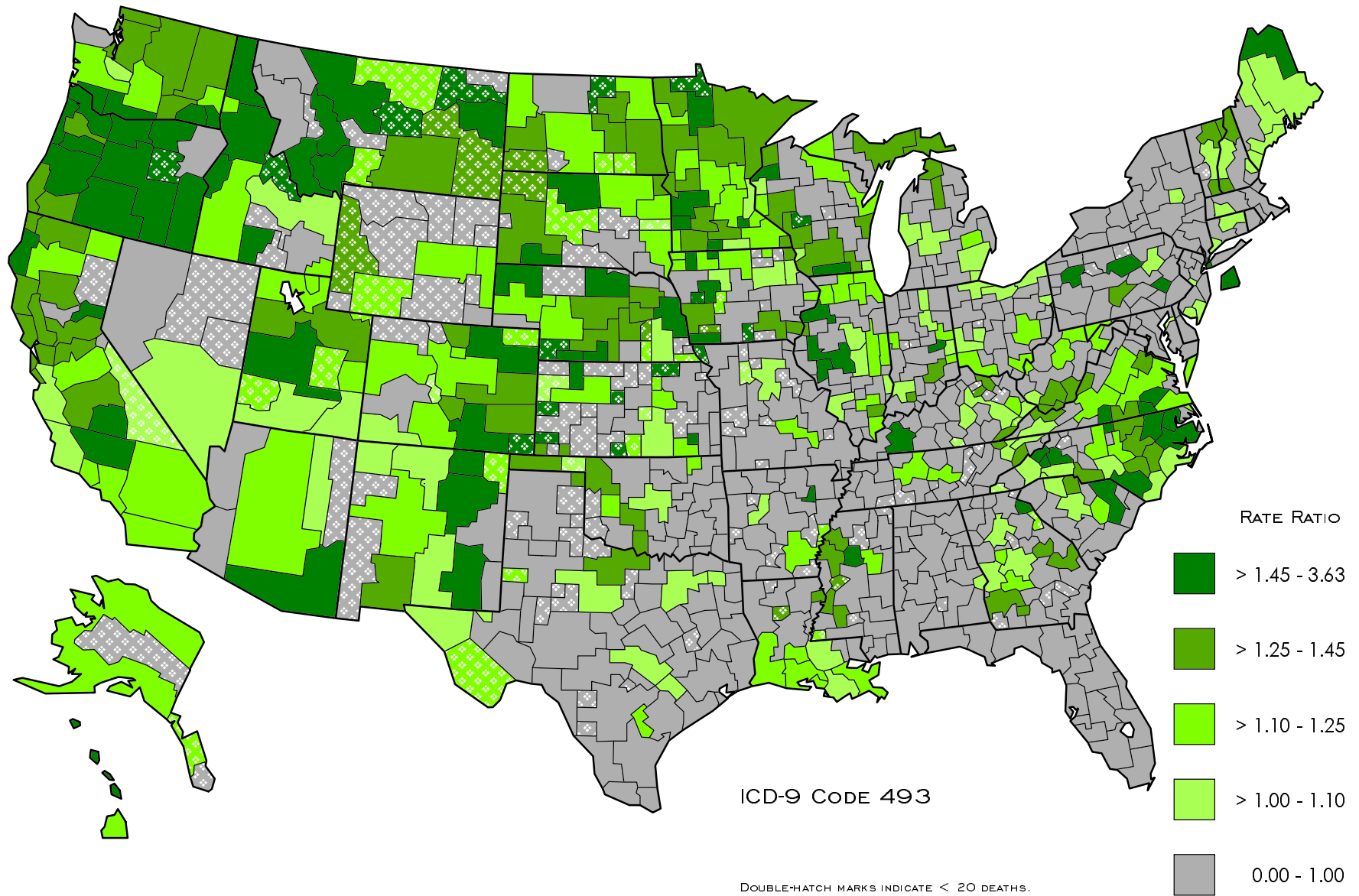
CHRONIC OBSTRUCTIVE PULMONARY DISEASE
DEATH RATES OF EACH HSA COMPARED WITH U.S. RATE
U.S. RESIDENTS 15 YEARS OF AGE AND OLDER, 1982-1993



ASTHMA
AGE-ADJUSTED DEATH RATES BY HSA
U.S. RESIDENTS 15 YEARS OF AGE AND OLDER, 1982-1993



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DEATH RATES OF EACH HSA COMPARED WITH U.S. RATE
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Asthma (ICD-9 Code 493)

Asthma (ICD-9 code 493) is a clinical condition characterized by variable airflow limitation and increased responsiveness of the tracheobronchial tree to a variety of stimuli [ATS 1987]. Asthma can be induced by occupational exposures to airborne antigens [Chan-Yeung 1994]. Asthma can also be aggravated in individuals with underlying asthma who are exposed to airborne irritants or bronchoconstrictive agents in the workplace [Wagner and Wegman 1998].

Asthma has been estimated to affect approximately 5 to 7 percent of the United States population [Brooks 1992]. The overall prevalence of occupational asthma in the United States is unknown, but occupational exposures may cause up to 15 percent of adult asthma [Blanc 1987], and 21-33 percent of adult asthma may be caused or significantly worsened by such exposures [Milton et al. 1998; Ng et al. 1994]. Asthma is the most frequently reported work-related respiratory disease in the United Kingdom [Ross et al. 1997].

Little is known about the geographic variation of asthma mortality. In addition to possible differences in exposure to occupational agents that cause asthma, geographic variation in asthma mortality may be due to urban-rural differences, humidity and climate conditions affecting both indoor and outdoor allergen exposures, ambient air pollution, nutrition, genetic predisposition, access to quality medical care, ethnic and socioeconomic factors [Sears 1997].

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Hypersensitivity Pneumonitis (ICD-9 Code 495)

Hypersensitivity pneumonitis (HP), also referred to by the British as extrinsic allergic alveolitis (ICD-9 code 495), is a group of immunologically-mediated lung diseases caused by inhalation of a wide variety of antigenic materials [Schuyler 1998]. Most cases are induced by particulate organic antigens, but some reported cases have been caused by soluble antigens and low molecular weight reactive chemicals such as isocyanates and trimellitic anhydride. At the present time, farmer's lung disease (caused by inhalation of thermophilic actinomycetes growing on moldy hay), bird fancier's disease (caused by bird proteins), HP associated with moisture and bioaerosols in buildings (caused by microbes contaminating humidifiers, dehumidifiers, and heating, ventilation, and air conditioning systems), and Japanese summer-type HP (caused by house dust contaminated with *Trichosporon cutaneum*) are the most commonly recognized forms of HP. The disease is often occupationally-related, and new types of HP continue to be reported as changing agricultural and industrial practices lead to new types of antigenic exposures. For example, HP has recently been recognized in machinists exposed to aerosolized metal working fluids that are microbially-contaminated [Kreiss and Cox-Ganser 1997].

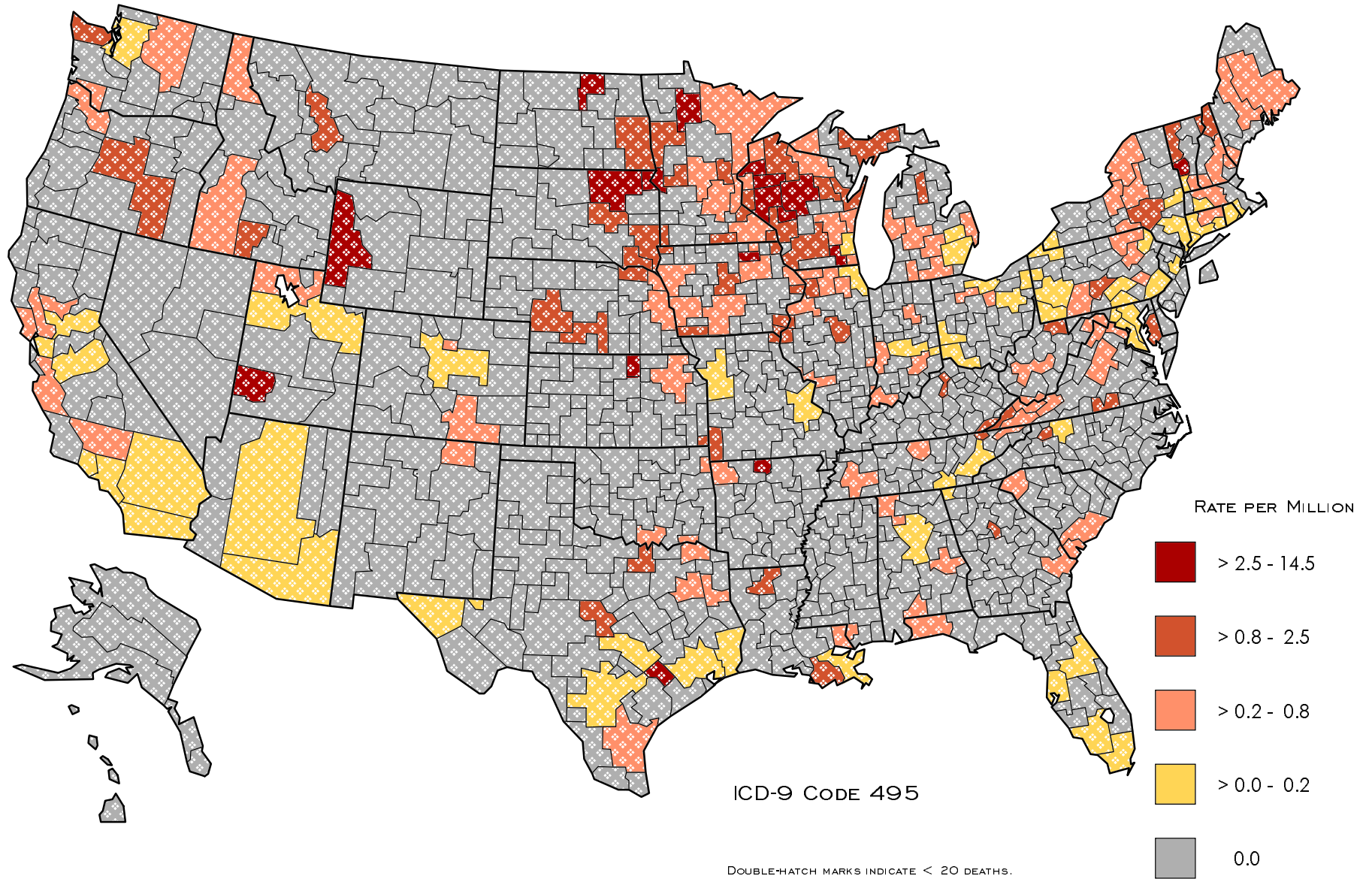
HP has two different clinical presentations. In *acute HP*, shortness of breath, nonproductive cough, generalized aches, chills, headache, and malaise occur 2 to 9 hours after exposure. *Chronic HP* presents as progressively severe shortness of breath, nonproductive cough, weight loss, and loss of appetite over a period of months to years. Chest radiographs show evidence of interstitial lung disease. Lung biopsies show chronic interstitial inflammation, usually with poorly formed nonnecrotizing granulomas; bronchiolitis obliterans is present in 25-50 percent of cases.

The prevalence of HP varies widely depending on the population studied and the method of case definition. Using a stringent case definition, a large population-based study of over 1400 Wisconsin dairy farmers found disease prevalence to be 0.4 percent [Gruchow et al. 1981]. In contrast, a study of Scottish farmers estimated a prevalence rate of 2.3 percent to 8.6 percent based on symptom and exposure histories [Grant et al. 1972]. Unlike some other lung diseases, the risk of HP is substantially lower among cigarette smokers, perhaps due to smoking effects on lung immunity [Schuyler 1998].

References

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